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COGNITIVE AND AFFECTIVE CONTROL

Hosted by
Gilles Pourtois, Wim Notebaert and
Tom Verguts

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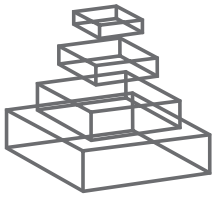

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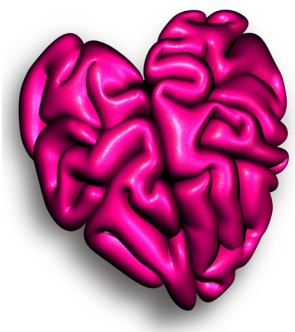

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Traditionally, cognition and emotion are seen as separate domains that are independent at best and in competition at worst. The French scientist and philosopher Blaise Pascal (1623-1662) famously said "Le cœur a ses raisons que la raison ne connaît point" (The heart has its reasons that reason does not know). Over the last century, however, psychologists and neuroscientists have increasingly appreciated their very strong reciprocal connections and interactions. Initially this was demonstrated in cognitive functions such as attention, learning and memory, and decision making. For instance, an emotional stimulus captures attention (e.g., Anderson and Phelps, 2001). Likewise, emotional stimuli are better learned and remembered than neutral ones (e.g., McGaugh, 1990) and they can provide strong incentives to bias decision making (Bechara et al., 1997).

In more recent years, cognitive control has also been found to be intimately intertwined with emotion. This is consistent with an approach that considers cognitive control as an adaptive learning process (Braver and Cohen, 1999), reinforcement learning in particular (Holroyd and Coles, 2002; Verguts and Notebaert, 2009). From this perspective, cognitive control is not a cool encapsulated executive function, but instead involves rapidly calculating the value of situational, contextual, and action cues (Rushworth and Behrens, 2008) for the purpose of adapting the cognitive system toward future optimal performance.

A wide array of research has shed light on cognitive control and its interactions with affect or motivation. Behaviorally, important phenomena include how people respond to difficult stimuli (e.g., incongruent stimuli, task switches), negative feedback, or errors and how this influences subsequent task processing. Neurally, an important target structure has been the anterior cingulate cortex (ACC) and its connections to traditional "emotional" (e.g., amygdala) and "cognitive" areas (e.g., (pre)motor cortex, dorsolateral prefrontal cortex). ACC seems to play a predominant role in integrating distant effects from remote cognitive and emotion systems in order to guide and optimize behavior.

The current special issue focuses on the bi-directional link between emotion and cognitive control. We invite studies that investigate the influence from emotion on cognitive control, or vice versa, the influence of cognitive control on emotion. Contributions can be of different types: We welcome empirical contributions (behavioral or neuroscientific) but also computational modeling, theory, or review papers. By bringing together researchers from the traditionally separated domains, we hope to further stimulate the crosstalk between emotion and cognitive control, and thus to deepen our understanding of both.

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EDITORIAL
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Cognitive and affective control

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Traditionally, cognition and emotion are seen as separate domains that are independent at best and in competition at worst. The French scientist and philosopher Blaise Pascal (1623–1662) famously said "Le coeur a ses raisons que la raison ne connaît point" (The heart has its reasons that reason does not know). Consistent with this quote, many studies in the past have underscored dissociable effects and non-overlapping brain structures of affect and cognition during the control and monitoring of goal-directed behavior (e.g., Bush et al., 2000). Over the last century, however, psychologists and neuroscientists have increasingly appreciated strong reciprocal connections and interactions between cognition and emotion. Initially this was demonstrated in cognitive functions such as perception, attention, learning, memory and decision-making. For instance, an emotional stimulus can alter low-level visual perception (e.g., Bocanegra and Zeelenberg, 2009), and it can capture attention (e.g., Anderson and Phelps, 2001). Likewise, emotional stimuli are better learned and remembered than neutral ones (e.g., McGaugh, 1990) and they can provide strong incentives to bias decision-making (Bechara et al., 1997).

Hence, the independent or competitive view is gradually being replaced by an interactive view. Currently, we focus on interactions of emotion and motivation with cognitive control. Empirical articles and review papers included in this Research Topic timely reveal the extent of overlap and synergistic effects between cognitive control and a wide range of affective processes, both in the normal adult brain, as well as in specific (pathological) conditions, best characterized by either poor or unripe prefrontal-based executive functions as well as impaired affective processes.

Broadly speaking, the original contributions included in this Research Topic tackle one (or more) out of three possible topics. The first and most represented consists of the influence of emotion on cognitive control. Krypotos et al. (2011) focus on the effect of individual differences in emotion regulation, measured by heart rate variability, on response inhibition. van Steenbergen et al. (2011) demonstrate attentional focusing after the presentation of negative pictures. Stürmer et al. (2011) discuss the effect of reward on conflict adaptation. Ridderinkhof et al. (2012) showed that positive affect restored decision learning in patients with Parkinson's disease. Reeck and Egner (2011) demonstrated that irrelevant emotional information distracts more than non-emotional information, supporting affective prioritization in human information processing. Demanet et al. (2011) study the effect of affective stimuli on voluntary task switching. Cavanagh et al. (2011) show that depression is associated with larger error (ERN) signals, suggesting an influence of motivational state on early error processing. Danielmeier and Ullsperger (2011) study the effect of errors (motivationally salient events) on post-error processing. Finally, Chiew and Braver (2011) review the influences of motivational state on early error processing.

In the second category, papers establish a conceptual or anatomical common substrate for cognitive control and emotion. Lowe and Ziemke (2011) endorse a perspective in which emotions are predictions of action tendencies. Aarts et al. (2011) review the literature supporting the hypothesis that (striatal) dopamine regulates the interaction between (appetitive) motivation and cognition. Mushtaq et al. (2011) look at similarities between uncertainty and cognitive control. Mueller (2011) reviews the developmental trajectories of cognitive and emotion control during adolescence. Berggren et al. (2011) emphasize the link between trait-related distractibility in healthy adults and their performance in standard cognitive tasks. Tops and Boksem (2011, 2012) propose that there are two cognitive control systems (one ventral and one dorsal), both of which are partially cognitive and partially affective. Su et al. (2011) propose the glance-look model, specifying how affect and cognitive control interact to produce the attentional blink.

In the third category, a relatively modest number of papers look at the influence of cognitive control on emotion. Krämer et al. (2011) demonstrate a correlation between cognitive control and aggression, suggesting an influence of the former on inhibiting the latter. The paper by Schmidt et al. (2011) reviews the effect of cognitive control on inhibition of thoughts for (being able to) sleep. Paret et al. (2011) demonstrate how cognitive control plays an important role in complex affective processes, such as emotion regulation and the reappraisal of our emotional life. Huizenga et al. (2012) investigate how repeated application of cognitive control influences motivational processing. Finally, the paper by Danielmeier and Ullsperger (2011) investigates the aftereffects of making an error.

In all, the main contribution of this special issue is to highlight similarities and reciprocal influences between cognitive control and emotion. Rather than separate modules, the papers gathered in this special issue concur in suggesting that emotion and cognitive control are two sides of the same coin, as they both contribute, through synergistic effects, to the optimization of behavior. As such, this special issue emphasizes the need to move beyond the classical division or dichotomy between cognitive control and emotion in order to model and account for human goal-directed behavior across various tasks and situations.

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Striatal dopamine and the interface between motivation and cognition

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Brain dopamine has long been known to be implicated in the domains of appetitive motivation and cognition. Recent work indicates that dopamine also plays a role in the interaction between appetitive motivation and cognition. Here we review this work. Animal work has revealed an arrangement of spiraling connections between the midbrain and the striatum that subserves a mechanism by which dopamine can direct information flow from ventromedial to more dorsal regions in the striatum. In line with current knowledge about dopamine's effects on cognition, we hypothesize that these striato-nigro-striatal connections provide the basis for functionally specific effects of appetitive motivation on cognition. One implication of this hypothesis is that appetitive motivation can induce cognitive improvement or impairment depending on task demands.

Keywords: dopamine, motivation, cognition, striatum, flexibility, prefrontal cortex, reward, Parkinson's disease

INTRODUCTION

The ability to control our behavior requires our actions to be goal-directed, and our goals to be organized hierarchically. Goals can be defined at different levels: motivational goals (e.g., rewards), cognitive goals (e.g., task-sets), and action goals (e.g., stimulus–response mappings). Thus, goal-directed behavior requires, among other things, the transformation of information about reward into abstract cognitive decisions, which in turn need to be translated into specific actions. The mechanisms underlying this hierarchy of goal-directed control are not well understood.

This paper focuses on the degree to which such goal-directed behavior is controlled by incentive motivation. We have restricted our discussion to the effects of appetitive motivation, while taking note of the wealth of evidence indicating that stimuli that activate the appetitive motivational system have an inhibitory influence on behavior that is controlled by the aversive motivational system (Konorsky, 1967; Dickinson and Balleine, 2002). Unlike aversive motivation, appetitive motivation refers to the state triggered by external stimuli that have rewarding properties and has been argued to have a general potentiating or enhancing effect on behavior and cognition (Dickinson and Balleine, 2002; Robbins and Everitt, 2003; Krawczyk et al., 2007; Pessoa, 2009; Jimura et al., 2010; Pessoa and Engelmann, 2010; Savine and Braver, 2010). Its effects on behavior and cognition have been associated with changes in neurochemical activity, such as increases in dopamine signaling in the striatum (Lyon and Robbins, 1975; Ikemoto and Panksepp, 1999; Robbins and Everitt, 2003; Berridge, 2007). This observation is generally in keeping with proposals that dopamine plays an important role in reward-related effort (Salamone et al., 2007) and generalized activation/energization of behavior (Robbins and Everitt, 2007). It is also consistent with data suggesting that dopamine might direct information flow from ventromedial frontostriatal circuits, implicated in reward and motivation, to more dorsal frontostriatal circuits, associated with cognition and action (Alexander et al., 1986; Haber and Knutson, 2010; **Figure 1**).

Although the widely distributed and diffuse nature of its projection system to large parts of the forebrain concurs with an account of dopamine in relatively non-specific terms, such as serving activation or energization, it is also clear that dopamine does not simply amplify (or suppress) all forebrain activity in a functionally non-specific manner. Indeed extensive evidence indicates that effects of dopamine depend on specific task demands and their underlying neural systems (Robbins, 2000; Cools et al., 2001a; Frank et al., 2004). In line with these insights, we suggest here that changes in appetitive motivation, which may result from changes in neurochemical activity, for example, due to stress, fatigue, or neuropsychiatric abnormality, also have functionally selective consequences for cognition.

More specifically, we put forward the working hypothesis that appetitive motivation might promote selectively our ability to switch between different tasks, providing us with some of the cognitive flexibility that is required in our constantly changing environment. Conversely, we speculate, based on preliminary data, that dopamine-mediated appetitive motivation might also have detrimental consequences for cognition, e.g., by impairing cognitive focusing and increasing distractibility. The implication of this speculation is that dopamine-mediated appetitive motivation might potentiate flexible behavior, albeit not by potentiating the impact of current goals on behavior. This speculation stems partly from the recognition that the motivational forces that drive behavior are not always under goal-direct control and can be maladaptive (Dickinson and Balleine, 2002). Moreover dopamine is well known to play an important role in mediating the detrimental (i.e., non-goal-directed) consequences of reward (Berridge, 2007; Robbins and Everitt, 2007).

Our working hypothesis is grounded in (albeit preliminary) empirical evidence indicating opposite effects of both dopaminergic and motivational/affective state manipulations on cognitive flexibility and cognitive focusing, which have been argued to reflect distinct striatal and prefrontal brain regions respectively (Crofts et al., 2001; Bilder et al., 2004; Dreisbach and Goschke, 2004; Dreisbach, 2006; Hazy et al., 2006; Cools et al., 2007; Rowe et al., 2007; van Steenbergen et al., 2009; Cools and D'Esposito, 2011). Indeed current models highlight a role for dopamine, particularly in the striatum, in the flexible updating of current task-representations (Hazy et al., 2006; Maia and Frank, 2011). The finding that appetitive motivation is associated with robust changes in dopamine levels particularly in the striatum, thus concurs with our hypothesis that appetitive motivation potentiates (at least some forms of) cognitive flexibility, perhaps even at the expense of cognitive focusing. Such a bias toward cognitive flexibility should be generally adaptive, given that motivational goals in the real world are not often readily available, thus requiring preparatory behavior that is flexible rather than focused (Baldo and Kelley, 2007).

Together these observations suggest that appetitive motivation acts to enhance cognition in a manner that is functionally specific, varying as a function of task demands, and that these functionally specific effects are mediated by dopamine. Clearly, as in the case of dopamine (Cools and Robbins, 2004; Cools et al., 2009), effects of appetitive motivation will vary not only as a function of task demands, but also as a function of the baseline state of the system. Thus both motivational and neurochemical state changes will have rather different effects in individuals with low and high baseline levels of motivation, consistent with the existence of multiple Yerkes Dodson "inverted U shaped" functions (Yerkes and Dodson, 1908; Cools and Robbins, 2004).

Let us briefly discuss the role of striatal dopamine in the two separate domains of motivation and cognitive control before addressing its role in their interaction.

DOPAMINE AND APPETITIVE MOTIVATION

The ventromedial striatum (VMS, including the nucleus accumbens) is highly innervated by mesolimbic dopaminergic neurons and is well known to be implicated in reward and motivation (Robbins and Everitt, 1992; Berridge and Robinson, 1998; Ikemoto and Panksepp, 1999; Schultz, 2002; Knutson and Cooper, 2005; Baldo and Kelley, 2007). Thus dopamine manipulations in the VMS affect performance on multiple paradigms thought to measure motivated behavior, including conditioned reinforcement, Pavlovian-instrumental transfer paradigms, effort-based decision making tasks, and progressive ratio schedules (Taylor and Robbins, 1984; Dickinson et al., 2000; Wyvell and Berridge, 2000, 2001; Parkinson et al., 2002). These experiments primarily reveal effects of dopamine on so-called preparatory conditioned responses, which are thought to reflect activation of a motivational system (Dickinson and Balleine, 2002), while leaving unaffected, or if anything, having the opposite effect on the more stereotypic patterns of consummatory responding (Robbins and Everitt, 1992; Baldo and Kelley, 2007). Thus administration of the indirect catecholamine enhancer amphetamine in the VMS of hungry rats potentiated locomotor excitement in the presence of food and increased lever pressing in response to, or in anticipation of a reward-predictive cue, while decreasing or leaving unaffected food intake as well as appetitive hedonic responses like taste reactivity (Taylor and Robbins, 1984; Bakshi and Kelley, 1991; Pecina et al., 1997; Wyvell and Berridge, 2000, 2001). Conversely, dopamine receptor blockade or dopamine lesions in the VMS reduced locomotor activity and cue-evoked incentive motivation for reward (Dickinson et al., 2000; Parkinson et al., 2002), while again leaving unaffected or even increasing food intake (Koob et al., 1978). These animal studies emphasize the importance of VMS dopamine in appetitive motivation and suggest that the hedonic or consummatory aspects of reward are likely mediated by a different, possible antagonistic system (Floresco et al., 1996; Robbins and Everitt, 1996, 2003; Berridge and Robinson, 1998; Ikemoto and Panksepp, 1999; Baldo and Kelley, 2007; Berridge, 2007; Phillips et al., 2007; Salamone et al., 2007; for similar suggestions in humans, see Aarts et al., 2010).

At first sight, this well-established observation provides apparently clear grounds for assuming that dopamine contributes to optimal reward- or goal-directed behavior. However, psychologists have also long recognized that there are multiple distinct components to the motivation of behavior (Konorsky, 1967; Dickinson and Balleine, 2002). Thus instrumental behavior is motivated not only by the goals that we set ourselves, but also by generalized drives and/or so-called Pavlovian "wanting," the latter two processes not necessarily always contributing to adaptive, optimized behavior. To clarify this point, it may help to consider the operational definition that psychologists have invoked for distinguishing instrumental behavior that is goal-directed from instrumental behavior that is not goal-directed, i.e., habitual (Dickinson and Balleine, 2002). Following this tradition, behavior is goal-directed only if it accords to two criteria; first, it has to be driven by knowledge about the contingency between the action and the outcome (as measured with contingency degradation tests); second, it has to be sensitive to changes in the value of the goal (as measured with outcome devaluation tests, involving for example selective satiety). Using these operational definitions, Dickinson and Balleine (2002) have established that Pavlovian conditioned stimuli that induce so-called "wanting" can modify instrumental behavior without accessing action–outcome representations, that is, in a manner that is not goal-directed. This is illustrated most clearly by the role of reward-predictive stimuli in compulsive craving for drugs of abuse or other targets of addiction, which of course almost always implicates dopamine dysfunction (Berridge and Robinson, 1998; Everitt and Robbins, 2005; Volkow et al., 2009). In keeping with this observation are suggestions that motivational influences on instrumental behavior by Pavlovian stimulus reinforcer contingencies might reflect modulation of well-established habits rather than of goal-directed behavior (Dickinson and Balleine, 2002). Data showing that dopamine D1/D2 receptor antagonists attenuated Pavlovian-instrumental transfer without affecting instrumental incentive learning (Dickinson et al., 2000) indeed suggested that dopamine might act through Pavlovian processes rather than through modifying action–outcome representations (Dickinson and Balleine, 2002).

In this context, it is perhaps not surprising that the effects of appetitive motivation on cognition that are mediated by dopamine are functionally specific, leading to cognitive improvement

or cognitive impairment depending on the specific task demands under study. An important implication of this observation is that effects of dopamine on interactions between motivation and cognitive control that appear to be mediated by a modification of motivational influences on cognitively mediated, goal-directed behavior may in fact reflect modification of motivational influences on habitual behavior.

DOPAMINE AND COGNITION

Accumulating evidence in the domain of cognition indicates that manipulations of dopamine can have contrasting effects as a function of task demands. For example, opposite effects have been observed in terms of cognitive flexibility and cognitive focusing (Crofts et al., 2001; Bilder et al., 2004; Cools et al., 2007; Durstewitz and Seamans, 2008; Durstewitz et al., 2010; Cools and D'Esposito, 2011). Mehta et al. (2004) have shown that dopamine D2 receptor blockade after acute administration of the antagonist sulpiride impaired cognitive flexibility (measured in terms of task-switching), but improved cognitive focusing (measured in terms of delayed response performance with task-irrelevant distractors). Similar contrasting effects on cognitive flexibility and focusing have been reported after dopamine lesions in non-human primates (Roberts et al., 1994; Collins et al., 2000; Crofts et al., 2001), after dopaminergic medication withdrawal in patients with Parkinson's disease (PD; Cools et al., 2001a, 2003, 2010a) and as a function of genetic variation in human dopamine genes (Bilder et al., 2004; Colzato et al., 2010). Evidence from functional neuroimaging and computational modeling work has suggested that these opposite effects might reflect modulation of distinct brain regions, with the striatum mediating effects on at least some forms of cognitive flexibility, but the prefrontal cortex (PFC) mediating effects on cognitive focusing (Hazy et al., 2006; Cools et al., 2007; Cools and D'Esposito, 2011). This hypothesis likely reflects an oversimplified view of dopamine's complex effects on cognition, with different forms of cognitive flexibility implicating distinct neural and neurochemical systems (Robbins and Arnsten, 2009; Kehagia et al., 2010; Floresco and Jentsch, 2011). In particular, the striatum seems implicated predominantly in a form of cognitive flexibility that involves shifting to well-established ("habitized") stimulus–response sets, that does not require new learning or working memory. For example 6-OHDA lesions in the striatum of marmosets impaired set-shifting to an already established set, but left unaffected set-shifting to a new, to-be-learned set (Collins et al., 2000). This finding paralleled the beneficial effects of dopaminergic medication in PD, which implicates primarily the striatum. These effects were restricted to task-set switching between well-established sets, and did not extend to set shifting to new, to-be-learned sets (Cools et al., 2001b; Lewis et al., 2005; Slabosz et al., 2006). The PFC might well be implicated in higher-order forms of set shifting that do involve new learning and/or working memory (Monchi et al., 2004; Floresco and Magyar, 2006; Cools et al., 2010b; Kehagia et al., 2010). Interestingly, the beneficial effects of dopaminergic medication in PD on this striatal form of well-established, habit-like task-set switching were accompanied by detrimental effects on cognitive focusing, as measured in terms of distractor–resistance during the performance of a delayed response task (Cools et al., 2010a). These findings paralleled pharmacological neuroimaging work with the same delayed response paradigm demonstrating that effects of dopamine D1/D2 receptor agonist administration to healthy young volunteers on flexibility (task-switching) and focusing (distractor–resistance) were accompanied by drug effects on the striatum and the PFC respectively (Cools et al., 2007).

In sum, dopamine's effects on cognition are known to be functionally specific rather than global, with opposite effects on cognitive flexibility and cognitive focusing. These opposite effects have been proposed to reflect modulation of distinct brain regions, with dopamine in the striatum playing a prominent role in a form of flexibility that involves shifting to well-established, i.e., "habitized" stimulus–response sets.

DOPAMINE AND THE MOTIVATION–COGNITION INTERACTION

So far we have seen that striatal dopamine's effect on motivated behavior is most prominent in terms of its preparatory component and that such preparatory effects can be maladaptive. This observation that dopamine's effect on motivation might have maladaptive consequences for behavior concurs with observations that effects of dopamine in the cognitive domain depend on task demands and associated neural systems, so that dopaminergic drugs can have detrimental as well as beneficial consequences for cognition. Together these insights have led to the speculation that incentive motivation might act to enhance cognitive performance by potentiating dopamine in the striatum in a manner that is functionally specific, i.e., restricted to a form of cognitive flexibility that involves shifting to well-established habits, and not extending to, or even at the expense of cognitive focusing. Below we review empirical evidence that address the different aspects of this working hypothesis.

EVIDENCE FROM NEUROANATOMICAL STUDIES

Motivation–cognition interactions have long been proposed to reflect dopamine-dependent interfacing between different parallel frontostriatal circuits associated with motivation and cognition (**Figure 1**). For example, neuroanatomical studies in rats from the 1970s have suggested that activity in the dorsal striatum is modulated by activity in the ventral striatum via the dopaminergic cells in the substantia nigra (Nauta et al., 1978). Tracer experiments in non-human primates have revived this notion by revealing an arrangement of spiraling striato-nigro-striatal (SNS) connections between the dopaminergic cells in the midbrain and striatal regions that were defined on the basis of their frontal cortical input (Haber et al., 2000; Haber, 2003). Similar connections have been found in rodents (Ikemoto, 2007). The SNS connections are thought to direct information flow in a feed-forward manner via stepwise disinhibition of the ascending dopaminergic projections from the VMS (including the nucleus accumbens), via the dorsomedial striatum (DMS, caudate nucleus), to the dorsolateral striatum (DLS, putamen). The resulting information flow from ventromedial to dorsolateral striatal regions provides a hierarchical (or heterarchical, see Haruno and Kawato, 2006) mechanism by which motivational goals can influence cognitive and subsequent motor control processes. Indeed, the VMS has long been hypothesized to provide the basis for the interface between motivation and action on the basis of its major inputs from limbic areas like the amygdala, hippocampus and the anterior cingulate cortex (ACC) and output to the motor areas

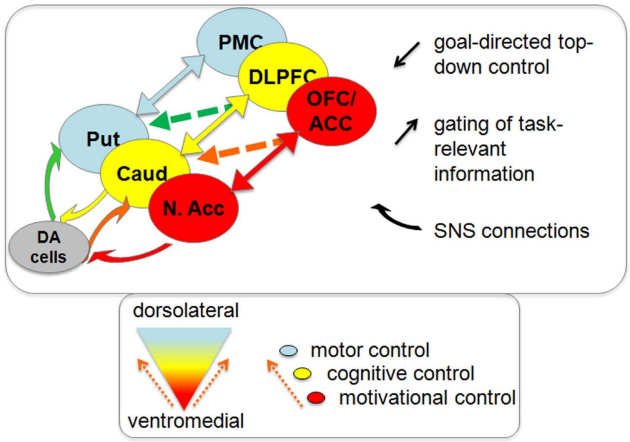

FIGURE 1 | Ventromedial to dorsolateral direction of information flow through frontostriatal-nigral circuitry. Interactions between the different frontostriatal loops involved in motivational control (red), cognitive control (yellow), and motor control (blue) can take place at the level of the SNS connections (bend arrows) or at the level of the frontostriatal connections (straight arrows). The direction of information flow is always from ventromedial to dorsolateral regions in the frontostriatal circuitry. SNS, striato-nigral-striatal; N. Acc, nucleus accumbens (ventromedial striatum); Caud, caudate nucleus (dorsomedial striatum); Put, putamen (dorsolateral striatum); OFC, orbitofrontal cortex; ACC, anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; PMC, premotor cortex.

via the globus pallidus (Mogenson et al., 1980; Groenewegen et al., 1996). However, rather than a direct limbic-motor connection, the SNS connections provide a more physiologically and psychologically plausible mechanism by which motivational goals exert their influence on action (Haber et al., 2000).

EVIDENCE FROM PSYCHOPHARMACOLOGICAL STUDIES IN ANIMALS

Rodent research on drug addiction has provided evidence for the functional importance of dopamine-mediated interactions between ventral and dorsal parts of the striatum. For example, Belin and Everitt (2008) have adopted an intrastriatal disconnection procedure in rats to investigate the necessity of the SNS connections in the transition of reward-directed drug-seeking behavior to habitual behavior associated with the DLS. The authors lesioned the VMS selectively on one side of the rat brain and, concomitantly, blocked dopaminergic input from the substantia nigra in the DLS with a receptor antagonist on the contralateral side of the brain. Thus, they functionally disconnected the VMS and DLS on both sides of the brain, while leaving unilateral VMS and DLS on opposite sites intact. This functional disconnection between VMS and DLS greatly reduced the transition of VMS-associated to DLS-associated habitual behavior, whereas the unilateral manipulations were ineffective in isolation (Belin and Everitt, 2008). These data show the functional importance of the spiraling SNS connections in VMS control over dorsal striatal functioning in addiction (Belin et al., 2009).

Functional evidence for a role of dopamine in interactions between motivation and DMS-associated functions has also been established in non-human primates. For example, neurophysiological recordings by Hikosaka and colleagues during the performance of a memory-guided saccadic eye-movement task revealed sensitivity of neuronal firing in the DMS as well as midbrain dopamine neurons to appetitive motivation. In this task, one of four directions was randomly assigned as the target location by a cue that also signaled the anticipation of reward. Subsequently, the monkey had to make a saccade to the remembered location. It was found that cues that predicted reward resulted in earlier and faster saccades relative to cues that predicted no reward. Firing patterns in caudate nucleus (DMS) neurons correlated with the change in saccade behavior, changing their preferred direction to the rewarded direction (Kawagoe et al., 1998). In a follow-up study, the authors observed that reward-predictive cues resulted in increased firing of dopaminergic neurons in the midbrain, as well as in neurons of the caudate nucleus (DMS; Kawagoe et al., 2004). Together, these findings demonstrate that effects of reward anticipation on DMS activity and associated motor-planning behavior were accompanied by changes in dopamine activity.

In humans, a role for dopamine in the effects of motivation on cognition has so far been addressed only in the domain of long-term memory associated with the hippocampus (Wittmann et al., 2005; Adcock et al., 2006; Schott et al., 2006; for a review, see Shohamy and Adcock, 2010). This relatively young field suggests that dopamine may well play a role in the long-term plasticity-enhancing effects of motivation. In the next section, we address studies that focus on dopamine-dependent effects of motivation on shorter term plasticity, involving the striatum.

EVIDENCE FROM HUMAN STUDIES: MOTIVATION AND COGNITIVE FLEXIBILITY

Data from two recent studies support the hypothesis that dopamine is critical for interactions between motivation and cognition. Specifically, these studies highlight an important role for dopamine in the modification by appetitive motivation of switching between well-established habits. The set-shifting paradigm involved cued task-switching between well-learnt task-sets, minimizing learning and working memory processes (Rogers and Monsell, 1995). Subjects switched between responding according to the direction of the arrow (task A) and responding according to the direction indicated by the word (task B) of a series of arrow-word targets (consisting of the words "left" or "right" in a left or right pointing arrow; **Figure 2A**). Repetitions or switches of task-set were pseudo-randomly preceded by high or low reward cues. In the first study, young healthy adults performed the task in the magnetic resonance scanner and both behavioral and neural responses were assessed as a function of inter-individual variability in dopamine genes (Aarts et al., 2010). In particular, we focused on a common variable number of tandem repeats (VNTR) polymorphism in the dopamine transporter gene (*DAT1*), expressed predominantly in the striatum. Relative to the 10R homozygotes, the 9R carriers – with presumably increased striatal dopamine levels – exhibited significant reward benefits in terms of overall performance and increased reward-related BOLD responses in VMS. However, most critically, they also demonstrated significant reward benefits in terms of task-switching (i.e., reduced switch costs in the high versus low reward condition). This effect was accompanied by a potentiation of switch-related BOLD responses in DMS (caudate nucleus) in the high reward versus the low reward condition (**Figures 2B,C**). Importantly, the reward-related activity in VMS correlated positively with the effects

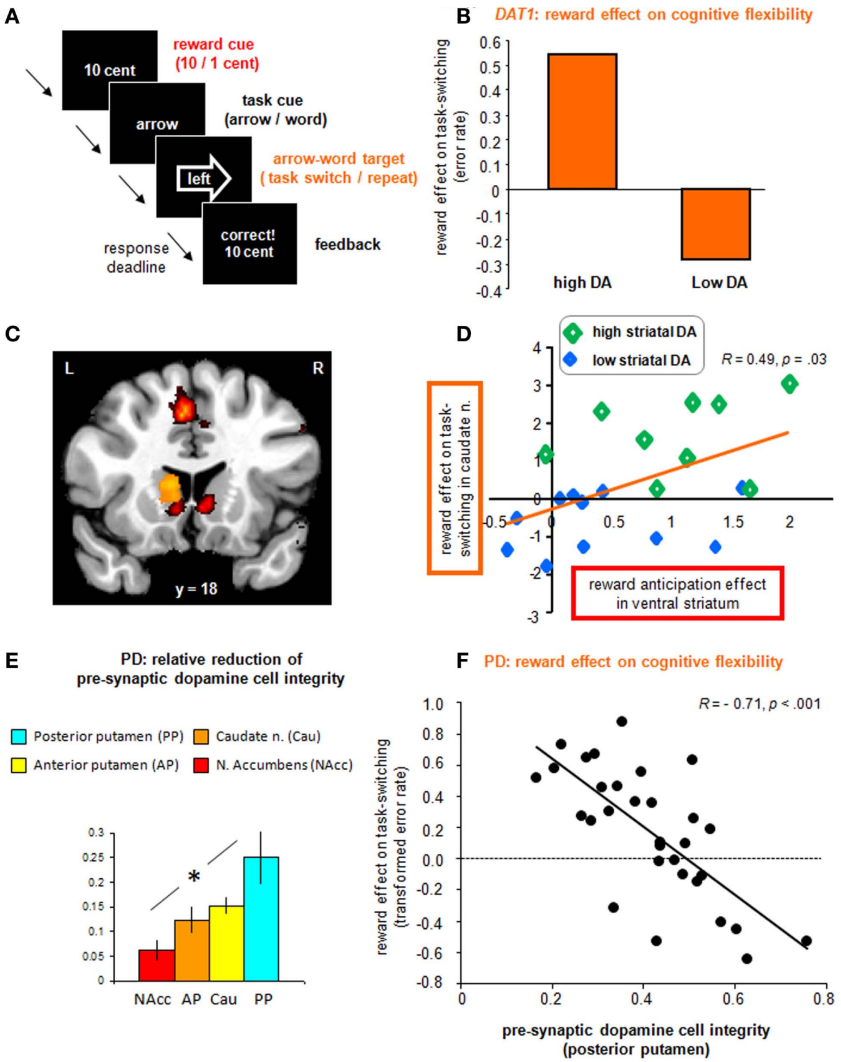

FIGURE 2 | Experimental evidence for the beneficial effect of motivation on cognitive flexibility in humans. (A) The rewarded set-shifting paradigm used in our studies to investigate the motivation–cognition interface. **(B)** In our genetic imaging study (Aarts et al., 2010), participants with genetically determined high striatal dopamine levels benefited more from reward anticipation in terms of set-shifting than participants with low dopamine levels. **(C)** In our genetic imaging study (Aarts et al., 2010), reward cues elicited activity in VMS (in red), whereas the dopamine-dependent effect of reward prediction on set shifting was observed in DMS (in yellow). **(D)** Activity in these striatal sub-regions [see **(C)**] was positively correlated, with high striatal dopamine subjects showing high activity in both VMS and DMS during reward anticipation and rewarded set-shifting respectively. **(E)** In our SPECT study in Parkinson's disease (Aarts et al., under review), patients showed the most marked dopamine depletion in dorsolateral striatum (posterior putamen), whereas ventromedial striatum (n. accumbens) was least affected. **(F)** Patients with the greatest dopamine depletion (i.e., least dopamine cell integrity) showed the greatest effects of anticipated reward in reducing the switch cost in the dominant arrow task [(switch-repeat)low – (switch-repeat)high]; presumably by increased reward-induced dopamine release in the relatively intact neurons in ventromedial striatum.

of reward on subsequent switch-related activity during the targets in DMS, with high dopamine subjects demonstrating high activity in both striatal regions (**Figure 2D**; Aarts et al., 2010). These dopamine-mediated motivation–cognition interaction effects were recently replicated in an independent dataset (van Holstein et al., 2011) and strengthened our working hypothesis that striatal dopamine mediates motivational modification of certain cognitive functions in humans.

In a second study, we investigated the effect of appetitive motivation on cognitive flexibility in patients with PD using the same paradigm (**Figure 2A**). Effects within the PD group were associated with the degree of dopamine depletion in different striatal sub-regions as measured with ^{123}I-FP-CIT single photon emission computed tomography (SPECT). First, we replicated previous studies by demonstrating a switch deficit in PD relative to healthy controls. Interestingly, this deficit was restricted to certain conditions of the task, revealing a disproportionate difficulty with switching to the best established, most dominant "arrow" task. Additionally, the SPECT measurements showed that this switch deficit in PD was associated with dopamine cell loss in the most affected striatal sub-region (posterior putamen, **Figure 2E**), thus demonstrating the involvement of striatal dopamine in this particular "habit-like"

type of cognitive flexibility. More critically, our results demonstrated compensatory capacity of reward-predictive signals to facilitate cognitive flexibility in mild PD. Specifically, when anticipating reward, patients were able to reduce the switch cost in the dominant arrow task to such an extent that the switch cost no longer differed from that of controls on high reward trials. Interestingly, the use of reward was also highly correlated with the amount of dopamine depletion in the most affected striatal sub-region (Aarts et al., under review). Patients with greater dopamine cell loss made more use of anticipated reward for reducing the switch cost than did patients with less dopamine cell loss (**Figure 2F**). Further exploration of this finding demonstrated that this effect of motivation on task-switching was driven by two opponent processes: first, patients with more dopamine depletion made more errors on repeat trials under high than under low reward. This detrimental effect of reward on repeat trials could reflect a form of impulsivity, where the current task representation is rendered unstable by reward, leading to reduced cognitive "perseverance" or maintenance (see also Hazy et al., 2006). Controls did not show such detrimental impulsive behavior on repeat trials under high reward. Second, patients with more dopamine depletion made fewer errors on switch trials under high than under low reward. Thus, anticipated reward proved beneficial for switching to the other task-set, which profits from reduced cognitive perseverance. This effect of reward on switch trials in patients did not differ from that of controls. The beneficial effects of anticipated reward on task-switching in the young healthy adults mentioned above (Aarts et al., 2010) was driven by a beneficial effect of reward on switch trials only, instead of opposite effects of reward on repeat and switch trials. In sum, PD patients differed from controls in showing detrimental effects of reward on repeat trials, which were greatest in patients with most dopamine cell loss in the striatum (Aarts et al., under review). This result fits with previous findings that a low baseline dopamine state contributes to trait impulsivity and addictive behavior (Cools et al., 2007; Dalley et al., 2007); presumably due to reduced auto-regulatory mechanisms, resulting in increased dopamine release (Buckholtz et al., 2010). Hence, we speculate that reward-induced impulsivity in our PD group was caused by increased reward-related dopamine release in the relatively intact dopamine cells projecting to the ventral striatum (**Figure 2E**). In line with this view are the findings of increased dopamine release in ventral striatum in PD patients diagnosed with impulsive–compulsive behavior relative to those without (Evans et al., 2006; Steeves et al., 2009; O'Sullivan et al., 2011). Our PD data are also in accordance with the working hypothesis that striatal dopamine mediates motivational effects on cognition depending on task demands.

EVIDENCE FROM HUMAN STUDIES: FUNCTIONALLY SPECIFIC EFFECTS OF MOTIVATION

Motivation has been shown to improve attentional processes in many perceptual and cognitive control domains (for reviews, see Pessoa, 2009; Pessoa and Engelmann, 2010). Data from a number of human imaging studies have suggested that motivation might have non-specific enhancing effects on cognitive processing. For example, in a functional neuroimaging study, motivational incentives increased PFC activity and connectivity during cognitive control tasks, in a manner that seemed to depend on the cognitive effort (i.e., cost–benefit ratio) rather than on the specific qualitative cognitive demand of the tasks (Kouneiher et al., 2009). Based on these data the authors argued that motivation and cognitive control can be regarded as two separate, additive instead of interactive factors of executive functioning (Kouneiher et al., 2009). However, such an additive view of motivation and cognition contrasts with the conclusion drawn by a different set of recent studies which enabled the disentangling of different cognitive control components. These studies have found that effects of appetitive motivation and affect may well depend on the type of cognitive processing at hand (Dreisbach and Goschke, 2004; Dreisbach, 2006; Rowe et al., 2007), consistent with our working hypothesis. Before turning to these studies, we will discuss preliminary data from our own lab.

So far we have seen that appetitive motivation can potentiate certain forms of task-switching to well-established stimulus–response mappings in a dopamine-dependent manner. The observation that these effects were driven by detrimental effects of anticipated reward on repeat trials and beneficial effects on switch trials in the PD group (Aarts et al., submitted) already indicates a level of functional specificity. To test more directly the hypothesis that these beneficial effects of appetitive motivation on some cognitive functions might come at the expense of impairments on other cognitive functions, we designed a Stroop-like conflict task with high and low reward conditions. This task resembled the previously used task-switching paradigm in many ways except that it required cognitive focusing instead of cognitive switching. Seventeen participants performed this Stroop-like task by responding with a left or right button press to the words "left" or "right" in a left or right pointing arrow (**Figure 3A**). The direction denoted by the word was either congruent or incongruent with the direction indicated by the arrow. Similar to the task-switching paradigm discussed above (Aarts et al., under review), all trials began with a cue predicting high or low reward for correct performance. Critically, following the reward cues, we explicitly informed participants about the (in)congruency of the upcoming Stroop target (see Aarts et al., 2008). In half of the trials, participants were informed about this congruency by informative cues (**Figure 3A**). In the other half of the trials, the targets were preceded by cues that gave no information about the upcoming congruency. The idea here was that incongruency-predictive cues (relative to non-informative cues) would encourage participants to reduce their attentional focus, whereas the congruency-predictive cues would encourage participants to widen their attentional focus. In other words, cues that signaled upcoming incongruent targets would encourage participants to proactively focus on the task-relevant word, preventing distraction by the task-irrelevant arrow, whereas cues that signal upcoming congruent words encouraged participants to proactively widen attention in order to comprise both the task-relevant word as well as the task-irrelevant arrow (see Aarts and Roelofs, 2010). The combination of reward and information cues enabled us to determine the effects of appetitive motivation on the cognitive focusing of attention.

Consistent with our previous results (Aarts et al., 2008) we showed that (irrespective of reward condition) participants responded faster and made less errors when informative cues preceded the congruent and incongruent targets relative to uninformed

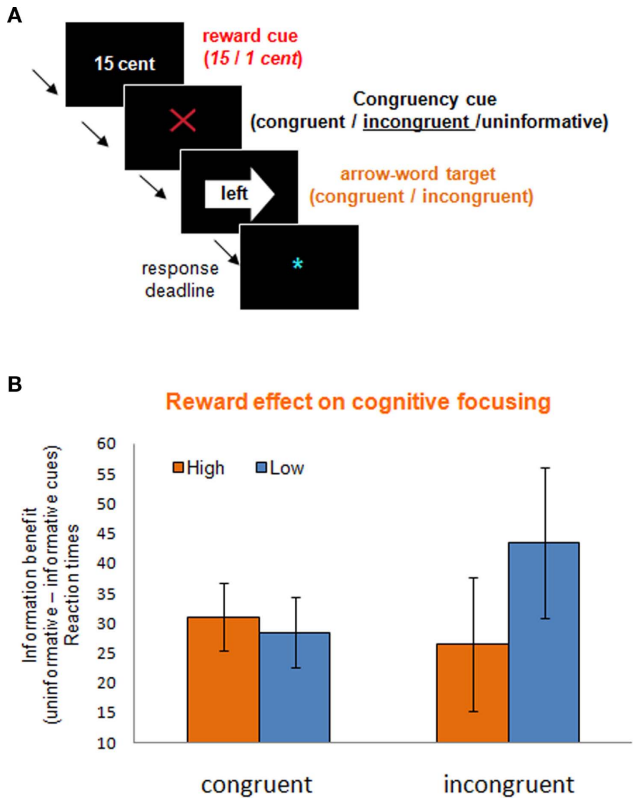

FIGURE 3 | Incentive motivation might have detrimental effects on cognitive focusing. (A) The rewarded Stroop paradigm, including a reward cue (1 or 15 cent), an information cue about the upcoming target congruency [informative: incongruent (this example) or congruent (green circle); or uninformative (gray question mark)], and an arrow-word Stroop target. The task was to respond to the direction indicated by the word. **(B)** Reward anticipation had opposite effects on widening and focusing of attention as measured with the information benefit (uninformed–informed) on congruent and incongruent targets respectively; with high anticipated reward particularly impairing proactive focusing on the incongruent trials (M. van Holstein, E. Aarts, R. Cools, unpublished observations).

targets (M. van Holstein, E. Aarts, R. Cools, unpublished observations). Importantly, as predicted, appetitive motivation significantly altered the information benefit depending on the congruency of the targets. That is, proactive widening of attention (uninformed–informed congruent targets) benefited from anticipated reward (15 versus 1 cent), whereas proactive focusing of attention (uninformed–informed incongruent targets) was hampered by anticipated reward (**Figure 3B**). Intriguingly, these data show that, depending on the task at hand, appetitive motivation can have both beneficial as well as detrimental effects on cognitive function.

Similar findings have been obtained when studying the effects of positive affect on cognitive control. Thus, positive affect has been shown to increase cognitive flexibility (i.e., decreasing perseveration), while increasing distractibility (i.e., decreasing cognitive stability) on different types of trials in a set-shifting paradigm (Dreisbach and Goschke, 2004). Opposite effects have also been observed in an AX continuous performance task: positive affect increased cognitive flexibility when a maintained goal unexpectedly changed (Dreisbach, 2006; van Wouwe et al., 2011), but, within the same task, positive affect decreased the ability to maintain the goal when nothing changed (Dreisbach, 2006). Functionally specific effects of positive affect have also been demonstrated in conflict paradigms, like the Eriksen flanker task. Some authors have shown that positive affect increased attention toward the distracting flanker arrows, thus, increasing "the breadth of attentional selection" (Rowe et al., 2007); others have found that positive affect reduced the ability to focus on the target arrow after experienced conflict (van Steenbergen et al., 2010). Our preliminary results from the rewarded Stroop conflict paradigm extend these effects of positive affect in the flanker conflict task, by revealing contrasting effects of appetitive motivation on the widening and focusing of attention within the same task and within the same participants. In sum, both appetitive motivation and positive affect enhance certain forms of cognitive flexibility at the expense of cognitive focusing. According to our working hypothesis, these effects might reflect dopamine-dependent flow of information processing related to Pavlovian incentives from ventromedial parts of the striatum to more dorsal regions in the striatum, associated with habit-like information processing.

It might be noted here again that multiple mechanisms have been proposed to underlie the motivational control of behavior (Dickinson and Balleine, 2002). We have highlighted that some motivational influences can be maladaptive, and these might implicate dopamine. However, there is also evidence for motivational influences on goal-direct behavior, that is, those mediated by instrumental incentive learning and acquisition of action–outcome representations (Dickinson and Balleine, 2002). These alternate mechanisms might account for findings that at first sight seem incompatible with the current working hypothesis. Specifically, appetitive motivation has been shown to increase spatial orienting to a target location in the face of distractors (Engelmann and Pessoa, 2007; Engelmann et al., 2009), or to reduce conflict by biasing visual selection (Padmala and Pessoa, 2011). Furthermore, in young and old adults as well as in medicated patients with PD, motivation increased anti-saccade performance, encompassing incompatible stimulus–response mappings like in Stroop and flanker paradigms (Harsay et al., 2010). The critical question is whether these effects are also dependent on striatal dopamine, or whether they implicate modulation by different neurochemical systems. Addressing this question requires controlled dopaminergic medication withdrawal and/or pharmacological manipulation approaches.

FRONTAL CONTROL OF DOPAMINE-DEPENDENT STRIATAL PROCESSING

The striatum does not act alone and requires interactions with specific frontal regions to operate effectively (Alexander et al., 1986; Passingham, 1993; **Figure 1**). Recent neuroimaging work in humans and monkeys has revealed that effects of appetitive motivation on cognitive control are accompanied by modulation of responses in the PFC (Ichihara-Takeda and Funahashi, 2008; Kouneiher et al., 2009; Beck et al., 2010; Ichihara-Takeda et al., 2010; Jimura et al., 2010; Savine and Braver, 2010; Wallis and Kennerley, 2010). For example, functional interactions between the medial and the lateral PFC have been shown to accompany effects of appetitive

motivation on the cognitive control processes involved in set shifting (Kouneiher et al., 2009). Another functional neuroimaging study concluded that the lateral PFC incorporates reward value in goal-directed control during working memory processes (Jimura et al., 2010).

These data concur with the existence of multiple mechanisms for the motivational control of behavior, which may interact in multiple ways, either competitively or synergistically. For example, signals in the PFC might control dopaminergic activity in striatal areas in a top-down manner, thus allowing controlled influences on value assignment to states or actions (Daw et al., 2005; Doll et al., 2009; see **Figure 1**). Consistent with this hypothesis are observations that stimulation of different parts of the frontal cortex (using transcranial magnetic stimulation) alters focal dopamine release in strongly connected topographically specific parts of the striatum (as measured using [11C]raclopride positron emission tomography; Strafella et al., 2001, 2003, 2005; Ko et al., 2008). The role of the PFC in integrating motivation, cognition, and action is also highlighted by anatomical tracer studies in non-human primates showing that value-sensitive regions in ventromedial PFC (i.e., ACC/orbitofrontal cortex) project not only to strongly connected regions in VMS, but also diffusely to more dorsal regions in the striatum that receive most projections from the DLPFC (Haber et al., 2006; **Figure 1**). Electrophysiological work with rodents has revealed that changes in dopamine release and receptor stimulation in the striatum can alter such PFC input to the striatum (Goto and Grace, 2005). More specifically, changes in tonic dopamine release were shown to modulate PFC inputs into the VMS – and to influence set-shifting behavior – through dopamine D2 receptors (Goto and Grace, 2005). These results show that striatal dopamine can modulate motivated behavior not only via altering striatal output but also via altering striatal input *from* the PFC.

CONCLUSION AND FUTURE DIRECTIONS

There are multiple mechanisms for the control of behavior and cognition by motivation. This paper focuses on the appetitive motivational system, while recognizing that opponent influences on behavior are likely seen of the aversive motivational system. In particular, we have concentrated on those effects of appetitive motivation that implicate dopamine. These dopamine-dependent effects of motivation likely have both detrimental as well as beneficial consequences for cognition, via altering information flow from ventromedial to dorsolateral parts of the striatum. This general observation is in line with the observation that motivational influences on behavior are not necessarily driven by representations of the goals of instrumental behavior, but might well reflect Pavlovian or habit-like anomalies. This is particularly likely in the case of dopamine, which is recognized to play a special role in Pavlovian and habit systems.

An important implication of this observation is that effects of dopamine on interactions between motivation and cognition that appear to be mediated by a modification of motivational influences on cognitively mediated, goal-directed behavior, like task-switching, may in fact reflect modification of motivation influences on habitual behavior. Findings that the dopamine-dependent effects of motivation on task-switching are strongest when participants are required to switch to well-established stimulus–response mappings are in line with this hypothesis, which requires testing in future work.

A further issue to be addressed in future research is the degree to which the contrasting effects of motivation on habit-like switching and on proactive focusing can be understood in terms of competition between a striatal system controlling habit-like processing and a prefrontal system controlling goal-directed behavior (Dickinson, 1985; Daw et al., 2005). Clearly these questions require a careful integration of traditional psychological approaches, which leverage well-operationalized behavioral definitions of goal-directed and habitual behavior, with pharmacological studies of cognitive control.

Furthermore, given the proposed opponency between appetitive and aversive motivational systems, one might ask what is the effect of punishment-predictive stimuli on cognition? This is particularly interesting in the context of empirical findings that conditioned inhibitors, i.e., stimuli predictive of reward omission do not trigger an increase, but rather if anything a decrease in midbrain dopamine firing (Tobler et al., 2005). Moreover, there is increasing speculation about the involvement of the part-opponent system of serotonin (Daw et al., 2002; Dayan and Huys, 2009; Boureau and Dayan, 2011; Cools et al., 2011), an area that is wide open for empirical work.

Finally, progress in the understanding of the motivational control of cognition will depend on the degree to which the balance between transient and sustained, e.g., context effects are taken into account (e.g., Higgins et al., 1997; Maddox and Markman, 2010; Savine et al., 2010). For example, Maddox and Markman (2010) propose that performance does not only depend on local incentives and task demands (as discussed in the current review), but also interacts with global incentives like an overall bonus or punishment at the end of a task. Such advances will no doubt benefit from the recognition that the impact of transient (phasic) changes in neurotransmitter activity depends critically on the tonic neurochemical state of the system.

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The effects of self-report cognitive failures and cognitive load on antisaccade performance

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Individuals reporting high levels of distractibility in everyday life show impaired performance in standard laboratory tasks measuring selective attention and inhibitory processes. Similarly, increasing cognitive load leads to more errors/distraction in a variety of cognitive tasks. How these two factors interact is currently unclear; highly distractible individuals may be affected more when their cognitive resources are taxed, or load may linearly affect performance for all individuals. We investigated the relationship between self-reported levels of cognitive failures (CF) in daily life and performance in the antisaccade task, a widely used tool examining attentional control. Levels of concurrent cognitive demand were manipulated using a secondary auditory discrimination task. We found that both levels of self-reported CF and task load increased antisaccade latencies while having no effect on prosaccade eye-movements. However individuals rating themselves as suffering few daily life distractions showed a comparable load cost to those who experience many. These findings suggest that the likelihood of distraction is governed by the addition of both internal susceptibility and the external current load placed on working memory.

Keywords: cognitive failures, antisaccade performance, distractibility, cognitive load

INTRODUCTION

Cognitive slips and errors are common in daily life, with most people at one time or another forgetting where they left their car keys or if they left a light switched on at home. However, some individuals are more likely to commit such slips than others. The cognitive failures questionnaire (CFQ; Broadbent et al., 1982) is an established measure of individual differences in daily cognitive slips, with a number of questions relating to everyday errors such as the likelihood of dropping objects or failing to keep a task goal in mind. While these kinds of questions might be seen to reflect lapses in memory, scores on this questionnaire are positively correlated with increased distraction of attention in a number of daily life situations: from absentmindedness while shopping (Reason and Lucas, 1984), to an increased number of car accidents (Larson and Merritt, 1991) and other mishaps or injuries at work (Wallace and Vodanovich, 2003). Notably, CFQ scores remain relatively constant over time (Broadbent et al., 1982), and spouse ratings consistently match self-reported scores (Hickox and Sunderland, 1992), demonstrating that CFQ is a robust index of daily life cognitive failures (CF).

In addition to correlates with daily life slips, high CFQ scorers also show specific deficits in experimental investigations of attention and cognitive control. For instance, increased levels of CF are associated with increased interference in the Eriksen flanker task and Stroop task (Broadbent et al., 1986; Tipper and Baylis, 1987), and impaired performance when dividing attention (Harris and Wilkins, 1982). Tipper and Baylis (1987) also found that high CFQ scorers showed no evidence of negative priming or inhibition to distractors while consistent negative priming effects were seen for low scorers, suggesting that CF may be associated with a reduced ability to inhibit task-irrelevant information. Importantly, CFQ scores do not appear to predict performance in memory tasks (Wilkins and Baddeley, 1978), though they have been noted to affect memory in tasks requiring the inhibition of unwanted memories (Groome and Grant, 2005).

One explanation for the relationship between CFQ scores and poor performance on tasks of selective attention maybe that both reflect a failure to maintain task goals in working memory (WM). Loading WM via secondary tasks has been shown to disrupt selective attention in a similar manner to that reported in the study of CFQ (see, e.g., Gazzaley, 2011, for review). One paradigm exemplifying this is the antisaccade task, in which participants are required to inhibit a prosaccade toward a sudden onset target, and initiate a saccade toward its mirror image location. Converging evidence suggests that antisaccade performance is linked to WM processes. For example, participants with low WM span show increased errors (prosaccades toward the target; Unsworth et al., 2004). Increased errors and increased correct antisaccade latencies are found in populations with known WM deficits, such as elderly participants, and for first-episode schizophrenic patients (Nieuwenhuis et al., 2004; see Hutton and Ettinger, 2006, for review). In addition, secondary WM loads such as mental arithmetic or n-back correspondingly have adverse effects on correct antisaccade latencies and error rates (e.g., Roberts et al., 1994; Kane et al., 2001).

While both internal factors such as personality and external factors including cognitive load can adversely affect selective attention, very few investigations have attempted to examine the extent to which these two factors interact. In daily life, we are required to perform cognitive tasks that range in difficulty, in different

environments that include varying sources of potential distractions, from a quiet office to driving on a busy road. Though one's susceptibility to CF and concurrent cognitive load are separate, in the real world they are not independent. Are individuals who experience little distraction in life more able to cope in situations where cognitive resources are taxed by load, and are those who experience many distractions less able to cope when situations are more cognitively demanding? Divided attention tasks provide some evidence that individuals reporting high levels of CF do suffer more distraction than low CF reporters, but these tasks tend not to compare performance across both single and dual-task conditions within the same experiment (e.g., Harris and Wilkins, 1982).

Accordingly, we investigated the role of both CFQ score and cognitive load using the antisaccade task. A secondary auditory task of either low or high load was also employed, requiring either passive verbal response or more complex pitch discrimination respectively. A previous investigation using the antisaccade task found that high CFQ score was correlated with faster antisaccade latencies but a greater number of errors (Larson and Perry, 1999). However, this finding might imply an unusual speed–accuracy trade-off in high CFQ scorers rather than evidence of a cognitive efficiency deficit, and the experiment also contained a number of other methodological concerns (see Discussion). We hypothesized that high levels of CF would be correlated with increased antisaccade latencies in line with previous findings involving groups characterized by deficits in cognitive inhibition (e.g., individuals with high levels of anxiety; Derakshan et al., 2009; see Derakshan and Eysenck, 2009, for a review). We also predicted that cognitive load would impair antisaccade latencies while not impacting upon prosaccade latencies. Finally, we examined the effect of load on both low and high CFQ scorers, while also measuring levels of state anxiety to ensure stressors did not affect scorers differently.

MATERIALS AND METHODS

PARTICIPANTS

Forty-two participants (25 female) were recruited via advertisements at the University of London to take part in the experiment (mean age 24.36, range 20–36). Participants had normal or corrected-to-normal vision. They reported no auditory impairments.

APPARATUS

Eye-movements were recorded using an SR Research Eyelink 1000 eye-tracker (SR Research, ON, Canada).Only one eye was tracked during the experiment. Nine-point calibration across the computer screen was used to ensure tracking accuracy was within 1° of visual angle. Images were presented on a 21″ Viewsonic CRT monitor (140 Hz), and a chinrest was used to ensure a constant viewing distance of 60 cm. The experiment was presented using the SR Research Experiment Builder software. A separate laptop played the auditory tones, presented through E-Prime software.

STIMULI AND PROCEDURE

The experiment took place in a dimly lit and sound-protected room. Prior to the experiment proper, participants completed the CFQ (Broadbent et al., 1982) in addition to the state version of the State-Trait Anxiety Inventory (Speilberger et al., 1983; also completed at the end of the experiment). In the main experiment, participants were instructed to look "AT," or "AWAY" from a white oval-shaped item appearing on-screen depending on the block, while also concurrently responding to tones presented in the background verbally (i.e., low or high load).

Each trial began with a fixation cross (0.95° × 0.95°) presented in the center of the screen for 1000 ms. Participants were instructed to fixate this cross. If participants fixated the cross between 500 and 1000 ms after its onset, the trial moved forward immediately, acting as a drift correct to tracking. The oval-shaped target subtending 2.58° × 4.77° then appeared either in the left or right periphery of the screen for 600 ms, at an eccentricity from fixation to the center of the oval of 11.04°. In the prosaccade block, participants were asked to move their eyes from fixation to the target as quickly and as accurately as possible. In the antisaccade block, participants were instructed to move their eyes to the mirror image location as quickly and as accurately as possible, while trying to avoid looking toward the target. An inter-trial interval of 1500 ms then occurred.

For the secondary task, auditory tones were played in the background of the room via a laptop. In the high load condition, one of three tones differing in pitch was randomly presented every 1900–2300 ms (five choices of 100 ms increments). Participants were asked to respond with "low," "mid," or "high" depending on the pitch, while concurrently performing anti- and prosaccades. In the low load condition, participants were asked to simply say the word "tone" whenever one was played. Only the mid tones were used during this block, to ensure that participants did not implicitly discriminate the tone pitch despite not being required to. The experimenter informed the participant at the start of each block what combination of "AT"/"AWAY" and "TONE"/"PITCH" they would be conducting. The experimenter started the tones at the same time as the participant pressed the escape key on the keyboard to begin the block, and stopped the auditory tones when a block ended. The experimenter monitored the participants' performance and prompted them if they made errors in the discrimination. Good speed and accuracy was emphasized for both tasks.

Participants were given initial practice at distinguishing the tones, along with 16 practice trials for anti/prosaccades. The main experiment consisted of 8 blocks of 36 trials (two blocks for each condition), and block order was counterbalanced across participants. Participants were thanked, debriefed, and paid £5 for their contribution, at the end of the experiment.

RESULTS

The data for 34 participants were used in the analysis[1]. Trials in which no saccade was made, or trials in which a saccade was made in under 80 ms (anticipatory saccade; see Fischer et al., 1993) were excluded from analysis. This led to an average of 6.04% of trials being removed (no effects of CF group on percentage of trials removed were observed). Median CFQ score for the entire sample was 42 (SD = 13.36). Participants were divided based on the median split as either low ($N = 17$) or high scorers ($N = 17$). The two groups' scores significantly differed from each other

[1]Data from eight participants were removed either for the percentage of excluded trials being above 40% or for error rates of over 50%.

[Med = 30.41 vs. 52.29; $t(32) = 8.47$, $p < 0.001$]. Additionally, participants' self-reports of state anxiety before and after the experiment were averaged to obtain a composite single state anxiety score reflecting levels of state anxiety during the experimental session. In this respect mean state anxiety for the entire sample was 36.51 (SD = 7.84).

RESPONSE LATENCIES

Response latency was analyzed only for correct trials. A 2 (Load: low, high) × 2 (Task: Prosaccade, Antisaccade) × 2 (Group: low CF, high CF) mixed ANOVA was conducted on median latencies, with the means of individual median scores reported herein. This revealed a trend for a main effect of Load [$F(1,32) = 3.56$, $p = 0.07$] and a significant effect of Task [$F(1,32) = 228.8$, $p < 0.001$]. Participants were moderately slower under high load compared to low load (low load: $M = 221$, SD = 33; high load: $M = 228$, SD = 38), while also much slower on antisaccade ($M = 270$, SD = 47) vs. prosaccade trials ($M = 178$, SD = 29). Importantly, there was a significant Load × Task interaction [$F(1,32) = 9.75$, $p < 0.005$] with load significantly increasing antisaccade response latencies [$t(33) = 3.48$, $p = 0.001$; low load: $M = 262$, SD = 48; high load: $M = 279$, SD = 51], while having no effect on prosaccade latencies ($t < 1$; low load: $M = 179$, SD = 31; high load: $M = 176$, SD = 33).

Effects of CFQ scores and state anxiety

While no main effect of Group was observed [$F(1,32) = 2.2$, $p = 0.15$], a significant Task × Group interaction [$F(1,32) = 4.08$, $p = 0.05$] was found. To assess this relationship further, we employed correlational analysis on each condition using CFQ score as a continuous variable. There was a positive relationship between CFQ scores and antisaccade latencies, $r = 0.411$, $p = 0.01$ (see **Figure 1**), and this relationship was significant for both low ($r = 0.439$, $p < 0.01$) and high cognitive load ($r = 0.352$, $p < 0.05$) conditions. The two correlation coefficients did not differ significantly from each other ($Z = 0.41$, $p = 0.68$) Meanwhile, no significant correlation was found with prosaccade latencies under low, $r = 0.03$, or high cognitive load, $r = 0.13$, p's > 0.1. Finally, Group did not significantly interact with Load or Load and Task together (F's < 1); load cost specifically on antisaccade latencies was comparable when examining CF groups separately (low CF: M diff = 26 ms, SD = 35; high CF: M diff = 14 ms, SD = 39).

Self-reported state anxiety correlated with CFQ scores ($r = 0.397$, $p < 0.03$; see **Figure 2**), while showing a moderate relationship also with antisaccade latencies ($r = 0.33$, $p = 0.055$). A hierarchical regression analysis was performed with CFQ scores entered on Step 1 and state anxiety on Step 2, to examine if state anxiety explained additional variance in antisaccade latencies after allowing for the main contributing effect of CFQ scores. State anxiety did not significantly predict AS latencies (unstandardized β = 1.19, SE β = 1.04, $t = 1.14$, $p = 0.24$), after allowing for the effect of CFQ scores (unstandardized β = 1.44, SE β = 0.56, $t = 2.55$, $p < 0.02$).

ERROR RATES

Errors were defined as saccades either to the oval target when participants were instructed to look away, or saccades away from the target when instructed to look at it. A 2 (Task; Antisaccade, Prosaccade) × (Load: low, high) × 2 (Group: low CF, high CF) mixed ANOVA assessed reflexive errors (antisaccade trials) and possible incorrect inhibition (prosaccade trials) as percentages of the total number of trials. Analysis revealed a main effect of Load, $F(1,32) = 88.92$, $p < 0.001$, and a main effect of Task, $F(1,32) = 4.7$, $p < 0.05$. Participants made more errors under high load ($M = 13.99$, SD = 7.69) compared to low load ($M = 11.62$, SD = 7.71) and, as would be expected, far more errors were made on antisaccade trials ($M = 19.93$, SD = 10.62) vs. prosaccade trials ($M = 5.68$, SD = 4.83). The Task × Load interaction was not significant ($F < 1$).

There was no main effect of Group or interaction with Load (F's < 1). Furthermore, Task × Group did not interact

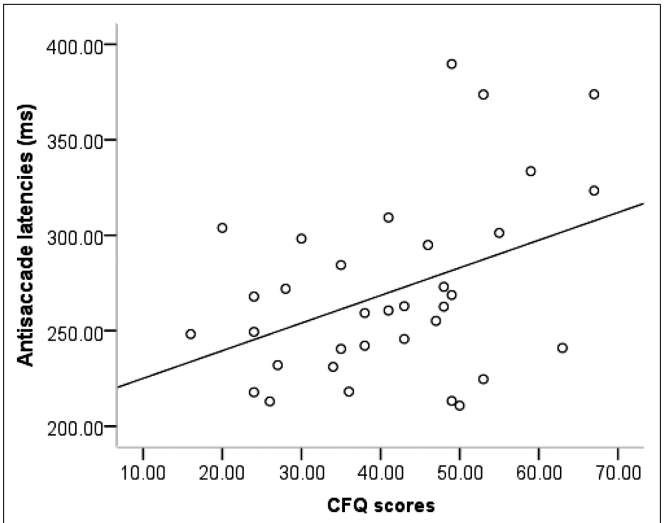

FIGURE 1 | Relationship between CFQ scores and antisaccade latencies (collapsed across load).

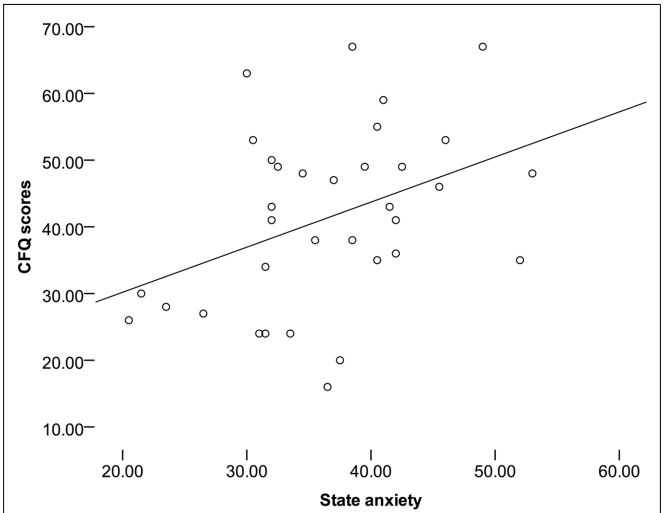

FIGURE 2 | Relationship between CFQ scores and state anxiety.

[$F(1,32) = 1.57$, $p > 0.2$], and there was no three-way interaction [$F(1,32) = 1.68$, $p > 0.2$]. Scores or state anxiety correlated with errors committed under high or low load in either anti- or prosaccade tasks (all r's < 0.1).

DISCUSSION

The present study establishes that both one's dispositional susceptibility to CF and the situational cognitive load imposed on task goals can additively increase the likelihood of distraction as assessed by the antisaccade task. Both CFQ score and load caused clear detrimental effects on antisaccade latency, while having no effect on prosaccades, thus demonstrating that both measures purely affected trials requiring inhibition. Importantly, we found no evidence of a differential effect of load for low and high CFQ scorers on latencies and this would suggest an additive, rather than interactive, role of both these factors in distraction.

Our findings build upon previous research in a number of ways. To our knowledge, only one previous study has documented a relationship between CFQ score and lapses in overt attention (Larson and Perry, 1999). Establishing this point is important considering that many previous laboratory investigations of CFQ score do not always present visual displays for periods that preclude eye-movements. Differences in overt attention might therefore account for increased distractor processing in high CFQ scorers. Additionally, this previous study on CFQ score and overt attention had a number of methodological concerns, the most pressing being long eye-movement latencies within a normal population of over 480 ms on average for a reflexive prosaccade. Unusually, this average is far longer than reported here or even for latencies reported in clinical populations (e.g., Gooding and Tallent, 2001). Moreover, the experiment had no counterbalancing of block order, with prosaccade blocks always first, and this could potentially interact with individual differences in the latter condition (see Kane et al., 2001, for comment). Here, we distinguished between trials that were characterized by no saccades, anticipatory saccades, and erroneous reflexive saccades, establishing that CFQ only affected correct antisaccade latencies as opposed to reflexive errors. This finding is comparable to other studies investigating antisaccade differences within the general population (e.g., trait anxiety; Derakshan et al., 2009), and contests the point that CFQ is necessarily associated with overt attention as we did not observe any effects on error rates. In other words, high CFQ scorers were merely slower to disengage from the target on an antisaccade trial, which possibly only reflects a lapse in covert attention.

Additionally, our manipulation of load also slowed antisaccade latencies while having no effect on prosaccade latencies. Previous manipulations of load in overt attention tasks have relied upon concurrent n-back (Mitchell et al., 2002), mental arithmetic (Roberts et al., 1994), or concurrent tapping at set intervals (Stuyven et al., 2000). Roberts et al. (1994) presented numbers which participants were required to add together mentally in sets of five items, and so it is somewhat difficult to operationalize effects of load as every five sequences a load began from zero, while additionally numbers were given at no set interval by the experimenter vocally, creating a possible experimenter bias in sequence timing in the low load (repeat the number spoken by the experimenter) and high load (add each number spoken) conditions. Tapping load also has potential issues with participants timing saccades to keypress metronome, evidenced by the finding that tapping increases prosaccade as well as antisaccade latencies (Stuyven et al., 2000). Here, our load manipulation appears a useful tool for future research, avoiding the issue of keypresses by using auditory responses. That said, our method of monitoring participants' accuracy in the secondary task, with the experimenter prompting volunteers when appropriate, could be improved upon in future research, with the use of more quantitative methods registering participant responses. It should be noted however that our pitch discrimination of low, mid, and high tones may have been an effective load because it involved a spatial aspect of pitches relative to one another. Indeed, n-back load also requires an understanding of the spatial order of items, and so it is possible that cognitive load only affects antisaccade and overt attention performance when that load taxes the same sub-component of WM needed to perform a task (see Kim et al., 2005, for a similar argument on covert attention).

The finding that load did not affect CFQ scorers differently is particularly important to our understanding of daily life distraction. As mentioned, if anything high CFQ scorers showed a smaller increase in latencies under load than low scorers. That said, CFQ scores significantly predicted slower response latencies under both low and high load alike. We suggest that CF may cause an increased likelihood of lapses in attention, but that cognitive load simply adds to this susceptibility by linearly decreasing cognitive resources for all individuals equally. Considering that a link between CFQ score and WM capacity has not been established in previous studies, our findings suggest that CFQ score may be associated with deficits in cognitive efficiency (i.e., inhibition) rather than a reduced capacity. Hence, cognitive load would act by reducing the amount of available cognitive resources, but would not interact with a factor like CFQ score, which instead reduces the efficiency of cognitive resources in suppressing task-irrelevant information.

The present study also measured levels of state anxiety, due to conceptual concerns that state experiences of worry, and anxiety during a demanding cognitive task could explain any observed differences between low and high CF scorers. In contrast, our evidence suggested that although state anxiety was associated with antisaccade latency performance, CFQ was incrementally a much strong predictor on performance. Furthermore, state anxiety did not predict performance when the influence of CFQ score was taken into account. Thus, our findings suggest that anxiety during a difficult cognitive task can predict performance, but this anxiety is inherently explained by one's self-perceptions of distractibility. It should be noted that we did not assess the relationship between *trait* anxiety (sustained personality characteristics of anxiety) on CFQ score's predictive power. Previous work has shown that high trait anxious individuals exhibit larger costs on performance under high cognitive load (Berggren et al., in press), and also that trait anxiety score and CFQ score are positively correlated (e.g., Smith et al., 1995). Thus, one might argue that trait anxiety could account for some of the variance explained by CFQ score, despite our evidence suggesting that CFQ scorers are no differently affected in magnitude by load. Future research would benefit from

more directly comparing these two personality factors together in affecting lapses in overt attention.

As well as that high CFQ scorers did not show greater costs on performance as load increased, it is also interesting to interpret results that low scorers, while under both low and high load faster than high scorers, did not show any reduced cost by cognitive load. This finding has relevance to the study of individual risk perception in the context of increasingly demanding daily tasks. White et al. (2004), for example, showed that while drivers agreed that concurrently using a mobile phone was one of the riskiest activities one could engage in, they perceived their own personal risk of an accident by doing this as less likely than for other people. This optimism bias is clearly not supported in the present study, where the addition of a demanding dual-task impaired performance at a similar magnitude both for individuals who perceive themselves as making few CF in daily life, and those who consider themselves to commit many. An interesting avenue of future research would be to examine more closely the relationship between individuals' risk perception and their ability to perform a task under varying cognitive demands, such as within a driving simulator.

In summary, both self-reported CF in daily life and cognitive load predict performance in overt attention and the ability to ignore distraction. The contributions of both these internal and external factors appear to cumulatively govern the likelihood of focused attention and cognitive efficiency.

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Larger error signals in major depression are associated with better avoidance learning

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The medial prefrontal cortex (mPFC) is particularly reactive to signals of error, punishment, and conflict in the service of behavioral adaptation and it is consistently implicated in the etiology of major depressive disorder (MDD). This association makes conceptual sense, given that MDD has been associated with hyper-reactivity in neural systems associated with punishment processing. Yet in practice, depression-related variance in measures of mPFC functioning often fails to relate to performance. For example, neuroelectric reflections of mediofrontal error signals are often found to be larger in MDD, but a deficit in post-error performance suggests that these error signals are not being used to rapidly adapt behavior. Thus, it remains unknown if depression-related variance in error signals reflects a meaningful alteration in the use of error or punishment information. However, larger mediofrontal error signals have also been related to another behavioral tendency: increased accuracy in avoidance learning. The integrity of this error-avoidance system remains untested in MDD. In this study, EEG was recorded as 21 symptomatic, drug-free participants with current or past MDD and 24 control participants performed a probabilistic reinforcement learning task. Depressed participants had larger mid-frontal EEG responses to error feedback than controls. The direct relationship between error signal amplitudes and avoidance learning accuracy was replicated. Crucially, this relationship was stronger in depressed participants for high conflict "lose–lose" situations, demonstrating a selective alteration of avoidance learning. This investigation provided evidence that larger error signal amplitudes in depression are associated with increased avoidance learning, identifying a candidate mechanistic model for hypersensitivity to negative outcomes in depression.

Keywords: major depressive disorder, FRN, reinforcement learning, computational psychiatry, theta, anterior cingulate cortex

INTRODUCTION

At the interface of emotion and cognition, affective neuroscience has the potential to advance the characterization of disease states away from idiosyncratic symptom-based criteria toward common brain-based nosology (cf. Insel et al., 2010). One promising example is evidenced by the convergence of cognitive, emotional, and neurological accounts of major depressive disorder (MDD). In addition to cardinal features of anhedonia and low mood, cognitive processing in MDD is characterized by a negative emotional distortion of the world, the self, and the future (Beck, 1976). Eshel and Roiser (2010) have suggested that these symptoms of MDD may reflect an impairment in basic reward (hypo-responsive) and punishment (hyper-reactive) processing systems. In this investigation, we propose a mechanism by which the hyper-reactive distortion of punishment information in MDD biases avoidance learning, possibly increasing the salience of "bad" outcomes.

The medial prefrontal cortex (mPFC), particularly the anterior cingulate cortex, appears to be centrally involved in a self-monitoring network. This system is consistently activated in neuroimaging investigations of reward and punishment (Carter et al., 1998; Ridderinkhof et al., 2004) and it is strongly implicated in the etiology of MDD (Davidson et al., 2002). The ACC has been described as a functional node in complex processes such as adaptive control over behavior and acquisition of reinforcement contingencies, as a dynamic processing hub for attention and action selection, and as a sensitive determinant of motivational functions including emotional reactivity and willful engagement (Devinsky et al., 1995; Vogt, 2005). The combined activities of this particular neural system identify it as a focal node by which emotion may be internalized to affect cognitive functioning.

One reliable measurement proposed to reflect mPFC functioning is the feedback-related negativity (FRN), a scalp-measured electrical voltage deflection occurring after feedback indicating a loss of value, or a performance error. The FRN reflects phase-locked theta band activities and is thought to reflect the functions of an action monitoring system that uses signals of error, conflict, or punishment to adapt future behavior (Holroyd and Coles, 2002; Frank et al., 2005). Larger error signals have been found in MDD

participants, both to negatively valenced feedback (Tucker et al., 2003) and to response errors (Chiu and Deldin, 2007; Holmes and Pizzagalli, 2008, 2010). Yet paradoxically, depressed participants are characterized by deficits in performance adaptations following error, conflict, and punishment (Elliott et al., 1996; Pizzagalli et al., 2006; Holmes and Pizzagalli, 2007; Compton et al., 2008), even in the context of larger error signals (Holmes and Pizzagalli, 2008). Thus, it remains unknown if larger error signals in depression reflect a functional increase in performance-monitoring integrity.

There is another, longer-term consequence associated with larger error-related mid-frontal activities: increased ability in learning to avoid stimuli that have been previously associated with punishment, especially for very difficult (high conflict) choices (Frank et al., 2005, 2007a; Cavanagh et al., 2010a,b). This learning is suggested to reflect the involvement of the mPFC with basal ganglia systems during slow probabilistic integration of action values (Frank et al., 2007b). We have previously detailed how emotional reactivity to social stress can instantiate a reinforcement learning bias in this slow integrative system (Cavanagh et al., 2010a). In that study, negative affect altered the processing of punishment information (as indicated by mid-frontal theta), which in turn predicted the efficacy of avoidance learning. Depressed patients have been shown to overreact to punishment information (Elliott et al., 1996, 1997), but the functioning of this error-avoidance system in MDD remains unknown. Our previous findings suggest a novel and testable hypothesis. Since larger error signals lead to better avoidance learning, enhancement of this relationship in MDD might reveal a mechanistic explanation for hypersensitivity to negative outcomes in MDD.

MATERIALS AND METHODS

PARTICIPANTS

All participants provided written informed consent that was approved by the University of Arizona. Participants were recruited from introductory psychology classes based on mass survey scores of the beck depression inventory (BDI). Recruitment criteria included: (1) age 18–25, (2) no history of head trauma or seizures, and (3) no current psychoactive medication use. Control participants ($N = 24$, 14 female) had stable low BDI (<7) between mass survey and preliminary assessment, no self-reported history of MDD, and no self-reported symptoms indicating the possibility of an Axis 1 disorder as indicated by computerized self-report completion of the Electronic Mini International Neuropsychological Interview (eMINI: Medical Outcome Systems, Jacksonville, FL, USA). Depressed participants needed to have a stable high BDI (>13), and needed to meet criteria for current or past MDD during a Structured Clinical Interview for the DSM-IV. A total of $N = 21$ (14 female; 10 current MDD, 11 past history of MDD) symptomatic participants met these criteria. Participants with current and past MDD history were grouped together in this study to increase power; this decision was additionally motivated by the fact that BDI score reflected a moderate severity of depression and did not differ between the current and past history groups (current: $M = 22$, SD = 5.54; past: $M = 21$, SD = 5.54). All subsequent task procedures and EEG processing steps are identical to Cavanagh et al. (2010a) except where otherwise indicated.

TASK

Participants performed a probabilistic learning task twice, with a self-paced break between tasks, using different pseudo-randomly assigned character sets. Each task included a forced choice training phase followed by a subsequent testing phase (Frank et al., 2004), as shown in **Figure 1**. During the training phase the participants were presented with three stimulus pairs, where each stimulus was a Japanese Hiragana character associated with a different probabilistic chance of receiving "Correct" or "Incorrect" feedback. These stimulus pairs (and their probabilities of reward) were termed A/B (80%/20%), C/D (70%/30%), and E/F (60%/40%). All training trials began with a jittered inter-trial-interval between 300 and 700 ms. The stimuli then appeared for a maximum of 4000 ms, and disappeared immediately after the choice was made. If the participant failed to make a choice within the 4000-ms, "No Response Detected" was presented. Following a button press, either "Correct" or "Incorrect" feedback was presented for 500 ms (jittered between 50 and 100 ms post response).

During the testing phase all possible stimulus pairs were presented eight times (120 trials total). Trials in the test phase began with an ITI of 500 ms. Stimuli were presented for a maximum

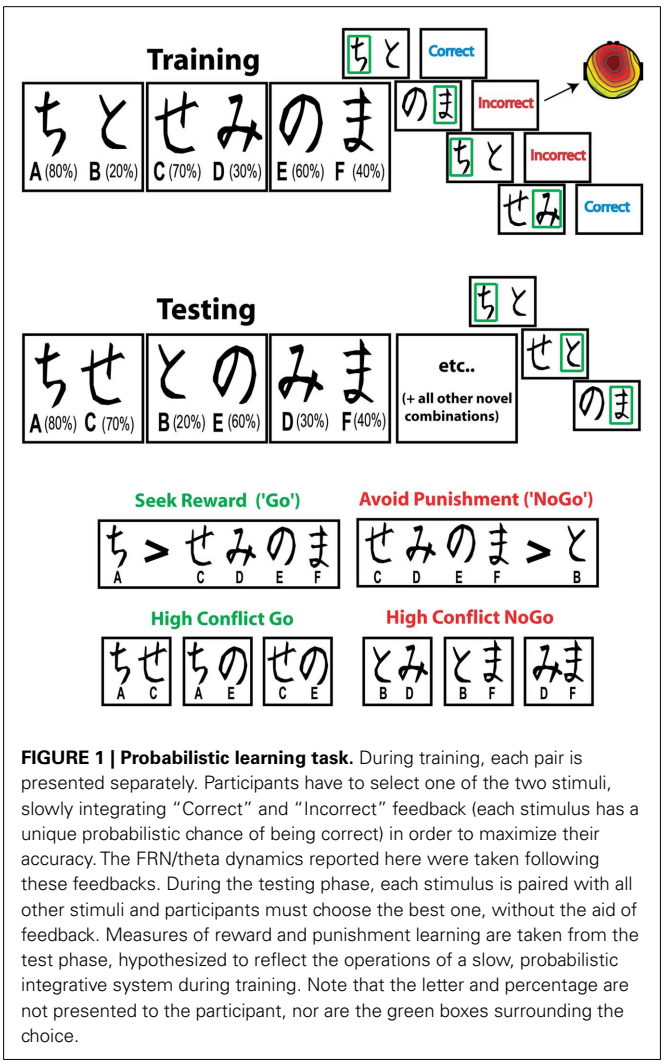

FIGURE 1 | Probabilistic learning task. During training, each pair is presented separately. Participants have to select one of the two stimuli, slowly integrating "Correct" and "Incorrect" feedback (each stimulus has a unique probabilistic chance of being correct) in order to maximize their accuracy. The FRN/theta dynamics reported here were taken following these feedbacks. During the testing phase, each stimulus is paired with all other stimuli and participants must choose the best one, without the aid of feedback. Measures of reward and punishment learning are taken from the test phase, hypothesized to reflect the operations of a slow, probabilistic integrative system during training. Note that the letter and percentage are not presented to the participant, nor are the green boxes surrounding the choice.

of 4000 ms, and disappeared as soon as a choice was made. No feedback was provided in the testing phase. Reward seeking ("Go learning") was defined as the accuracy of choosing A over C, D, E, and F (i.e., seeking A), whereas punishment avoidance or "NoGo learning" was defined as the accuracy of choosing C, D, E, and F over B (i.e., avoiding B). Conflict trials were defined based on the reinforcement value difference between the available choices (with smaller, more subtle differences in reinforcement values associated with increasing conflict). Thus, we analyzed performance separately for high conflict Go (AC, AE, CE), high conflict NoGo (BD, BF, DF), low conflict Go (AD, AF), and low conflict NoGo (BC, BE). We have previously referred to these types of high conflict valenced decisions as "win–win" (Go) and "lose–lose" (NoGo) situations (Frank et al., 2007c; Cavanagh et al., 2010a). To increase sensitivity, data from the two administrations of the task were combined if participants were able to select the most rewarding stimulus (A) over the most punishing stimulus (B) at least 50% of the time during the testing phase on each administration (based on this criterion, five participants in each group had data from only one administration). For this investigation, EEG signals were taken from the training phase (responses to feedback during learning), and behavioral indices of learning were taken from the testing phase. This analytic strategy allows an assessment of how the neural processing of feedback during learning relates to value-based decision making at a later point in time.

ELECTROPHYSIOLOGICAL RECORDING AND PROCESSING

Scalp voltage was measured using 64 Ag/AgCl electrodes using a Synamps^2 system (bandpass filter 0.5–100, 500 Hz sampling rate, impedances $<10\,k\Omega$), referenced offline to averaged mastoids. Eyeblinks were removed with Independent Components Analysis (Delorme and Makeig, 2004). Because the FRN represents phase-locked theta activity following feedback, data were processed to obtain both time-domain FRN amplitudes, as well as time–frequency theta band activity in this same time range. Event-related EEG was time-locked to correct and incorrect feedback during training and baseline corrected to the average power from 300 to 200 ms before feedback. Baseline-independent amplitudes of the incorrect ERPs (filtered 0.5–15 Hz) were computed as the difference between the mean values in 20 ms windows around the grand average peak (P2 or P3) and the trough (FRN) at FCz (P2: 200 ms, FRN: 276 ms, P3: 376 ms), see **Figure 2A**, yielding two difference scores: P2-FRN, and P3-FRN. Thus, larger values indicate larger amplitude deflections. Note that these time differences correspond to the period of a 5–7-Hz (theta) rhythm. This type of peak-to-trough quantification of ERP components has been shown to correlate with between-subjects differences in theta power better than baseline corrected mean amplitude (Cavanagh et al., 2011).

Time–frequency calculations were computed using custom-written Matlab routines (Cohen et al., 2008; Cavanagh et al., 2009). Time–frequency measures were computed by multiplying the fast Fourier transformed (FFT) power spectrum of single trial EEG data with the FFT power spectrum of a set of complex Morlet wavelets (defined as a Gaussian-windowed complex sine wave: $e^{-i2\pi tf}e^{-t^2/(2*\sigma^2)}$, where t is time, f is frequency (which increased from 1 to 50 Hz in 50 logarithmically spaced steps), and σ defines the width (or "cycles") of each frequency band, set according to $3/(2\pi f)$, and taking the inverse FFT. The end result of this process is identical to time-domain signal convolution, and it resulted in estimates of instantaneous power (the magnitude of the analytic signal), defined as $z[t]$ (power time series: $p(t) = \text{real}[z(t)]^2 + \text{imag}[z(t)]^2$). Whereas our previous

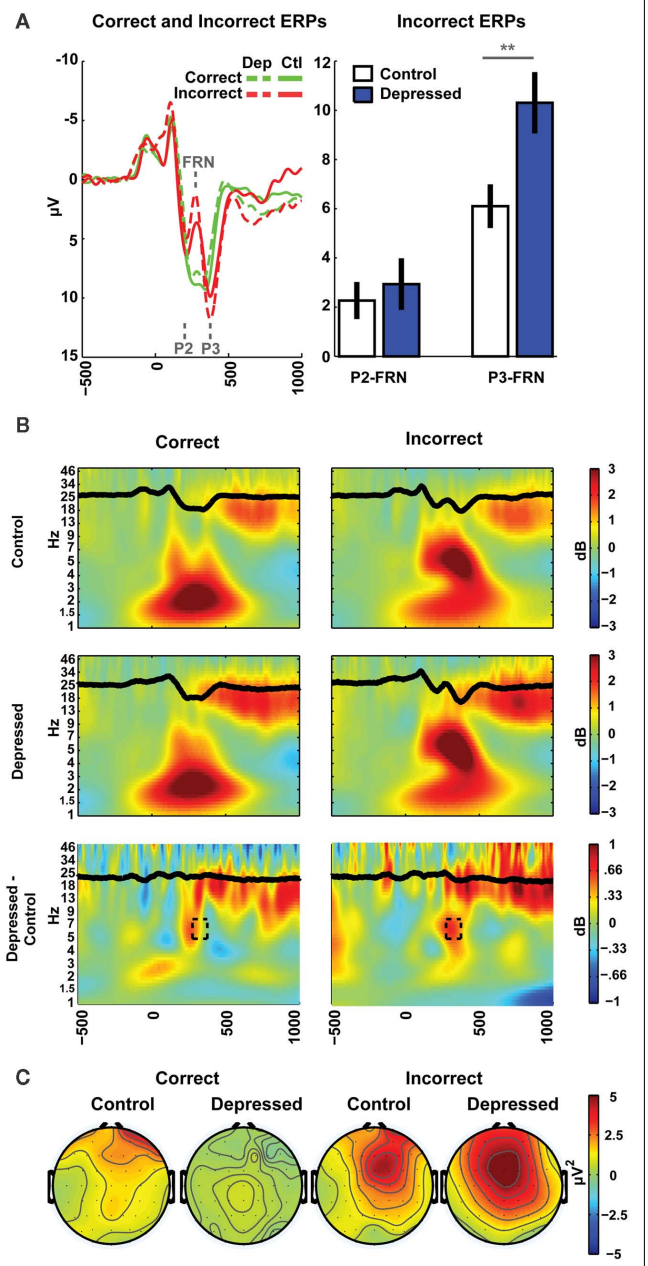

FIGURE 2 | EEG results. (A) The depressed group was characterized by a larger P3-FRN amplitude in the ERP to incorrect feedback. **(B)** Time–frequency plots show the theta band dynamics that occur following incorrect feedback (ERPs are superimposed in black). The time and frequencies of interest associated with the P3-FRN are identified by the dashed box in the difference plot. **(C)** Theta power during this time range showed a strong mid-frontal distribution for incorrect feedback.

investigations have favored a Gaussian width (σ) of $4.5/(2\pi f)$, here we utilize a width of $3/(2\pi f)$ to better resolve the temporally specific theta activities suggested by the ERP analyses.

One second of data was removed from each end of the transformed single trial EEG data (to account for edge effects) prior to averaging. Averaged power was normalized by conversion to a decibel (dB) scale (10*log10[power(t)/power(baseline)]) from a baseline of 300–200 ms, allowing a direct comparison of effects across frequency bands. Whereas the ERPs reflect phase-locked amplitude changes, these time–frequency measures reflect total power (phase-locked and phase-varying). As indicated by the topographic plots, and as in most other studies of these phenomena, values for statistical analysis were averaged over time and frequency at the FCz electrode (276–376 ms post feedback, 5–8 Hz), see **Figure 2B**. Topographic plots (**Figure 2C**) show theta power in this same time–frequency window, detailing a mid-frontal distribution peaking at FCz.

RESULTS

There were no group differences in any performance measures, including training or test phase accuracies or reaction times, immediate post-punishment adaptation, test phase accuracy for Go or NoGo, nor in high or low conflict variants of each valence (see **Table 1**). Importantly, **Table 1** also demonstrates that there were no group differences in the number of correct or incorrect feedbacks as evidenced by the EEG epoch counts. As shown in **Figure 2A**, there was a significant difference between groups in the P3-FRN amplitude of the ERP [$t(43) = 2.85, p < 0.01$], but not for the P2-FRN amplitude ($t < 1$). Error-related theta power in this P3-FRN time range did not significantly differ between groups [$t(43) = 1.3, p = 0.22$]. However, both P3-FRN amplitude and theta power predicted individual differences in NoGo accuracy ($rs > 0.34$, $ps < 0.05$), replicating previous findings (Frank et al., 2005, 2007a; Cavanagh et al., 2010a,b). BDI score did not significantly correlate with brain or behavioral variables within the depressed group with linear or quadratic fits.

The moderating effect of depression on this error-NoGo relationship was tested using repeated measures general linear models (GLMs) with NoGo accuracy as the dependent variable, and within-subjects factors for conflict (Low, High) and valence (Go, NoGo), a between-subjects factor for group (depressed, control) and a continuous moderator of theta power to incorrect feedback. Planned comparisons were first split by valence, then by conflict. As expected, group differences in the coupling between error signal theta power and avoidance learning were specific to high conflict NoGo cases [four-way interaction $F(1,41) = 8.7$, $p < 0.01$; three-way high conflict interaction $F(1,41) = 4.9, p < 0.05$, two-way high conflict NoGo interaction $F(1,41) = 5.1$, $p < 0.05$; all other interactions F's < 1.3, p's > 0.25]. Substituting P3-FRN amplitudes for theta power as a continuous moderator produced a similar two-way interaction for high conflict NoGo [$F(1,41) = 5.8$, $p < 0.05$; other two-way interactions Fs < 1] but higher-order statistical tests were non-significant. **Figure 3** demonstrates how error signal–avoidance learning coupling was specifically enhanced in MDD groups compared to control in high conflict NoGo conditions. As described by the GLM and indicated in **Figure 2**, the high conflict NoGo correlations were significantly different between the groups (Fisher's r to z test: $z = 2.37$, $p = 0.018$).

Table 1 | Group means, SD, and *t*-test results for demographics, task performance, and EEG epochs.

	Control Mean (SD)	Depressed Mean (SD)	*t*	*p*
Beck depression inventory (score)	1.62 (1.58)	21.71 (5.32)	−17.65	<0.001
Age (years)	19.21 (1.53)	18.86 (1.35)	0.81	0.42
Training RT (ms)	977 (305)	892 (170)	1.13	0.26
Training accuracy (%)	66 (9)	69 (11)	−0.77	0.45
Training lose-switch (%)	45 (8)	42 (11)	1.14	0.26
Training lose-switch/all switch (%)	85 (29)	98 (49)	−1.13	0.26
Training post-punishment RT (ms)	994 (320)	914 (180)	1.01	0.32
Training post-punish/post-Cor RT (%)	1.02 (0.06)	1.05 (0.10)	−0.69	0.50
Test RT (ms)	1152 (421)	1036 (317)	1.02	0.31
Test accuracy (%)	66 (10)	70 (10)	−1.41	0.17
Test go–nogo "bias" (%)	2 (26)	1 (14)	0.21	0.83
Test go accuracy (%)	68 (16)	71 (14)	−0.65	0.52
Test hi conflict go accuracy (%)	59 (17)	57 (14)	0.54	0.59
Test lo conflict go accuracy (%)	74 (17)	79 (14)	−1.08	0.29
Test nogo accuracy (%)	66 (17)	70 (15)	−0.89	0.38
Test hi conflict nogo accuracy (%)	61 (14)	61 (14)	0.03	0.97
Test lo conflict nogo accuracy (%)	71 (22)	81 (17)	−1.74	0.09
Correct trial EEG Epochs (count)	233 (124)	183 (109)	1.41	0.16
Incorrect trial EEG Epochs (count)	187 (103)	154 (99)	1.1	0.28

DISCUSSION

Numerous investigations have shown that larger error signals predict better avoidance learning, and the present report reveals that this relationship is enhanced among non-medicated depressed participants. This mood-related effect was specific to high conflict lose–lose cases, revealing the specificity of increased error signals in depression on avoidance learning.

RELATIONSHIP TO PREVIOUS INVESTIGATIONS

In the current investigation, the MDD group was characterized by larger feedback-locked error signals and enhanced error signal–avoidance coupling, yet these occurred in the context of similar behavioral performance to controls. A similarly powered study of depressed patients recently reported null results for behavioral measures of punishment adaptation and NoGo learning in this same task (Chase et al., 2010). The lack of behavioral effects are convergent with those reported here, indicating that depression-specific effects on the link between brain error

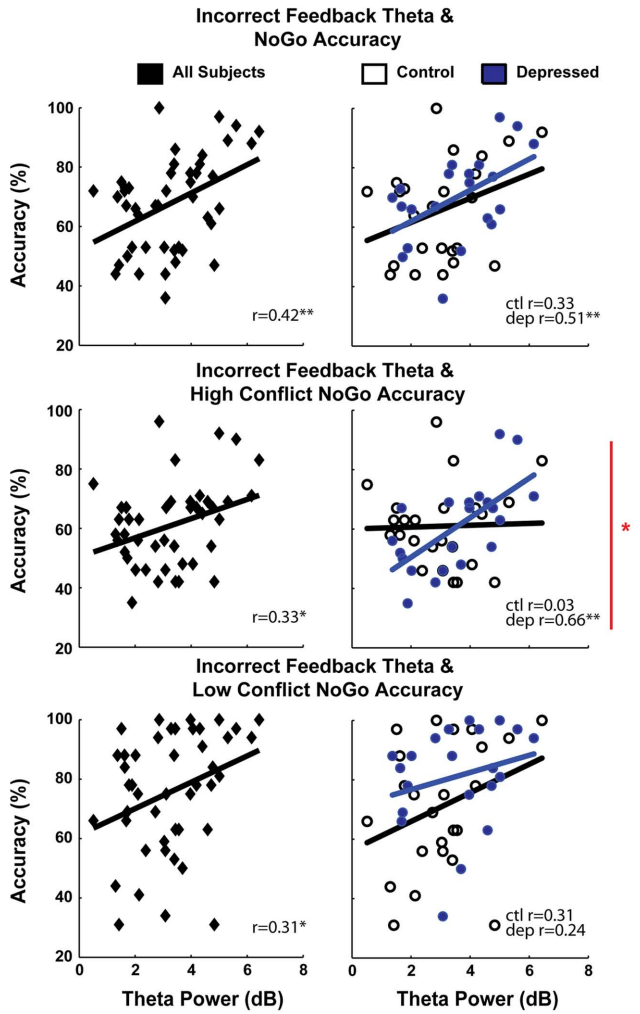

FIGURE 3 | Scatterplots demonstrating error signal–avoidance learning relationships, along with correlation test results. The first row shows total NoGo accuracy, which can be split into high and low conflict cases (rows 2 and 3). Only in the high conflict "lose–lose" cases did controls and depressed participants significantly differ from each other, demonstrating the specificity of increased error signal–avoidance learning acuity among those with depression.

monitoring systems and performance are critical variables for understanding learning-related changes.

While larger error signals have been previously been found in MDD participants (Tucker et al., 2003; Chiu and Deldin, 2007; Holmes and Pizzagalli, 2008, 2010), many other studies report complicated patterns and divergent contrasts (Ruchsow et al., 2005; Compton et al., 2008; Schrijvers et al., 2008, 2009; Olvet et al., 2010; Georgiadi et al., 2011) in addition to compromised post-error adaptation (Elliott et al., 1996; Pizzagalli et al., 2006; Holmes and Pizzagalli, 2007; Compton et al., 2008). These complexities suggest that in order to successfully interpret the meaning of altered error-related signals, it may be critical to understand how these signals are (or are not) being used for behavioral adaptation.

DEPRESSION, ERRORS, AND AVERSIVE LEARNING: WHAT DOES IT ALL MEAN?

Avoidance learning acuity is proposed to be reflected by NoGo behavioral accuracy, and high conflict choices reflect two outcomes that are hard to distinguish. Thus, high conflict NoGo trials all consist of "lose–lose" forced choice decisions. Notably the mood-related effect reported here was specific to these lose–lose cases, revealing the specificity of increased error signals in depression on avoidance learning. A mechanistic explanation of this effect may be that an increased salience of error signals is related to larger or more extended pauses in tonic dopamine release.

The temporal specificity of the enhanced error signals in the MDD group support this interpretation. While earlier stages of feedback evaluation have been associated with valence-specific differences, MDD-related modulation of later stages may be associated with an enhanced prediction error magnitude (Philiastides et al., 2010). A neural network model of cortico-striatal function in this same task suggests that a larger negative prediction error would cause a larger/longer dopamine dip, which would increase learning for stimulus–action combinations in the D2 receptor mediated indirect cortico-striatal pathway, contributing to a tendency to not make this action again (Frank, 2005). This effect would be behaviorally reflected by particularly increased accuracy in lose–lose choices, which are most sensitive to individual differences in the ability to resolve subtly different probabilities of negative events.

The finding reported here suggests an error-related mechanism by which punishment hypersensitivity may be related to affective and behavioral distress. We previously proposed that an affect-related increase in mid-frontal error signals and avoidance learning reflected a cortical bias on the integration of action values (Cavanagh et al., 2010a). Using the exact same task and methods, our prior study of social threat found that emotional reactivity to stress predicted an increase in mid-frontal theta and a related increase in high conflict NoGo learning amongst highly punishment sensitive participants (Cavanagh et al., 2010a). Note that increased high conflict NoGo learning accuracy in the context of increased mid-frontal theta was paralleled between highly stress-reactive participants in the prior investigation and the depressed participants reported in this investigation. The similarities between the previous and current studies warrant comparison, as they may provide a window into the processes underlying these common effects: affect-modulated mPFC activities may bias mood-congruent learning.

CONCLUSION

An integrative explanation of the findings and possible mechanisms reported here focuses on the fact that the mPFC is involved in cognitive control, affective reactivity, and the adaptation of behavior to reinforcement. It is likely no coincidence that this system is intimately implicated in the etiology of MDD. The combined activities of this particular cortico-striatal system identify it as a focal node by which emotion may be internalized to affect cognitive functioning. In this investigation, we have identified a measure of how, and a possible mechanism by which, negatively valenced information is internalized in the genesis and expression

of MDD: error and punishment signals are increasingly coupled with the salience of "bad" outcomes.

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Positive affect versus reward: emotional and motivational influences on cognitive control

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It is becoming increasingly appreciated that affective influences can contribute strongly to goal-oriented cognition and behavior. However, much work is still needed to properly characterize these influences and the mechanisms by which they contribute to cognitive processing. An important question concerns the nature of emotional manipulations (i.e., direct induction of affectively valenced subjective experience) versus motivational manipulations (e.g., delivery of performance-contingent rewards and punishments) and their impact on cognitive control. Empirical evidence suggests that both kinds of manipulations can influence cognitive control in a systematic fashion, but investigations of both have largely been conducted independently of one another. Likewise, some theoretical accounts suggest that emotion and motivation may modulate cognitive control via common neural mechanisms, while others suggest the possibility of dissociable influences. Here, we provide an analysis and synthesis of these various accounts, suggesting potentially fruitful new research directions to test competing hypotheses.

Keywords: emotion, motivation, cognitive control, reward, dopamine

INTRODUCTION

Human nature is uniquely characterized by the flexibility, complexity, and sophistication with which thought and behavior can be deployed in the service of a goal. This ability is thought to depend on *cognitive control*, a collection of mechanisms, including perceptual selection, response biasing, and online maintenance of contextual or goal information, by which the human cognitive system adaptively configures itself to optimally perform specific tasks (Miller and Cohen, 2001; Braver et al., 2002). Most of the goals pursued in daily life are emotionally or motivationally meaningful – i.e., to obtain outcomes that are pleasurable or important for survival, and avoid outcomes that are not. It has long been understood that such affective significance is central to determining the goals around which human behavior is organized; indeed, impairments in affectively driven goal-pursuit may be a critical component of a number of psychiatric disorders, such as depression and schizophrenia (Pessoa, 2008). Consequently, the psychological and neurobiological mechanisms by which affective influences modulate cognitive control have become of major interest in recent years and continue to be an important emerging topic of study.

Much of the experimental research examining how affect modulates cognitive control has involved one of two types of manipulations: emotional manipulations, in which affectively valenced subjective experience is directly induced (e.g., through mood inductions or exposure to emotional stimuli), or motivational manipulations, where motivational state is altered through the introduction of rewarding or punishing incentives. Both types of manipulations are thought to carry affective significance, and both have been hypothesized to impact goal-pursuit and/or cognitive control. However, for the most part, these bodies of research have been carried out independently of one another. In a recent review, Pessoa aimed to ameliorate this situation by considering examples of both an emotional manipulation (threat) and a motivational manipulation (reward) on cognition within a common conceptual framework (Pessoa, 2009). His review suggests that both threat and reward operate in highly similar ways, impacting cognitive performance at both perceptual and executive stages of information processing. However, Pessoa acknowledges that emotion and motivation are broad constructs, the impacts of which may not be comprehensively characterized by the phenomena of threat and reward alone. In contrast, and as described further below, other theories of emotion and motivation suggest the possibility of dissociations between the two constructs (e.g., "liking" versus "wanting"; Berridge, 1996) but this remains an understudied issue. Thus, the goal of the present paper is to discuss more explicitly existing theoretical accounts regarding the relationship of emotion and motivation to cognitive control, examine how they may relate to one another, and speculate on commonalities and differences in the mechanisms by which they operate. We also suggest future research directions that could be pursued to clarify ambiguity regarding the emotion versus motivation distinction.

EMOTION AND MOTIVATION: TERMINOLOGY AND CONCEPTUALIZATION

Emotion and motivation are highly related constructs within the domain of affect (Rolls, 2000; Lang and Bradley, 2008), but their influences on cognition generally have not been explicitly considered in relation to one another. When examining the literature regarding the impact of each on cognitive performance, it is important to provide working definitions of relevant terms, so as to begin more carefully examining how these constructs may relate to one another.

One review suggests that emotions are best functionally defined as "psychological or physiological states that index occurrences of value" (Dolan, 2002). As this description suggests, emotion is generally conceptualized as a construct that can be decomposed into multiple subcomponents defining the relation between individual and environment. Davidson et al. (1990) suggest that emotions are comprised of three elements: autonomic reactions, cognitions, and behaviors. More recently, Roseman (2008) asserts that emotion can be thought of as a syndrome of *phenomenology* (thought and feeling qualities), *physiology* (neural, chemical, and other physical responses in the brain and body), *expressions* (signs of emotion state), *behaviors* (action tendencies or readinesses), and *emotivations* (characteristic goals that people want to attain when the emotion is experienced). Gendron and Barrett (2009) similarly claim that emotions are comprised of subprocesses, including an affective and cognitive (e.g., situational construal) component, and are highly contextualized in nature. Common to all of these definitions is the idea that emotions are an affective experience that can be characterized by physiological changes and defined by a cognitive construal of some kind. As states indexing occurrences of value, emotions have been proposed to carry functional value in physiologically preparing the body for action, permitting flexibility of behavioral responses to reinforcing stimuli, facilitating communication and social bonding, and influencing cognitive processes including evaluation, memory encoding, and memory recall (Rolls, 2000).

Motivations are similar to emotions in that they also serve to define the relation between the individual and the environment (Roseman, 2008), but differ from emotions in being more tightly linked to action and explicit goal associations; motivated action can be thought of as behavior that is at least partly determined by a desired and hedonically laden end-state (i.e., it is goal-directed). Pessoa (2009) suggests that motivation can be commonly defined as what makes one work to obtain reward or to avoid punishment. Similarly, Roseman (2008) proposes that a motivation is an internal state producing behavior which moves the individual toward desirable reference values or away from undesirable reference values.

Carver suggests a useful distinction between the two constructs as they relate to goals: while motivation may be the drive toward goal fulfillment, emotion may be emergent from one's sensed *rate of progress* toward goals; the difference between one's present status and one's goal state is experienced as affect and may lead to goal reprioritization in order to maximize goal fulfillment (Carver, 2006). Similarly, Rolls (2000) suggests that emotions are states elicited by rewarding and punishing reinforcers of behavior. Likewise, Lang and Bradley (2008) claim appetitive and defense-related brain circuits have evolved to cope with motivationally significant stimuli in the environment; positive and negative emotion, respectively, are associated with the experience of these brain circuits being activated. Thus, according to this general view, emotion can be considered an emergent property of motivationally driven neural activity. However, the Lang and Bradley view also suggests that emotion is highly characterized by hedonic experience, which accordingly is also tied to the activation of motivational neural circuitry. They postulate that "...evaluative reports of *pleasure/displeasure* roughly index which motivational system is activated by a stimulus event (i.e., appetitive or defensive)" (Lang and Bradley, 2008). Buck (2000) and Laming (2000), commenting on Rolls' (2000) review of emotion and motivation, argue that emotion and motivation cannot be considered separately of one another: Laming (2000) argues that emotion is the subjective experience of being motivated, thus there is no separation between the two; Buck argues that motivation and emotion cannot be distinguished from one another if emotion is, as he describes, the "manifestation or 'read-out' of motivated potential" (Buck, 2000).

A contrasting perspective can be drawn from the work of Kent Berridge, which has highlighted the potential dissociation between activation of motivational circuitry and the neural systems that code for hedonic experience (Berridge, 1996, 2003; Berridge and Robinson, 1998, 2003; Berridge et al., 2009). He proposes that the hedonic (i.e., subjective experiences of pleasure/displeasure) and motivational (i.e., attribution of incentive salience) facets of reward, shorthanded as "liking" and "wanting" respectively, are neurobiologically dissociable: evidence from rodents indicates that hedonic activation may depend on opioid-related circuitry while attribution of incentive salience may depend on the mesolimbic and neostriatal dopamine (DA) systems. This work suggests that the constructs of emotion and motivation might involve separable neural mechanisms, and as such may have distinct influences on cognitive processing.

Psychological accounts postulating theoretical distinctions between emotion and motivation have been less common. However, Roseman (2008) has recently suggested key differences: while both may lead to goal-directed action, he proposes several differences between emotion and motivation. Roseman argues that motivations are specific, relatively deliberate, and associated with a specific goal. In contrast, emotions are produced by multiple contingencies, are somewhat more impulsive, and are not tightly linked to a particular goal. Additionally, he suggests that emotions typically take precedence over motivations: specifically, by engendering *emotivations*, emotion-specific motivations, that take precedence over non-emotional motivations. These emotivations could potentially be understood (in terms of Carver's conceptualization of emotion) as a manifestation of goal reprioritization resulting from emotion as an indicator of motivational status.

From these working definitions and theoretical accounts of the relationship between emotion and motivation, we suggest that an emotion may be presently considered a construct of multiple processes that together serve to provide an index of value associated with an internal or externally experienced state. While a motivation may be similarly comprised of multiple components, a motivation should be considered a state that produces behavior specifically oriented to carry out a goal that has hedonic value. Thus, whereas an emotion may emerge from one's status relative to motivational goals, it may not necessarily be directly relevant to a particular goal.

Examining the influences of emotion and motivation on cognitive performance may be fundamental to clarifying the relation between these constructs; currently, however, these investigations have been conducted largely in parallel. The goal of this paper is to integrate these literatures by highlighting some key theoretical accounts of emotional and motivational influences on cognition, and illustrating where empirical evidence suggests these influences

may diverge. To facilitate comparison between the emotional and motivational literatures, we have chosen to restrict the focus to studies involving positive emotions and reward incentives. It is, of course, also crucial to explore the relationship between negative emotions and punishment/avoidance-based motivational states, but as positive and negative emotion may be independent of one another (Watson et al., 1988), the extent to which they share the same mechanisms on cognition remains unclear.

POSITIVE AFFECT AND COGNITIVE CONTROL

Gray and Braver (2002) postulate that investigations of emotional influences on cognitive control should fulfill two global aims. The first is to determine whether emotional influences can and do have a selective influence on cognitive control: this must be established first and independently of the nature of these influences. The second aim is to elucidate the mechanisms by which such influences operate. In the present section, we discuss psychological theories regarding the adaptive value of positive emotion on cognition as well as theories regarding the specific mechanisms by which positive emotion takes its effect.

It has been suggested that positive emotion might be an adaptive signal indicating safety and security in the environment, giving the organism the freedom to explore and engage in new opportunities (Fredrickson, 2004). Building on this postulation, several psychological theories have suggested that positive affect serves to broaden cognition, promote creative problem-solving, and improve cognitive flexibility. Foundational work in this area was conducted by Isen and Daubman (1984), who observed that positive affect induction led to broader categorization and facilitated creative problem-solving (Isen et al., 1987). Relatedly, Fredrickson (2004) proposed the *broaden-and-build theory of positive emotions*, which posits that positive emotions broaden one's repertoire of thought and action, promoting building of intellectual, social, and psychological resources. In the cognitive domain, empirical support for the broaden-and-build theory has come from visual processing and semantic association tasks suggesting a broader scope of attention (Fredrickson and Branigan, 2005). Dovetailing nicely with this work is Carver's (2003) "coasting hypothesis," which suggests that the security of positive affect might emerge from one's sensed rate of progress toward goals, and result from goal completion at a faster rate than anticipated. Under such circumstances, one is free to "coast" on the goal in question and consider new ideas and/or the pursuit of other goals (leading to changes in goal prioritization).

The neural mechanisms underlying positive emotion's effects on cognition remain unclear. Different theories have been posited to explain these effects. One influential theory, the *dopaminergic theory of positive affect* (Ashby et al., 1999) was developed to address findings that positive emotion is linked to broadened cognition. Ashby and colleagues extrapolated from the literature on the neural substrates of reward processing to propose that the psychological effects of positive emotion are specifically linked to increased dopamine (DA) release (via the substantia nigra and ventral tegmental area) in these states. The particular cognitive effects of increased DA release during positive affect were postulated to occur through mesocorticolimbic system projections to the anterior cingulate cortex (ACC) and substantia nigra projections to striatum, with increased DA facilitating the ability of ACC and striatum to initiate a switch among active task sets, rules, or goal representations maintained in lateral prefrontal cortex (PFC). This facilitation of switching among task-set representations under positive affect enables unusual or non-dominant sets to become active with a greater probability than under neutral affect conditions, which then facilitates creative problem-solving. In connectionist simulations, the account was tested and exhibited an ability to account for certain behavioral performance patterns observed by Isen and colleagues under positive affect manipulations (i.e., improved performance on creative problem-solving and semantic association tasks; Ashby et al., 1999, 2002).

Dreisbach and Goschke (2004), Dreisbach (2006), and Muller et al. (2007) developed a related theoretical framework, which emphasizes that the cognitive flexibility associated with positive affect may have systematic costs *in addition to* the benefits posited by broadening theories. Specifically, Dreisbach proposed that changes in dopamine activity triggered by positive affect lead to a shift in the balance between cognitive stability and cognitive flexibility, by increasing the tendency to update to new task goal representations and decreasing the tendency to perseverate in maintaining old ones. Empirical evidence from performance in set-shifting and context maintenance paradigms was consistent with this hypothesis, demonstrating positive affect induced facilitation of performance under conditions that depended on flexibility, but impairment under conditions stressing maintenance (Dreisbach and Goschke, 2004; Dreisbach, 2006).

A separate theoretical account, put forward by Gray (2001) and Gray and Braver (2002), argued for a hemispherically specialized basis of interactions between positive affect and cognitive control. In this account, an important congruence is noted between prior affective research associating positive emotions with increased activity in the left frontal cortex (Davidson et al., 1990; Davidson, 1992; Davidson and Irwin, 1999; Canli et al., 2001), and cognitive research linking the left frontal cortex to the active maintenance of verbal information in working memory (D'Esposito et al., 1998; Smith and Jonides, 1998). The primary theoretical claim of the account is that different affective states (positive versus negative) should trigger associated behavioral goals in working memory, and that it is adaptive for these goals to be hemispherically segregated such that they can be selectively prioritized by the appropriate affective state. Thus, the theory postulates that positive affect states should selectively facilitate verbal working memory – a hypothesis that was confirmed experimentally (Gray, 2001). Nevertheless, this account is agnostic about why positive and negative affect would be selectively linked with particular stimulus modalities in working memory.

In summary, theoretical accounts of the effect of positive emotion on cognition have tended to emphasize influences on cognitive flexibility, potentially by enhancing updating of goal information in working memory. Although most work has emphasized the adaptive value of such influences, it has also been suggested to come at a cost to goal maintenance. At the level of neural mechanisms, the focus has been on the dopamine system and PFC, which, as is discussed next, has strong parallels to theoretical accounts regarding how motivation might modulate cognitive control.

REWARD MOTIVATION AND COGNITIVE CONTROL

Theoretical accounts of motivation suggest a strong linkage to cognitive control (Simon, 1967; Carver and Scheier, 1998; Kruglanski et al., 2002). Although early motivational theories suggested a general drive or energization function for motivation (Miller, 1951; Hull, 1952; Duffy, 1962), information processing accounts of higher-level cognition have instead emphasized that motivational signals may play a more focused role in the prioritization, updating, and termination of goal representations that provide hierarchical control of behavior (Simon, 1967). Over the last 20 years, the intrinsic relationship between motivation and goals has been a central focus of researchers primarily working within the social and individual differences tradition, based on the central claim that motivations are expressed primarily as the activation and representation of specific cognitive and behavioral goals over others (Kruglanski et al., 2002). More recent work has been geared toward demonstrating that goal-directed behavior can be primed and biased by implicit and/or subliminal motivational cues, suggesting a relatively direct route for motivation–cognition interactions (Bargh et al., 2001; Custers and Aarts, 2010). In particular, Aarts et al. (2008b) have suggested an affective/motivational account of goal-pursuit in which positive motivational signals strengthen goal activation and maintenance, even when this occurs outside of conscious awareness. More recently, psychological theories postulating the role of motivation in activating goals and guiding behavior have begun to be bridged with neuroscience-based studies to more clearly specify the mechanisms by which motivation might influence cognitive control.

A primary focus of neuroscience studies on motivation and cognitive control has been to demonstrate that these two processes are integrated within specific brain regions, such as the lateral PFC. Early work involving single-unit recording in primates demonstrated that task-related neuronal activity in PFC was modulated by the expected reward value associated with performance (Watanabe, 1996; Leon and Shadlen, 1999; Watanabe et al., 2002). In one compelling demonstration, it was found that reward value directly enhanced the fidelity of active maintenance in working memory (Leon and Shadlen, 1999). More recent fMRI studies carried out in humans have used designs that orthogonally manipulate cognitive control demand and motivational value across a range of task domains, including working memory (Pochon et al., 2002; Taylor et al., 2004), context processing (Locke and Braver, 2008; Kouneiher et al., 2009), task-switching (Savine and Braver, 2010), and selective attention (Padmala and Pessoa, 2011). These studies have confirmed the presence of specific regions within lateral PFC (along with effects in other associated regions, such as the ACC) that are sensitive to the interaction of the two factors, consistent with a specific role in integrating motivational and cognitive control functions.

The DA system also plays a central role in accounts of both motivation and cognitive control. Dopamine has long been thought to be a critical component of motivation and reward processing (Wise and Rompre, 1989; Mirenowicz and Schultz, 1996; Robbins and Everitt, 1996; Schultz, 1998). More recent accounts have suggested that DA shows phasic, cue-triggered responses to specific events that indicate reward availability (Montague et al., 1996; Schultz et al., 1997) and/or high motivational salience (Berridge, 2007). This signal, particularly when a reward is different from anticipated (i.e., prediction error), may serve as a mechanism for reward-based associative learning (Schultz et al., 1997; Schultz, 2002; Arias-Carrion and Poppel, 2007). While the role of dopamine as a learning versus salience signal in reward has been debated (Berridge, 2007), both kinds of accounts are compatible with the idea of phasic DA involvement in processing motivational incentives and thus consistent with our account. Additionally, a separate theoretical account has emphasized that the motivational utility of the current environmental context might be reflected in tonic, rather than phasic, DA activation (Niv et al., 2007). Together, these accounts suggest DA activity will be increased both by transient cues and sustained contexts that indicate high reward or motivational value.

It is worth noting a completely separate literature focused on the influence of DA release within PFC, which suggests that the DA system provides modulatory role on cognitive control functions. Neurophysiological studies in primates show that application of DA into PFC sharpens actively maintained stimulus representations (Sawaguchi et al., 1988; Sawaguchi and Goldman-Rakic, 1991; Arnsten et al., 1994). In contrast, DA antagonists reduce both active maintenance related PFC activity, and also cause behavioral impairments in working memory and cognitive control tasks (Sawaguchi et al., 1990; Sawaguchi and Goldman-Rakic, 1994; Williams and Goldman-Rakic, 1995). Similar effects have been observed in human pharmacological and fMRI studies, with DA agonists (administered systemically) being associated with improvements in working memory and cognitive control, and leading to associated modulations of PFC activity (Kimberg et al., 1997; Gibbs and D'Esposito, 2006). It is striking that the effects of pharmacological manipulations of DA in PFC are so similar to the effects that have been observed from motivational manipulations (Leon and Shadlen, 1999; Watanabe et al., 2002).

These linkages between the role of DA and PFC in motivation, and the effects of DA modulation on PFC-mediated cognitive control functions have prompted the development of theories that explicitly link these two mechanisms. The gating model account, put forward by Braver and Cohen (2000), emphasized the importance of phasic DA activity within PFC for the updating and active maintenance of goal representations. Specifically, this account suggests that the phasic DA responses to cues signaling reward prediction could also be exploited as a means of learning which task-related information should be actively maintained in PFC, and when to update such information. Simulation studies demonstrated that a system could in fact learn appropriate updating and maintenance of task context or goal information based on reward prediction cues. Thus, this account suggests the possibility of a linkage between the reward/motivational and cognitive control functions mediated by DA–PFC interactions. This point was made even more explicitly in the recent Dual Mechanisms of Control framework (Braver et al., 2007), which specifically suggests that signals of reward motivation will bias cognitive control toward a "proactive" mode, in which task cues trigger sustained goal activation and maintenance in the service of preparation for anticipated control demands, via the aforementioned DA–PFC interaction. Proactive control is distinguished from reactive control, in which the same task cues only trigger transient control related processes,

rather than sustained active maintenance, under conditions of low DA activity, such as when the environmental context is perceived to have reduced motivational value. This account has been supported by recent experimental evidence from an fMRI study of motivational influences on task-switching (Savine and Braver, 2010). Task trials that had high reward value were associated within increased activity in both lateral PFC and the midbrain DA system. Moreover, the increased cue-related PFC activation on high reward trials was associated with behavioral measures of improved task preparation, supporting the idea of a reward-related shift toward proactive control.

In summary, investigations of motivation and cognitive control have primarily focused on the role of goal representations in psychological models, and on interactions of the DA system in PFC in neuroscience studies. One mechanistic account suggests that DA activity triggered by reward cues can serve as an updating and prioritization signal, that modulates active maintenance of goal information within PFC. The potential similarity of the proposed mechanisms underlying both positive affect and reward motivation influences on cognitive control, according to the Ashby model (i.e., cognitive changes due to increased dopamine release to cortical areas such as the PFC and ACC), is striking, but has not yet been directly confirmed by experimental studies. This issue is discussed next, followed by suggestions for promising future research directions.

EMOTION VERSUS MOTIVATION: COMMON OR DISSOCIABLE INFLUENCES?

Emotion and motivation are closely related constructs, but it is still not clear how to relate their influences on cognitive control. Some theories do not clearly distinguish between the two, while others more explicitly state that emotion and motivation, while highly related, remain distinct constructs. Given the state of current evidence, this is still a question calling for clarification. Hints of dissociation between the two serve as starting points for future research.

Most proposals regarding affective influences on cognitive control have not drawn a clean distinction between emotion and motivation. This includes Gray's (2001) hemispheric specialization hypothesis, which explicitly does not distinguish between positive/negative emotion and approach/avoidance motivation, and Ashby et al.'s (1999) dopaminergic theory of positive affect, which posits a relation between DA and positive emotion *because* of DA's involvement in reward processing. Aarts et al. (2008b) go even further, by explicitly suggesting an equivalence between positive emotion and reward motivation in terms of their effects on goal-pursuit and executive control. In their model, positive affective signals occurring in temporal proximity to activation of a cognitive goal should strengthen the maintenance, accessibility, and pursuit of that goal, regardless of whether those affective signals relate to goal attainment (i.e., whether or not they serve as direct reward motivation cues).

Despite these claims, the relationship between positive affect, approach motivation, and DA system activity is somewhat tenuous. Experimental studies have shown that affective valence (positive/negative) and motivational direction (approach/avoidance) can be dissociated (Harmon-Jones, 2003; Harmon-Jones and Gable, 2009). Likewise, in Carver's (2006) theoretical model, positive and negative affect emerge from detection of the rate of progress toward fulfilling *either* approach or avoidance-related goals: positive emotions result from above-anticipated goal progress; negative emotions result from below-anticipated goal progress. Thus, positive affect could potentially arise while pursuing avoidance-motivational goals. Finally, in Berridge's neuroscience-based account, dopamine is only critical for the transfer of motivational salience, or stimulus-triggered "wanting" to new cues or events, and does not generate hedonic experience, or "liking" (which is thought to be represented in the ventral pallidum via neurochemicals such as opiates and endocannabinoids; Berridge, 2007; Berridge et al., 2009). Thus, without supportive direct evidence, links between positive affect, approach motivation, and DA system activity should not be assumed.

Deconfounding emotional and motivational influences from one another is a challenge for exploring and clarifying these relationships. We argue that, as a starting point, emotional and motivational manipulations need to be operationalized (i.e., with exposure to emotional stimuli during or just prior to the task, or motivational performance-contingent incentives, respectively) and examined with performance-independent measures. Examining psychophysiological signatures of these influences [e.g., startle reflex, facial electromyography (EMG)] that have been linked to valence but not motivational orientation of affective experiences (Cacioppo et al., 1986; Lang et al., 1990) may be useful in dissociating these influences from one another.

Although these influences have yet to be directly disentangled, independent empirical work from the emotion and motivation literatures indirectly hints that these are dissociable influences. While several theories have proposed that positive emotion can lead to cognitive broadening and flexibility (Isen et al., 1987; Ashby et al., 1999; Fredrickson, 2004; Rowe et al., 2007) and exploration of alternate goals (Carver, 2003), growing evidence suggests that reward incentives enhance goal maintenance/representation and influence proactive cognitive control (Savine and Braver, 2010; Padmala and Pessoa, 2011). Empirical evidence is mixed, suggesting that positive emotion and reward may have similar effects on some cognitive processes, such as task-switching (reducing switch costs; Yan-Mei and De-Jun, 2008; Savine et al., 2010), opposite effects on other processes, such as selective attention (Rowe et al., 2007; Padmala and Pessoa, 2011) and mixed results on yet other processes such as goal maintenance (Dreisbach and Goschke, 2004; Dreisbach, 2006; Aarts et al., 2008b; Locke and Braver, 2008). How these diverging effects relate has yet to be systematically clarified, since positive affect and reward motivation have not yet been directly examined and compared.

Nevertheless, there is at least one example from the literature in which the same experimental paradigm – the AX continuous performance task (AX-CPT; Cohen and Servan-Schreiber, 1992; Servan-Schreiber et al., 1996; Braver et al., 2001) – has been utilized (in separate studies) to explore the effects of positive affect and reward motivation on cognitive control. The AX-CPT is a potentially advantageous paradigm for this purpose, because it permits selective examination of goal maintenance capability and proactive control. However, surprisingly, the results of the positive affect and reward motivation studies appear to be somewhat different. Under

positive affect induction via valenced pictures displayed prior to each trial, participants showed evidence of reduced maintenance capability relative to neutral affect (Dreisbach, 2006). Conversely, under manipulations of reward motivation, participants showed evidence of enhanced maintenance capability relative to baseline (Locke and Braver, 2008). In both studies, the results were interpreted as arising from modulation of DA activity in PFC, but it is not clear if the two different manipulations actually led to similar or distinct effects within these brain systems. Thus, direct comparisons are clearly needed, with monitoring of neural activity, to determine whether there is a potential dissociation between positive affect and reward motivation effects on goal maintenance.

One possibility that is worth considering is that DA activity in PFC underlies both positive affect-related reduction in maintenance capability and reward-related increases in maintenance capability, but that the two effects reflect dissociable temporal dynamics of DA influence. In particular, it is well-accepted that DA activity should be considered in terms of both tonic and phasic components, which can interact with each other (Grace, 1991). Further, prior research has more strongly linked facilitation of goal maintenance with tonic DA activation in PFC, primarily via D1 receptors; in contrast, phasic DA activity in PFC may promote updating and cognitive flexibility, potentially via D2 receptors (Cohen et al., 2002; O'Reilly and Frank, 2006; Durstewitz and Seamans, 2008). Thus, distinctions between reward motivation and positive affect may reflect a distinction in the balance between tonic versus phasic DA activation and/or a D1 versus D2 dominated state. However, the relationship between emotional and motivational processes and differential temporal dynamics of DA activity has not been directly shown and has yet to be investigated.

DIRECTIONS FOR FUTURE RESEARCH

The constructs of emotion and motivation are closely interrelated, and typically their influences on cognition have not been explicitly separated from one another (e.g., Pessoa, 2009). However, in the light of accumulating evidence suggesting they may be dissociable, their influences on cognition must be clarified. As alluded to above, there are unresolved questions in this domain that provide promising routes for future investigation. Here we discuss some of these in greater detail.

A first, straightforward suggestion is for studies to be conducted that directly test and compare the effects of positive affect manipulations with those involving reward motivation within a single sample, and using closely matched experimental designs. Of course, this suggestion does beg the question of what exactly differentiates a positive affect manipulation from one involving reward motivation, especially since some studies advertised as examining positive affect have actually used manipulations involving delivery of rewards (Isen et al., 1987; van Steenbergen et al., 2009). Although this question is one that may involve a deeper discussion that is outside of the scope of this article, we want to highlight that this issue has not really been adequately considered by researchers working in this area. Nevertheless, it is at least possible to operationalize a distinction in which positive affect is manipulated by influences such as mood inductions or strongly valenced stimuli, whereas reward motivation is manipulated by varying the incentives provided for task-performance.

With such an approach, it would be possible to directly test the hypothesis that positive emotion promotes cognitive flexibility while reward incentives promote greater goal maintenance. As mentioned previously, cognitive paradigms that specifically probe active maintenance and proactive control, such as the AX-CPT, could be used with emotional and motivational experimental manipulations to probe the possibility of dissociable versus common behavioral effects and/or their associated physiological bases. Other paradigms should also be examined, such as the remote associates and global/local paradigms, that have been frequently and fruitfully examined to demonstrate increased cognitive flexibility under positive affect (Isen and Daubman, 1984; Fredrickson and Branigan, 2005; Rowe et al., 2007), but have not previously been studied with respect to reward motivation. Finally, the use of a common paradigm would be useful for exploring other hypotheses relating to positive affect and/or reward motivation such as Carver's coasting model. Unfortunately, this model has not previously been studied within cognitive experimental paradigms, so further work in how to operationalize maintained goal-pursuit versus goal-switching would need to occur (e.g., via exploration of potentially approach paradigms such as volitional task-switching).

A second promising direction for future research is a more direct examination of the role of neurotransmitter systems in mediating affective and motivational influences on cognitive control. This is especially important with regards to the effects of positive emotion on cognition, since theories such as Ashby's posit a neurotransmitter mechanism without direct evidence for its involvement. Study of the role of neurotransmitter systems in positive affect is somewhat challenging, since detecting positive affect in animal models is difficult (although potentially not impossible, e.g., Berridge, 2000), as is direct monitoring of neurotransmitter activity in humans. However, studies that examine state-related changes in neurotransmitter receptor binding (e.g., using PET radioligand-labeling methods and dynamic imaging approaches) do provide a promising avenue for this research, especially for the monitoring of DA system activation (Egerton et al., 2009). In particular, receptor binding studies may provide a powerful means of directly testing whether increases in positive affect are associated with increases in DA system activity. Pharmacological manipulations provide another method by which to examine the link between neurotransmitter activity and cognitive change. Pharmacological challenge studies involving agents that influence DA activity have been used fruitfully in a range of domains to understand the role of this system in cognitive functions such as working memory (e.g., Cools et al., 2007), as well as in mediating basic aspects of reward processing (e.g., Pessiglione et al., 2006). Thus, targeted studies are needed that directly investigate how pharmacological manipulation of this system impacts motivational versus affective effects on cognitive control.

Although dopamine has received much attention for its involvement in reward and cognitive control processes, other neurotransmitters such as norepinephrine (NE) may also play crucial roles. NE activity, historically linked to physiological arousal (Foote et al., 1980), is becoming increasingly appreciated as a complex and specific mediator in the control of behavior as well (Aston-Jones and Cohen, 2005). The locus coeruleus (LC)-NE system may play a critical role in the regulation of exploitation (i.e., optimizing current

task-performance) and exploration (i.e., disengagement in search of alternative behaviors) tendencies during task-oriented behavior – phasic and tonic NE activity may respectively reflect these control tendencies; Aston-Jones and Cohen, 2005). It may be that exploration is highly analogous to Carver's (2003) idea of "coasting" or that the exploitation–exploration balance is analogous to the maintenance-flexibility balance discussed with regard to dopamine and control (Dreisbach and Goschke, 2004; Muller et al., 2007), but connections between these bodies of work have yet to be made. In particular, it is still not well understood what the relative roles of dopamine and norepinephrine are in emotional and motivational interactions with cognition. Exciting new evidence suggests that pupil dilation can be used as a non-invasive marker of LC-NE activity in humans, with pupil dilation changes indexing shifts in the exploitation–exploration balance during performance of cognitive control tasks (Gilzenrat et al., 2010). Thus, pupillometric methods might be one promising avenue for exploring the role of the LC-NE system in affective versus motivational influences in cognitive control, along with the psychopharmacological and neurotransmitter imaging methods described above.

More generally, the use of psychophysiological measures may provide another approach by which possible dissociations between emotional and motivational influences on cognition can be investigated. Pupil dilation has been well-established to index fairly specific changes in cognitive demand and effort (Beatty, 1982; Granholm et al., 1996; Beatty and Lucero-Wagoner, 2000) and thus may provide a measure of cognitive control, independent from behavior, that can finely index changes in temporal control dynamics. For example, pupil dilation has been successfully utilized to examine the temporal dynamics of goal maintenance and proactive versus reactive control within the AX-CPT paradigm (related to developmental changes; e.g., Chatham et al., 2009). Additionally, pupil dilation is sensitive to emotional variables as well, and may reflect emotional arousal (Bradley et al., 2008). Thus, pupillometric methods might be exceptionally well-suited for examining interactions of affective/motivational systems and cognitive control, although such interactions have almost never been explored (e.g., Satterthwaite et al., 2007). Other psychophysiological indices might be promising as well in this regard – skin conductance (SCR), startle probes, and EMG are all well-established autonomic indicators of affective state (Bradley et al., 2001). Thus, comparison of the effects of affective versus motivational manipulations on these indicators might help to reveal potential dissimilarities in autonomic profile, as well as whether some indicators serve as better predictors of the cognitive control effects of such manipulations.

Another means by which to probe for distinctions between emotion and motivation might be to more clearly assess the role of subjective experience. Subjective experience is thought to be a core ingredient to the construct of emotion (Barrett et al., 2007), but it remains unclear whether it should be considered key to the construct of motivation as well. Recent evidence suggests that subliminally presented reward cues can lead to similar effects on behavior and cognitive control as stimuli that are consciously perceived (Aarts et al., 2008a; Bijleveld et al., 2010; Zedelius et al., 2010), suggesting that subjective awareness of motivational value is not critical. In the emotion realm, subliminal information has been shown to influence affective preference (Zajonc, 1980), and non-emotional facial changes (i.e., to a smile-like position) have been associated with changes in emotional response (Strack et al., 1988). However, subliminal presentation of positively valenced stimuli seems to only impact motivated behaviors such as the amount consumed and willingness-to-pay for rewards, but not subjectively experienced affective responses such as positive mood or reward liking (Winkielman et al., 2005). Thus, it is not clear whether subliminal manipulations of positive affect would impact cognitive control in the same manner as manipulations that are subjectively experienced.

As a final consideration, the relationship between emotion and motivation could be explored by examining the effect these manipulations have on *each other*. Some evidence suggests that reward incentives promote positive mood (Meloy et al., 2006), but such research is relatively sparse and, to our knowledge, effects of emotion on the effectiveness of a motivational manipulation remains unknown.

SUMMARY AND CONCLUSION

While emotion and motivation have been assumed to be related, they have largely been investigated independently with relation to cognitive control. Research in both areas may benefit from greater theoretical and empirical integration. Current theories suggest that the influences of positive emotion and reward incentives may depend on a common neuroanatomy: both may increase midbrain dopaminergic activity (i.e., ventral tegmental area), which projects to control-associated areas, such as the PFC and the ACC. The PFC in particular has received attention as a potential integration site for task and affective information in both emotional and motivational lines of research.

Despite these parallels, many research questions remain to be addressed. Perhaps most fundamentally, the neurobiological mechanism, independent of reward, by which positive emotion influences cognition remain unclear. Specifically, there is as of yet no direct evidence that supports a model in which positive emotion, independent of other components of reward, is associated with dopamine activity. Clarifying this mechanism will be key to establishing whether positive emotion and reward motivation influences on cognitive control are common or distinct. Second, further exploration of differential contributions of tonic and phasic activity in both the DA system and other relevant neurotransmitter systems, such as LC-NE, could help characterize both emotional and motivational influences on cognition, especially in probing further the hypothesis that positive affect and reward might be associated with more reactive and proactive (and/or exploratory versus exploitative) modes of cognitive processing, respectively. Third, inclusion of both psychophysiological measures and assessment/manipulation of subjective experience may be beneficial in theoretically distinguishing constructs of emotion from motivation and improving understanding of how they impact cognition. Fourth, effects of emotion and motivation on one another should be probed to help clarify their relationship.

Emotions may be characterized as emergent from one's current status relative to one's optimal goal status (Carver, 2006; Lang and Bradley, 2008). Thus, while emotion and motivation are closely related, emotions as an evaluator of goal status serve a slightly

different functional purpose than motivations, and consequently may be more fluid in relation to any given goal. Accordingly, they may have a more variable influence on cognitive control relative to motivational influences, which serve to optimize goal-relevant processing directly. Clarifying these constructs will help unite disparate lines of research within a common theoretical framework, and provide a more nuanced picture of these interactions and their complexities. By doing so, this research effort may help to significantly advance the emerging field of affect–cognition interactions.

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Post-error adjustments

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When our brain detects an error, this process changes how we react on ensuing trials. People show post-error adaptations, potentially to improve their performance in the near future. At least three types of behavioral post-error adjustments have been observed. These are post-error slowing (PES), post-error reduction of interference, and post-error improvement in accuracy (PIA). Apart from these behavioral changes, post-error adaptations have also been observed on a neuronal level with functional magnetic resonance imaging and electroencephalography. Neuronal post-error adaptations comprise activity increase in task-relevant brain areas, activity decrease in distracter-encoding brain areas, activity modulations in the motor system, and mid-frontal theta power increases. Here, we review the current literature with respect to these post-error adjustments, discuss under which circumstances these adjustments can be observed, and whether the different types of adjustments are linked to each other. We also evaluate different approaches for explaining the functional role of PES. In addition, we report reanalyzed and follow-up data from a flanker task and a moving dots interference task showing (1) that PES and PIA are not necessarily correlated, (2) that PES depends on the response–stimulus interval, and (3) that PES is reliable on a within-subject level over periods as long as several months.

Keywords: post-error slowing, post-error reduction of interference, post-error improvement in accuracy, cognitive control, orienting response, inhibition, posterior medial frontal cortex

When we realize that we have just committed an error, we often stop our current movement for a brief moment or at least we slow down a little bit. This might help us to focus on our task again – that is, to pay more attention to the relevant aspects of the task and ignore irrelevant information – or to realize why we committed an error or what exactly the error was. Alternatively, we slow down just because we are surprised about the unexpected event of committing an error. Sometimes our performance improves after the commission of an error. However, it is yet unknown under which exact circumstances we improve. At least three types of post-error adjustments have been observed. These are post-error slowing (PES; e.g., Rabbitt, 1966; Debener et al., 2005; Eichele et al., 2010), post-error reduction of interference (PERI; Ridderinkhof et al., 2002; King et al., 2010), and post-error improvements in accuracy (PIA; e.g., Laming, 1968; Marco-Pallares et al., 2008; Danielmeier et al., 2011). There is increasing evidence that PES is independent from the other post-error adjustments, but that two or more post-error adjustment processes might occur in parallel. In the following, we will summarize what is known about each of these post-error adjustments and complement this with own data on PES.

POST-ERROR SLOWING

Post-error slowing describes the prolonged reaction time (RT) in trials subsequent to an error compared to RTs in trials following correct trials (Rabbitt, 1966; Laming, 1968). PES has been observed in a variety of different tasks, for instance in flanker (Debener et al., 2005; Krämer et al., 2007; Cavanagh et al., 2009; Eichele et al., 2010), Stroop (Gehring and Fencsik, 2001), Simon (King et al., 2010; Danielmeier et al., 2011), or categorization (Jentzsch and Dudschig, 2009) tasks. However, other studies reported conditions under which no PES effects were observed (e.g., Ullsperger and Szymanowski, 2004; Fiehler et al., 2005). Two recent experiments found PES only in conditions where error trials were infrequent, while observing post-correct slowing when correct trials were infrequent (Notebaert et al., 2009; Nunez Castellar et al., 2010). Thus, it is yet unclear under which conditions PES can be observed, and what the underlying mechanisms are. It has been suggested that PES is either related to cognitive control processes associated with the error (Ridderinkhof et al., 2004), or that it is related to inhibitory motor processes (Marco-Pallares et al., 2008), or that it reflects attentional re-orientation (orienting account; Notebaert et al., 2009) not fulfilling any specific and direct function in terms of performance improvement. In the following we present these theoretical accounts in more detail, then we discuss various factors that might influence PES. We also report two experiments investigating (1) the influence of trial timing on PES, and (2) the intra-individual reliability of PES.

THREE ACCOUNTS EXPLAINING PES

On the one hand it has been argued that PES is related to cognitive control mechanisms which are implemented after the commission of errors (Botvinick et al., 2001). On the other hand there is evidence that PES reflects an orienting response following infrequent events like errors (Notebaert et al., 2009). Additionally, an inhibition account is supported by functional and structural anatomical studies and EEG experiments showing that motor inhibition is related to PES (Ridderinkhof, 2002b; Marco-Pallares et al., 2008).

In the following we will discuss these three explanations for PES, which, to our opinion, are not mutually exclusive.

Cognitive control account

Gehring and Fencsik (2001) suggested that PES is a compensatory control mechanism serving to improve subsequent performance. Thus, in contrast to the orienting account, it is assumed that PES serves a functionally meaningful purpose (cf. Carter and van Veen, 2007). Specifically, it has been suggested that PES serves to buy time to enable more controlled responding (Ridderinkhof et al., 2004). Alternatively, it has been suggested that cognitive control in post-error trials leads to engagement of attentional top-down modulations (MacDonald et al., 2000).

Within the conflict monitoring theory (Botvinick et al., 2001) PES is explained in terms of decreased activity in the response priming unit, which equals an increased motor threshold. This strongly links the cognitive control account with the inhibition account (see below). The assumption of decreased activity in the response unit is supported by fMRI results showing reduced motor activity in post-error trials (King et al., 2010) which is negatively correlated with PES (Danielmeier et al., 2011), i.e., participants with less motor activity in post-error trials show larger PES effects. Motor activity, in turn, was predicted by activity in the performance monitoring network, comprising the posterior median frontal cortex (pMFC), in preceding error trials, with more error-related pMFC activity predicting less motor activity. Since this is a correlational finding one cannot conclude whether pMFC activity causes the decrease in motor activity, for instance via a purposeful adjustment in speed–accuracy thresholds as suggested by Botvinick et al. (2001), or whether the observed activity adjustments just occur coincidentally together. Increased pMFC activity might be the reaction to an error, which also leads to an orienting response that in turn causes the slowing of motor responses.

The cognitive control theory assumes that post-error adjustments are triggered by top-down signals from the performance monitoring system, which has been associated with the pMFC. Several fMRI findings support this assumption by showing a correlation between pMFC activity and PES (Garavan et al., 2002; Kerns et al., 2004; Klein et al., 2007a; Chevrier and Schachar, 2010). Also, the amplitude of the error-related negativity (ERN; Gehring et al., 1993; Debener et al., 2005; West and Travers, 2008; Wessel and Ullsperger, 2011) as well as a mid-frontal power increase (Cavanagh et al., 2009) have been shown to predict the amount of PES in EEG experiments. Some fMRI studies associated PES with increased dorsolateral prefrontal activity in post-error trials (Kerns et al., 2004), which is thought to be driven by pMFC activations (for a review see Carter and van Veen, 2007).

However, some studies did not find any correlation between pMFC activity and PES (Gehring and Fencsik, 2001), while others found a correlation between PES and the error positivity (Pe), instead of the ERN (Nieuwenhuis et al., 2001; Hajcak et al., 2003). Although these results might seem contradictory, one potential reason for some studies finding a correlation and others not, might be that pMFC activity and PES are linked only indirectly. It has been shown that PES is linked to pMFC activity in error trials via an activity decrease in the motor system (King et al., 2010; Danielmeier et al., 2011).

Thus, there is evidence that PES is related to activity in the performance monitoring system, whether directly or indirectly, and therefore, PES might reflect the implementation of cognitive control.

Orienting account

Notebaert et al. (2009) suggested that PES reflects an orienting response to an unexpected event. Since errors are usually rare, they represent unexpected, motivationally salient events. Thus, according to this account, PES is independent from cognitive control processes. Based on this idea, Notebaert et al. (2009) compared the reaction to errors with the reaction to oddball stimuli, which are also infrequent events. Usually, RT slowing can also be observed after surprising (infrequent) events. This might be due to an orienting response elicited by these stimuli. A similar orienting response might be elicited by errors and can be observed as PES. Notebaert et al. (2009) compared an experimental condition where errors were relatively infrequent with another condition where errors were committed frequently and correct trials were infrequent. With infrequent errors, PES was observed as expected. However, when correct trials were infrequent, they observed a post-*correct* slowing instead. This indicates that PES is not necessarily only reflecting post-error adaptation processes, but depends on the relative frequency of errors. Thus, PES might be an orienting response to an unexpected event, instead of an error-driven cognitive control adjustment.

In an EEG experiment, Nunez Castellar et al. (2010) observed that post-error RTs correlate with the P3 amplitude. The P3, specifically the P3a, has been associated with the novelty processing of an orienting response (Friedman et al., 2001). Thus, the orienting account is corroborated by this ERP finding.

It has also been suggested that PES results from a failure to disengage from the error (Carp and Compton, 2009) or, more generally, from a failure to disengage from performance problems including increased response conflict (Compton et al., 2011). Usually people briefly disengage from the task after correct responses. This has been observed as alpha power increase (indicating a more relaxed status) after correct trials compared to periods following high conflict (e.g., errors). Reduced alpha power after erroneous responses might reflect heightened arousal or orienting responses and therefore could also support the orienting account.

Studies that potentially support the orienting idea of PES are those where PES only occurs in conditions where fewer errors are committed. For instance, Ullsperger and Szymanowski (2004) found PES only in the accuracy condition, where participants committed fewer errors than in the speed condition. Also, Fiehler et al. (2005) divided their subjects in two groups: one group was explicitly instructed to correct their errors immediately, whereas the second group was not aware that they could correct their errors. The second group committed fewer errors than the first group, and only the second group showed PES.

If, in contrast, PES reflected cognitive control processes in the sense that PES serves to improve behavior, one would expect improvements in post-error performance (PIA) to co-occur with PES. Indeed, there are several studies reporting both PES and PIA (Laming, 1968, 1979; Marco-Pallares et al., 2008; Danielmeier et al.,

2011). But there are also studies reporting PES in combination with *decreased* accuracy in post-error trials (Rabbitt and Rodgers, 1977; Fiehler et al., 2005).

However, even if both PES and PIA are shown within the same experiment, this does not necessarily involve a correlation between both factors (Carp and Compton, 2009), i.e., participants with a large PES effect are not necessarily the same participants who show large post-error improvement rates (but see Hajcak et al., 2003, who did find a correlation between PES and PIA). In one of our experiments (Danielmeier et al., 2011) we found both PES and PIA, but the effects did not correlate on the individual subjects level ($r = 0.25$, $p = 0.28$; **Figure 1**). The fact that PES does not necessarily go along with a subsequent performance improvement seems to speak against a direct role for PES in enhancing accuracy. Also, PERI, which is assumed to reflect strategic adjustments in response to errors, and therefore could represent the result of cognitive control processes, does not correlate with PES (King et al., 2010). One possibility could be that PES and cognitive control processes sometimes co-occur within a similar time period, but that these processes are more or less independent. Although, we suggest that PES might have a permissive effect on top-down modulations.

However, if PES just represented a general orienting response to surprising events, one would expect a comparable response after errors and after surprising false feedback (i.e., signaling the subject an error although the response was correct). But at least three studies have shown that subjects slow down only after self-committed errors, but not after inserted or externally induced errors, i.e., when subjects actually responded correctly (De Bruijn et al., 2004b; Logan and Crump, 2010; Steinhauser and Kiesel, 2011). This suggests that, although there is slowing to a certain extent following infrequent events (e.g., Notebaert et al., 2009), a surprising external event is not always sufficient to evoke PES.

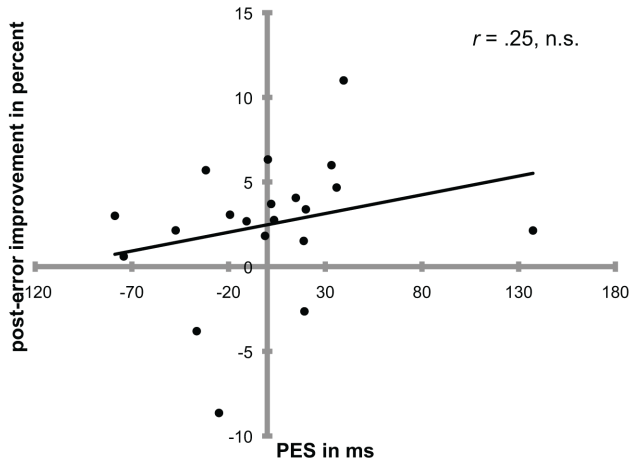

FIGURE 1 | Post-error slowing and post-error improvement in accuracy (PIA) did not correlate significantly in a colored-moving dot interference task (Danielmeier et al., 2011). Participants showed either both PES and PIA (upper right quadrant) or only PIA without PES (upper left quadrant). One participant showed only PES without improvement and two subjects showed neither PES nor PIA following errors.

Inhibitory account

Within the activation–suppression hypothesis Ridderinkhof (2002b) already suggested that the commission of an error leads to an increase in selective suppression in the post-error trial. This was revealed by more negative slopes in the RT delta plots of post-error compared to post-correct trials. RT delta plots depict the interference effect as function of response speed, with negative slopes indicating a decreased or reversed interference effect with increasing RTs. In general, the activation of the incorrect response tendency should lead to more inhibition in the subsequent trial (Ridderinkhof, 2002a). There is growing evidence, that a right-hemispheric network, consisting of presupplementary motor area (pre-SMA), lateral inferior frontal cortex (IFC), and subthalamic nucleus (STN) is involved in PES. This network has been associated with motor stopping or slowing (Aron et al., 2007), thus linking PES to motor inhibition. The following studies provide evidence for this assumption.

The inhibition hypothesis of PES was supported by data of Marco-Pallares et al. (2008). They showed that PES correlates with an increase in beta band power in an EEG experiment. The increase in beta band power has been associated with inhibitory processes, and specifically with motor inhibition (Kühn et al., 2004; Marco-Pallares et al., 2008; Swann et al., 2009). In contrast, suppressed beta band oscillations have been associated with faster responses (van Ede et al., 2011). Furthermore, inhibition-related beta band modulations have been associated with the STN (Kühn et al., 2004) and the right IFC (rIFC; Swann et al., 2009). The pre-SMA has been shown to modulate the inhibitory influence of the rIFC (Neubert et al., 2010). The STN, rIFC, and pre-SMA are interconnected and constitute a network that has been associated with motor stopping or slowing (Aron et al., 2007) and action reprogramming (Neubert et al., 2010). Chevrier and Schachar (2010) reported that activity in the right SMA and the dorsal substantia nigra (among other areas) is positively correlated with PES. Since the dorsal substantia nigra is directly adjacent to the STN, it might be difficult to distinguish these areas in fMRI contrasts. We recently found a correlation between individual PES values and structural measures in this right-hemispheric pre-SMA–IFC–STN network (Danielmeier et al., 2011), indicating that this inhibition network also contributes to PES, and complementing the findings that this network operates in the beta band frequency, which in turn is correlated with PES.

The primary motor cortex (M1) is a downstream target of inhibitory control (Swann et al., 2009). In two of our studies, we showed reduced motor cortex activity in post-error trials (King et al., 2010; Danielmeier et al., 2011). This reduction was related to PES, that is, the less M1 activity in post-error trials the more PES was observed in that person. This result supports both the inhibitory account of PES as well as the suggestion of the conflict monitoring theory (Botvinick et al., 2001) that PES reflects an increased response threshold in post-error trials. Furthermore, PES is also correlated with baseline cortisol levels, which in turn have been associated with inhibitory behavior (Tops and Boksem, 2011), suggesting an additional link between PES and behavioral inhibition.

Together, there is evidence that motor inhibition plays a crucial role during PES. However, this does not exclude the possibility that

other, probably more cognitive, processes or orienting responses are executed simultaneously. Indeed, we showed that other adjustments can be observed in extrastriate visual areas in post-error trials (King et al., 2010; Danielmeier et al., 2011), but these adjustments were not directly related to PES.

PES IS MODULATED BY TRIAL TIMING

Jentzsch and Dudschig (2009) showed that the PES effect is influenced by the response–stimulus interval (RSI). They manipulated the RSI in a categorization task either between subjects or within subjects in a block-wise fashion. The RSI was either short (50 ms in Experiment 1 or 100 ms in Experiment 2) or long (1000 ms). Although they found significant PES effects in both conditions, PES was considerably larger under short RSI conditions. One question that arises from this experiment is, whether parts of this effect are due to long-term adjustments (macro-adjustments; Ridderinkhof, 2002b), that is, subjects adapt to the higher frequency of stimulus presentation, and thus larger time–pressure, in one block compared to the other block, or whether this effect can also be observed when the RSI varies from trial to trial and subjects cannot adapt to a general stimulus frequency. In Experiment 1, we replicated the timing-dependent effect of PES with a different task, more RSI intervals, and with a randomized trial-to-trial variation of the RSI instead of a block-wise manipulation to exclude the effect of macro-adjustments.

Experiment 1

We conducted an experiment that employed a modified flanker task (Eriksen and Eriksen, 1974). While Jentzsch and Dudschig (2009; Experiment 2) kept the RSI constant for the first half of their experiment and switched to the other RSI in the second half, here RSIs varied from trial to trial in pseudo-random order.

Participants. Nineteen healthy volunteers (eight male; mean age: 24 years; range: 20–27 years of age) participated in this experiment after signing informed consent. All participants had normal or corrected-to-normal vision, one participant was left-handed according to the Edinburgh Handedness Inventory (Oldfield, 1971). The experiment was carried out in accordance with the Declaration of Helsinki.

Task. An arrow version of the flanker task was employed as described in Danielmeier et al. (2009). The only difference to that previous study was the introduction of four different RSIs, which was either 200, 750, 1500, or 3000 ms. In this task a left- or right-pointing arrow was presented as a target in the center of a screen for 50 ms. Two flanker arrows were presented above the target and two below the target. Flanker arrows could either point in the same direction as the target (congruent condition) or in the opposite direction (incongruent condition). Flankers were either presented close to the target (CLOSE condition, i.e., 1.75° and 3.5° above and below the target) or further away from the target (FAR condition, i.e., 4.0° and 6.5° above and below the target), thereby creating modulations in response conflict (cf. Danielmeier et al., 2009). Flanker presentation preceded the target presentation by 80 ms. Speed and accuracy were emphasized equally in the instructions. After the participants' response, a black screen was presented for the durations of one of four possible RSIs. Each RSI occurred equally often in every experimental condition, but RSI duration varied in pseudo-randomized order. In case no response was recorded, the next trial started after 2000 ms. In total, 996 trials were presented. Stimuli were presented using Presentation 13.1 (Neurobehavioral Systems, San Francisco, CA, USA).

Results and discussion. The effects of congruency and flanker distance showed the same results as in Danielmeier et al. (2009) for each RSI. These results are omitted for brevity, since they are not relevant for our conclusion here. Error rates were 9.12, 9.97, 9.0, and 10.23% for RSI 250, 750, 1500, and 3000 ms, respectively. The RSIs here refer to the time interval *following* an error, because this interval is crucial for post-error adaptations. There was a trend for error rates being different for the four RSIs [$F(3,16) = 2.68$; $p = 0.08$]. However, note that there was no way for participants to influence the RSI following their errors, because the order of RSIs was random and predetermined.

Post-error changes in accuracy were evaluated by comparing post-error error rates with post-correct error rates. There was no significant change in any RSI. PES values were calculated as mean post-error RT minus mean post-correct RT, with post-correct trials being correctly performed trials preceded by at least two and followed by at least one other correct trial. This analysis was done for each RSI separately to investigate the effect of RSI on PES. The indicated RSI here was that between error and post-error trial.

Post-error slowing differed significantly between RSIs [$F(3,16) = 4.97$; $p = 0.013$], with shorter RSIs leading to more PES [mean PES values (SEM): 59 ms (21), 11 ms (12), −9 ms (10), and −1 ms (9) for RSI 200, 750, 1500, and 3000, respectively; **Figure 2**]. Only for RSI 200, the RT in post-error trials was significantly slower than RTs in post-correct trials [$t(18) = 2.68$; $p = 0.015$].

Thus, we replicated the finding by Jentzsch and Dudschig (2009), showing that PES varies with RSI. Slowing is most

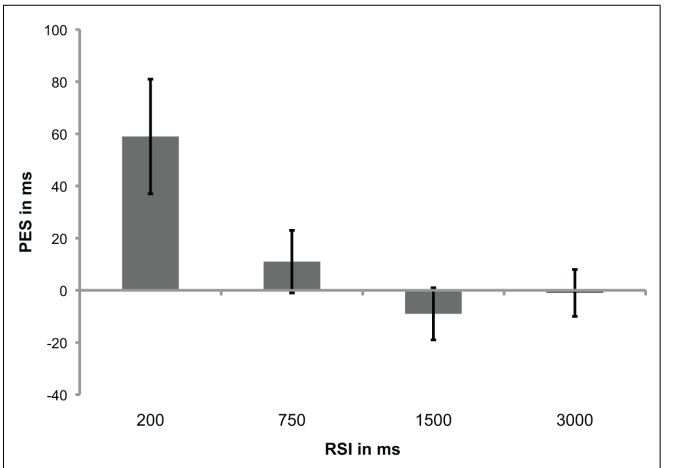

FIGURE 2 | Mean post-error slowing (PES) values (and SEM) for each response–stimulus interval (RSI). PES for RSI 200 is significantly larger than for RSI 1500 and 3000 (both $p < 0.005$), and there is a trend for a difference between RSI 200 and 750 ($p = 0.088$). There is also a marginally significant difference between RSI 750 and 1500 ($p = 0.088$). RSI 1500 and 3000 do not differ.

pronounced if error and post-error trials are separated only by short time intervals. With longer intervals -in the present task with an RSI of 750 ms or longer- PES was not observed anymore. We extended the results by Jentzsch and Dudschig (2009) by showing that the RSI-dependent PES effect is not exclusively due to macro-adjustments, but is also present when the experimental timing is more variable. That is, the RSI-dependent PES effect in their experiment cannot solely be attributed to the assumption that participants are in a general "speed mode" during one half of the experiment.

Furthermore, the RSI-dependent PES effect can be observed in different types of tasks. It seems like there is a "decay" of PES over time that is independent of the exact task. However, in other experiments PES has been observed with much longer RSIs than 750 ms. For instance, in some fMRI studies there was a substantial PES effect even after 4–5 s (King et al., 2010; Danielmeier et al., 2011). We would speculate that also in those experiments a PES decay takes place, but that the exact timing of this decay might depend on specific requirements of the task, e.g., on task difficulty or whether the general task timing is faster or slower. For instance, Jentzsch and Dudschig (2009) demonstrated a significant PES effect also for RSIs of 1000 ms. As described above, in their task design, RSIs were varied in a block-wise manner, that is the general timing of the task was either very fast (with short RSIs of 50 or 100 ms) or slower for longer periods of time.

PES IS LARGER AFTER CONSCIOUS ERROR PERCEPTION THAN AFTER UNNOTICED ERRORS

Several studies have investigated conscious error perception or "error awareness." These tasks aim at evoking both consciously perceived errors and errors that go unnoticed. PES has rarely been observed in "error awareness" tasks, that is, in this type of tasks there seems to be no general PES effect. However, if trials are split into consciously perceived and unperceived errors, an RT slowing has been described following perceived errors, but not after errors that were unnoticed (Nieuwenhuis et al., 2001; Wessel et al., 2011). This suggests that conscious error perception is correlated with PES. However, in a very similar antisaccade task, no PES was observed, neither over all post-error trials nor for perceived errors only, although a subgroup of participants did show PES (Klein et al., 2007a). In this study by Klein et al., however, the average trial duration was 6 s, which presumably reduced the chances of observing PES (cf. Jentzsch and Dudschig, 2009, and Experiment 1). In a Go–Nogo task, Cohen et al. (2009) reported a significant PES effect after unnoticed Nogo errors, although this effect was rather small in magnitude (3.1 ms). However, PES following conscious Nogo errors was considerably larger than PES following unnoticed errors. Hester et al. (2005) investigated post-error RTs in a different error awareness task. They reported PES only for unaware errors, but not for aware errors. Conclusions from this task with respect to PES are limited though, because in the trial directly following an aware error, subjects were required to signal their previous error instead of responding to the stimulus. That is, subjects did not need to process the stimulus in the post-(aware-)error trial. Therefore, it is not surprising that this study found a post-error *decrease* in RT instead of an increase. In a follow-up study, Hester et al. (2007b) demonstrated that cocaine users do show PES after consciously perceived errors while control subjects did not show PES in the second trial after error commission.

Thus, there is preliminary evidence that PES is larger after consciously perceived errors than after unnoticed errors. However, in order to evaluate the difference between aware and unaware errors on post-error adjustments, further studies employing a larger variety of error awareness tasks are necessary. Ideally, these studies would also be controlled for possible RSI effects.

PES IS GREATEST WHEN ACCURACY IS IMPORTANT

In addition to RSI and possibly error awareness, PES also depends on the importance of responding correctly. When participants were asked to perform the same experiment twice, once with an instruction stressing speeded responding and once with an instruction emphasizing accuracy, participants showed PES only in the accuracy condition, but not under speed instructions (Ullsperger and Szymanowski, 2004). Note that participants also committed fewer errors in the accuracy condition.

Fiehler et al. (2005) compared two groups of participants: one group was instructed to immediately correct their errors, whereas the other group was not instructed to correct themselves, and therefore was unaware that correction responses were recorded. PES was only observed in the non-instructed group. The correction instruction might have reduced subjective error relevance, because of the possibility to correct mistakes. Note that the non-instructed group also committed fewer errors.

These experiments both suggest, that PES is only pronounced if participants try hard to avoid errors and belief that accuracy is crucial. Participants might adapt their motor threshold according to speed or accuracy requirements. This could lead to more PES when accuracy is emphasized. Alternatively, these results suggest that PES occurs predominantly under conditions, when errors are rather infrequent. The latter interpretation is corroborated by findings from Notebaert et al. (Notebaert et al., 2009; Nunez Castellar et al., 2010). They also observed PES only when errors were infrequent, but not when errors were committed frequently.

THE RELIABILITY OF PES

It has been shown that the ERN amplitude is rather stable within individuals (Segalowitz et al., 2010). Thus, the question arises whether post-error adjustment processes, such as PES, are also reliable within subjects. To investigate whether the PES effect is rather stable over time, we ran a longitudinal study, where participants performed the same flanker task twice at intervals of several months.

Experiment 2

Participants and task. Fifteen volunteers (nine women; mean age: 23.3 years; range: 18–32 years of age) with normal or corrected-to-normal vision participated in this experiment after signing informed consent. All but one participant were right-handed. The experiment was carried out in accordance with the Declaration of Helsinki. Participants were tested twice. On average, the second experimental session took place 4.6 month after the first session (range: 4–6 months).

The task was the same as in Danielmeier et al. (2009). This was the same arrow version of the flanker task as in Experiment 1, except that the RSI did not vary systematically, but was

set to 1000 ms. As in the original experiment, participants were instructed to signal their errors. Because participants would not reliably follow this instruction with an RSI of 1000 ms, the RSI was prolonged by 800 ms in case an error was signaled. One thousand trials were presented during the course of the experimental session.

Results and discussion. Error rates were 16.8% (SEM: 1.16%) in the first experimental session, and 14.9% (SEM: 1.78%) in session 2. Error rates did not differ between sessions [$t(14) = 1.42$; $p = 0.18$]. In session 1, the average post-error RT (only correct trials following errors) was 366 ms, and post-correct RT was 358 ms. In session 2, the average post-error RT was 364 ms, and post-correct RT was 359 ms. To account for potential baseline differences in RTs between sessions, PES was calculated here as percent RT change in post-error trials compared to RT in correct trials. Although the average PES effect was rather small in this experiment [2.8% RT increase in session 1 (SEM: 0.94% ms) and 1.5% in session 2 (SEM: 1.0%)], individual PES values of both sessions showed a significant correlation (Spearman's rho = 0.54; $p = 0.04$; **Figure 3**). A t-test revealed that PES did not differ between sessions [$t(14) = 1.33$; $p = 0.20$].

In sum, the size of the individual PES effect seems to be rather stable over a time period of several months, extending the findings by Segalowitz et al. (2010), who reported intra-individual reliability of PES over periods between 20 min and a couple of weeks. This reliability of PES is corroborated by the fact that the size of the PES effect corresponds to structural differences in white matter (WM) tracts (Danielmeier et al., 2011), which presumably are also stable over time.

NEUROANATOMICAL BASIS OF PES

Only few fMRI, diffusion-weighted imaging, and patient studies have provided evidence for PES-related brain structures. Probably due to this scarce evidence, the results are not unequivocal yet, although several studies seem to converge in that a network consisting of pre-SMA, lateral IFC, and the STN of the right hemisphere plays a crucial role during the emergence of PES. This network has originally been described by Aron et al. (2007) by using both diffusion-weighted tractography and fMRI. They have associated these brain areas with conflict-related slowing in a conditional-stopping task.

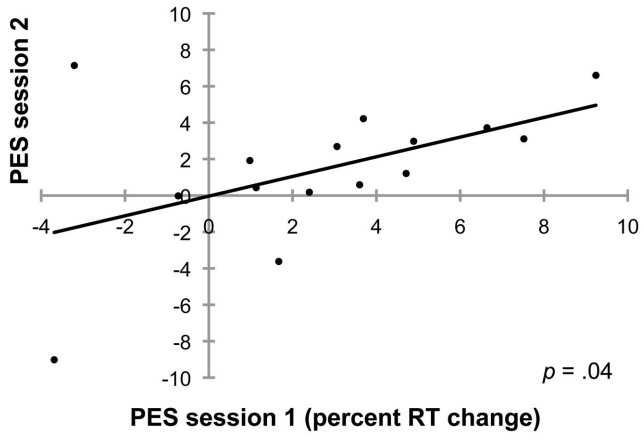

FIGURE 3 | Individual PES values (calculated as RT change in post-error trials relative to RT in correct trials) show a significant correlation between experimental session 1 and experimental session 2. There were on average 4.6 months between the first and the second session.

A recent study of our own group directly investigated PES correlates of both diffusion-weighted imaging and fMRI (Danielmeier et al., 2011). We showed that PES is correlated with fractional anisotropy (FA) values in white matter tracts beneath the right pre-SMA. Probabilistic tractography revealed that this WM area belongs to those tracts described by Aron et al. (2007) connecting pre-SMA with lateral IFC and the STN. This suggests that PES and conflict-related slowing engage the same network of brain areas. Additionally, we found FA values in the vicinity of the left anterior midcingulate region to be correlated with PES. This region is part of a network connecting the anterior midcingulate region with dorsolateral prefrontal cortex and more posterior parts of the brain. This could point to the fact that at least two different processes contribute to PES: first, an inhibitory process acting on the motor system, and second, the implementation of cognitive control processes in dorsolateral frontal areas.

By using a voxel-based lesion symptom mapping (VLSM) approach, Molenberghs et al. (2009) showed that the middle third of the right inferior frontal sulcus is crucial for PES. Patients with lesions in this brain area did not show any PES in the Sustained Attention to Response task, while patients with other brain lesions did show PES. This finding corroborates the PES tractography result, demonstrating that the network consisting of right pre-SMA, IFC (or probably more specific: inferior frontal sulcus), and STN is crucial for PES. Furthermore, di Pellegrino et al. (2007) showed that patients with lesions in the medial frontal cortex did not show PES, in contrast to patient with lesions outside the frontal cortex who showed a PES effect comparable to the healthy control group. There is also evidence from an fMRI study with healthy participants that pre-SMA activity is correlated with PES (Klein et al., 2007a).

In the functional data of our recent study, we found that PES is negatively correlated with the activation level in the motor system in post-error trials (Danielmeier et al., 2011). The activity decrease in the motor system in turn is predicted by the level of pMFC activity in the preceding error trial. Thus, pMFC activity (including pre-SMA) seems to downregulate the motor system which leads to PES. A negative correlation between motor activity and the individual PES effect has been shown before in a different task (King et al., 2010). This study by King et al. (2010) also found a positive correlation between PES and the right IFJ, a frontolateral brain area at the junction of the inferior frontal sulcus and the precentral sulcus (Derrfuss et al., 2009).

However, there seem to be two functionally separable brain areas in close proximity in this region of the brain: whereas the posterior inferior frontal gyrus (pIFG) has been associated with response inhibition, the IFJ has been associated with the processing of infrequent events (Chikazoe et al., 2009). Both functions might be crucial for PES. While the response inhibition function of the pIFG would support the inhibitory account, the processing of infrequent events in the IFJ would corroborate the orienting

account of PES. A recent TMS study also differentiated IFJ and pIFG of the right hemisphere functionally (Verbruggen et al., 2010). This study concluded that IFJ detects visual changes in stimulus features, whereas the pIFG is responsible for updating action plans.

Taken together, there is converging evidence that the right hemisphere is crucial for PES, and especially a network consisting of pre-SMA, lateral inferior frontal areas, and the STN.

CONCLUSIONS ON PES

Together, PES seems to be more pronounced (a) with short than with long RSIs, (b) following consciously perceived errors than following unperceived errors, (c) when errors are infrequent events, and (d) when accuracy is emphasized over speed.

In conclusion, there is evidence for all three accounts explaining PES: the cognitive control account, the orienting account and the inhibitory account. We do not think that these three accounts are mutually exclusive. There are elements in each account linking it to another account. For instance, an orienting response most likely recruits inhibitory processes (cf. Ursin, 2005). Also, the model by Botvinick et al. (2001) links cognitive control, and specifically PES, to increases in motor threshold, which could be implemented via inhibitory processes. However, PES seems to be independent from top-down control, but it might facilitate top-down modulations by providing time for attentional adjustments.

Whether cognitive processes co-occur with PES or not might depend, for instance, on task difficulty. If a task is not too difficult, it might be worth it to try to improve performance via top-down modulations. However, if a task is too hard (e.g., because stimuli are visually degraded to an extent that it seems impossible to encode them) additional recruitment of cognitive control processes might not improve performance anymore. Likewise, if the time interval between two stimuli is too short, so that cognitive control processes cannot be implemented to their full extent, we presumably cannot expect performance improvements after errors.

POST-ERROR REDUCTION OF INTERFERENCE

Ridderinkhof et al. (2002) described for the first time that the interference effect in a flanker task, i.e., the difference in RTs between compatible and incompatible trials, is reduced after errors. This is the so-called PERI effect, which is thought to reflect cognitive control processes, leading to improvements in interference resolution in post-error trials (e.g., King et al., 2010). PERI is not restricted to flanker tasks, but has also been shown in Simon tasks (Ridderinkhof, 2002b; King et al., 2010).

Interestingly, the PERI effect is influenced by macro-adjustments (Ridderinkhof, 2002b), i.e., adaptations to the broader context of a task that do not vary from trial to trial, but that reflect long-term adjustments, e.g., to be more cautious in difficult tasks. In the study by Ridderinkhof (2002b) the amount of incongruent trials was varied between blocks. When more congruent than incongruent trials were presented, PERI was observed. In contrast, when more incongruent than congruent trials were presented, there was no PERI effect. Ridderinkhof (2002b) argued that in the latter case micro-adjustments, specific to post-error trials, were precluded by long-term macro-adjustments, that is, an adaptation to the circumstance that most trials were incongruent. When the irrelevant stimulus-dimension corresponds to the incorrect response most of the time, this information will be more suppressed in general compared to situations when the irrelevant stimulus-dimension is often associated with the correct response.

Two studies investigated the effect of pharmacological treatments on the PERI effect. De Bruijn et al. (2004a) demonstrated that the PERI effect is absent under lorazepam, a benzodiazepine that modulates fast inhibitory transmission of $GABA_A$ receptors. In contrast, under the noradrenergic and specific serotonergic antidepressant mirtazapine, PERI was not different from the control condition without any pharmacological stimulation. Ridderinkhof et al. (2002) showed that PERI is absent after alcohol intake, whereas control subjects did show this reduced interference effect following erroneous trials. Alcohol intake also modulates $GABA_A$ receptors (e.g., Steffensen et al., 2011), amongst others.

In a recent fMRI study King et al. (2010) investigated the neuronal correlates of PERI in a face-version of the Simon task, where participants responded to faces and ignored their position. They showed that post-error activations within the left superior frontal sulcus, the left superior colliculus, and, most importantly, the fusiform face area were modulated by the individual PERI effect. This suggests that modulations in task-relevant visual brain areas are crucial for post-error interference reductions. The more activity in these areas was recorded, the greater was the PERI effect, that is, the resolution of interference in post-error trials.

Note that PERI and PES seem to be implemented in different neuronal networks (for a direct comparison see King et al., 2010), suggesting that interference resolution relies more on task-specific brain areas, whereas PES is more associated with brain areas that modulate or are modulated by the motor threshold, independent from the task at hand. Also, the underlying neurotransmitter systems seem to differ between PERI and PES: while PERI was abolished when lorazepam was administered, PES was unaffected by this $GABA_A$-modulating drug.

POST-ERROR IMPROVEMENT IN ACCURACY

One crucial question is whether humans can learn from their errors or otherwise improve their performance after committing an error. PIA can be observed on different time-scales. On the one hand, there are long-term learning effects following errors or negative feedback (e.g., Klein et al., 2007b; Hester et al., 2008) or adjustments that are observed only several trials after an error (Hester et al., 2007a), on the other hand, e.g., in interference tasks, there are short-term or trial-to-trial adjustments that are represented in decreased error rates directly after error commission. We here focus on the latter type of PIAs.

The behavioral findings are not unequivocal with respect to accuracy improvements after errors. Several studies demonstrated improved accuracy directly after error commission (Laming, 1968, 1979; Marco-Pallares et al., 2008; Danielmeier et al., 2011; Maier et al., 2011). Klein et al. (2007a) reported improved performance only after errors that were consciously perceived by the subject, but not after unnoticed errors. Other studies did not find any difference between post-error and post-correct error rates (cf. Experiment 1; Hajcak et al., 2003; Hajcak and Simons, 2008) or even a decrease in accuracy following errors, at least in certain

experimental conditions (Rabbitt and Rodgers, 1977; Fiehler et al., 2005). Carp and Compton (2009) showed that both ERN and Pe amplitude correlate with post-error accuracy, in that larger ERN/Pe amplitudes go along with better post-error accuracy. A recent study by Seifert et al. (2011) showed that PIA and PES are abolished in patients with thalamic lesions, whereas healthy controls did show PIA and PES.

Since PES and PIA do not always occur together, they seem to represent different processes. PES and PIA might follow different time courses. While PES is strongest following short RSIs (see Experiment 1), accuracy decreases after errors under short RSI conditions (Jentzsch and Dudschig, 2009). In contrast, with longer RSIs, PES decreases and there is no difference anymore between post-error and post-correct error rates. Post-error *improvements* in accuracy might predominantly be observed with even longer inter-trial intervals (e.g., mean ITI between 900 and 2250 ms in Marco-Pallares et al., 2008). Thus, time courses of PES and PIA seem to be dissociable.

Another reason, why PIA effects are not unequivocal, might be that there could be large inter-individual differences in the time course of attentional top-down control, which results in PIA. These might additionally interact with the type of task. The task context could be a further factor for variations in PIA results. PIA is presumably only observable when participants really have a chance to improve their behavior after an error. If a task is extremely difficult, i.e., task parameters are set to yield a high percentage of errors (e.g., stimuli are degraded and thus difficult to encode), participants might not have a chance to significantly improve their accuracy. Further studies are needed to specify the conditions, under which PIA can be observed, more precisely.

A practical problem regarding PIA measures arises when participants commit too many or not enough errors in a certain task, leading to ceiling or floor effects in post-accuracy measures. Thus, one needs to keep in mind that PIA results potentially depend on the number of trials/errors in an experiment. Related to this, researchers need to evaluate carefully, whether possible PIA values are fine-grained enough. For instance, if a subject commits 10 errors in a given task, every single double error leads to an increase of the post-error error rate by 10%. Similarly, post-error accuracy cannot be evaluated reliably, when experiments evoke "streaks" of errors, e.g., when experiments are designed in an (over)adaptive fashion so that the task becomes too hard for a too long time, if error rates are too low, and too easy, if error rates are too high. These externally triggered task adaptations potentially lead to error accumulations during periods when the task is harder which cannot be counteracted by internal post-error adaptation. Thus, whenever there is the possibility that error streaks are task-inherent, one should evaluate PIA with caution.

A recent paper by Maier et al. (2011) suggested that attentional post-error adjustments, which presumably are the prerequisite for performance improvements, only occur after certain error types. They employed a flanker task and found post-error attentional adjustments only after so-called flanker errors, but not after other error types. Flanker errors are those errors where subjects were misled by the irrelevant stimulus feature, i.e., the flankers. This suggests that, in order to investigate PIA in interference tasks, it is worthwhile to distinguish between post-error adaptations following those error, that are due to interference with irrelevant stimulus features, and other types of errors.

GENERAL CONCLUSION

We suggest that in most tasks there are at least two post-error processes taking place in parallel: On the one hand there is PES which is associated with inhibitions in the motor system. On the other hand there are adjustments in task-related brain areas, i.e., areas that are directly involved in encoding the stimuli. These latter adjustments might be interpreted in terms of attentional processes or post-error focusing (Verguts et al., 2011), forming the basis for PERI or PIA, which seem to be independent from PES. King et al. (2010) showed that enhanced activity in the task-relevant visual area is accompanied with post-error speeding, not PES.

Thus, although PES seems to be independent from PERI and PIA, it still might have facilitating effects for these other post-error adjustments (but see Verguts et al., 2011, suggesting that slowing and post-error focusing could also counteract each other). The tricky part in investigating this question is that different adjustments might follow different time courses. That is, there might be inhibitory or orienting processes following error commission, but if the time interval between the error and the following trial is too long, this slowing can most likely not be observed in the post-error RT anymore. The processes that lead to PES seem to decay with time. Furthermore, for PERI and PEA there might be other optimal time intervals to observe these effects, and these time intervals presumably also interact with the task context.

From a neuroanatomical perspective, PES is strongly associated with frontal and subcortical structures of the *right* hemisphere and also with activity decreases in motor areas. PERI and other attentional adjustments act more on task-related visual areas. However, all post-error adjustments seem to be triggered by the pMFC (including pre-SMA and ACC), supporting the idea that error-related pMFC activity signals the need for adjustments.

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Valence, arousal, and cognitive control: a voluntary task-switching study

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The present study focused on the interplay between arousal, valence, and cognitive control. To this end, we investigated how arousal and valence associated with affective stimuli influenced cognitive flexibility when switching between tasks voluntarily. Three hypotheses were tested. First, a valence hypothesis that states that the positive valence of affective stimuli will facilitate both global and task-switching performance because of increased cognitive flexibility. Second, an arousal hypothesis that states that arousal, and not valence, will specifically impair task-switching performance by strengthening the previously executed task-set. Third, an attention hypothesis that states that both cognitive and emotional control ask for limited attentional resources, and predicts that arousal will impair both global and task-switching performance. The results showed that arousal affected task-switching but not global performance, possibly by phasic modulations of the noradrenergic system that reinforces the previously executed task. In addition, positive valence only affected global performance but not task-switching performance, possibly by phasic modulations of dopamine that stimulates the general ability to perform in a multitasking environment.

Keywords: task-switching, voluntary task-switching, emotional control, cognitive control, affective stimuli, IAPS

INTRODUCTION

For many years, research on cognitive control has been conducted without taking into account that goal-directed behavior takes place in an environment consisting of a multitude of stimuli, some of which are emotional. Yet, research not only has shown that cognitive control modulates emotions (e.g., Gross, 2002) but also that emotions influence cognitive control (e.g., Gray, 1999; for a review see Pessoa, 2009). Gray et al. (2002) found that activation in the dorso-lateral pre-frontal cortex (DLPFC), which is part of the cognitive control system, also depends on the presentation of emotional information. Furthermore, the anterior cingulate cortex (ACC) is both connected to the DLPFC and to the limbic system (Bush et al., 2000), which is important for emotional control. Although the connections between emotional and cognitive control are obvious at the neuro-functional level, at the process level it is not always clear how both systems interact. In the present study, we investigated different mechanisms underlying this interplay by testing the influence of irrelevant affective pictures on cognitive flexibility.

Different mechanisms have been proposed that can account for the relation between cognitive and emotional control. A first mechanism is related to the function of the neurotransmitter dopamine (DA). Ashby et al. (1999, 2002) argued that positive information can lead to an increase of DA, resulting in an enhancement of cognitive control (see also Braver et al., 1999; Braver and Cohen, 2000; Cohen et al., 2002; Savine and Braver, 2010). In line with this account, it has been shown that positive information stimulates cognitive flexibility (e.g., Isen and Daubman, 1984; Greene and Noice, 1988; Isen et al., 1992; Kuhl and Kazén, 1999; Bolte et al., 2003; Dreisbach and Goschke, 2004; Dreisbach, 2006). More recently, this account has been adjusted in two ways (Cools et al., 2001, 2007, 2009; Cools and Robbins, 2004). First, phasic and tonic modulations of DA have been dissociated (see also Cools and Robbins, 2004; Cools et al., 2009). While high tonic DA favors reward-based learning, low tonic DA favors punishment-based learning. Second, Cools et al. (2001, 2007) suggested that the influence of phasic increases of DA on cognitive control depends on the demands of the task and on the neural structure in which the DA levels are changed (see also Frank et al., 2004; Maia and Frank, 2011). While phasic increases of DA in the striatum lead to more flexible behavior, and can thus enhance cognitive flexibility, phasic increases of DA in the PFC lead to less distractible behavior, which improves protection from irrelevant information, but deteriorates cognitive flexibility. In order to test this so-called valence hypothesis, we manipulated the valence of affective stimuli on trial basis, presumably resulting in phasic modulations of DA.

A second mechanism that can explain the interplay between emotional and cognitive control is related to the function of the neurotransmitter noradrenalin (NA) in the locus coeruleus (LC). As for DA modulations, Aston-Jones and Cohen (2005) argued that there are two modes of LC–NA function: a phasic mode that stimulates behavioral stability and a tonic mode that stimulates more flexible, but also more distractible behavior. Based on this original model, Verguts and Notebaert (2009) introduced a binding account for cognitive control. This account was based on the observation that when a response conflict arises during the execution of a particular task on trial *n*-1, a response conflict on trial impairs performance less severely than when no response

conflict was present on trial *n*-1 (Gratton et al., 1992). Verguts and Notebaert (2009) argued that the experience of a conflict causes arousal that triggers an immediate boost of levels of NA in the LC. This phasic increase of LC–NA function stimulates Hebbian learning (Hebb, 1949), which binds stimulus and response features into a task-set or event file (Hommel, 2004) and, as a result, task-sets prone to arousal become strengthened (see also Braem et al., 2011). As a consequence, performance based on such task-sets is impaired less by new response conflicts and can thus be interpreted as more stable and less flexible behavior (Aston-Jones and Cohen, 2005). In sum, the arousal hypothesis predicts that arousing affective stimuli, irrespective of their valence, affect cognitive control by strengthening the components of a particular task-set, leading to a decreased cognitive flexibility.

A third more cognitive mechanism for the interplay between emotional and cognitive control is related to the competition between both systems for the limited attentional resources (Schimmack, 2005). In line with this so-called attention hypothesis, it has been shown that arousal related to an affective picture makes it harder to withhold a pre-potent response in a stop-signal task (Verbruggen and De Houwer, 2007) and on no-go trials in a go/no-go task (De Houwer and Tibboel, 2010). In sum, this hypothesis entails that affective information interferes strongly with behavior that asks for cognitive control, such as processes responsible for withholding a pre-potent response, because arousal induced by affective information taxes cognitive resources.

The three aforementioned hypotheses for the interplay between cognitive and emotional control were investigated within the task-switching paradigm, which offers a lab-analog for cognitive flexibility. Task-switching is a well-established tool for studying cognitive control in a setting in which participants are frequently imposed to switch from one task to another (for reviews see Monsell, 2003; Kiesel et al., 2010; Vandierendonck et al., 2010). A typical finding is that switching tasks elicits a switch cost, which is indicated by longer RTs and more errors on task switches than on task repetitions. The switch cost is considered as an index of processes that cope with the reconfiguration of the cognitive system from one task to another but also with the interference this brings along (e.g., Allport et al., 1994; Rogers and Monsell, 1995; Meiran, 1996, 2008; Mayr and Kliegl, 2000; Waszak et al., 2003). In the present study we favored to use the voluntary task-switching (VTS) procedure over more traditional task-switching procedures for three reasons. First, traditional task-switching procedures only have a limited ecological validity because they impose tasks to the participants, resulting in a rather artificial situation (see Vandierendonck et al., 2010). We argue that VTS offers are more complete view of cognitive control, since participants can make free task choices. Second, recent studies have shown that switch costs in VTS are more likely to reflect cognitive control than switch costs observed in traditional task-switching procedures (e.g., Liefooghe et al., 2009, 2010; but see Yeung, 2010). Third, some studies have found evidence that the selection component and the execution component in VTS are underlain by distinct sets of processes and are taxing different sets of control processes (see also Arrington and Yates, 2009; Butler et al., 2011). Thus, besides a switch cost, this procedure also offers an additional index of choice behavior and thus of cognitive flexibility. Typically, participants prefer repeating tasks. This phenomenon is called the task-repetition bias and is thought to result from a difficulty to disengage from a previously executed task (Demanet et al., 2010; Vandamme et al., 2010).

In order to investigate the immediate influence of valence and arousal on VTS, in the present study, affective pictures were presented. Three types of pictures were used: (a) pictures with a positive valence and high arousal; (b) pictures with a neutral valance and low arousal; and (c) pictures with a negative valence and high arousal. These pictures were task-irrelevant and were presented within the interval separating two consecutive VTS trials. This procedure allowed us to investigate the influence of these pictures on consecutive behavior.

Based on the valence hypothesis one could predict that information with a positive valence influences the ability to switch tasks. On the basis of this hypothesis, we predict that positive information will enhance cognitive flexibility (e.g., Isen and Daubman, 1984; Greene and Noice, 1988; Isen et al., 1992; Kuhl and Kazén, 1999; Bolte et al., 2003; Dreisbach and Goschke, 2004; Dreisbach, 2006), possibly by increased levels of DA in the striatum (Aarts et al., 2011), resulting not only in improved global performance but also in an improvement of the ability to switch tasks. In addition, because the size of the task-repetition bias is inversely related to the efficiency of cognitive control to disengage from a previously executed task (e.g., Mayr and Bell, 2006; Demanet et al., 2010), this account predicts that the repetition bias will be smaller following positive affective pictures.

According to the arousal hypothesis one would predict that arousing stimuli strengthen the activated task-sets, irrespective to the valence of these stimuli (e.g., Aston-Jones and Cohen, 2005; Verguts and Notebaert, 2009). Consequently, a strengthened task-set should lead to more facilitation on task repetitions and more interference on task switches, resulting in an increased switch cost (Wylie and Allport, 2000; Yeung and Monsell, 2003). With respect to the task-repetition bias, this hypothesis may imply that because task-sets have been strengthened by arousal, their higher activation may encourage participants to re-select the previously executed task. In other words, a higher task-repetition bias is expected.

According to the attention hypothesis (Schimmack, 2005; Verbruggen and De Houwer, 2007; De Houwer and Tibboel, 2010), it is predicted that arousing stimuli will occupy the cognitive resources needed to switch tasks. As a result, this will lead to higher switch costs. In contrast to the arousal hypothesis, the attention hypothesis predicts that the higher switch cost following arousing pictures will only be caused by slower task switches and not by faster task repetitions. According to the attention hypothesis, all behavior, thus even behavior that requires less attentional resources, such as during task repetitions, will be impaired following arousing pictures, although to a smaller extent than during task switches (e.g., De Houwer and Tibboel, 2010). Therefore, this hypothesis entails that, next to a higher switch cost and task-repetition bias, arousal will lead to an impairment of global performance, which should for instance be reflected in a global increase of reaction times and error rates following arousing pictures.

The three hypotheses seem to suggest that manipulations that affect the size of the switch cost also automatically affect the size of the task-repetition bias. Although evidence was reported that both are related (e.g., Mayr and Bell, 2006), recent studies have

shown that the underlying processes are not identical (Arrington and Yates, 2009; Butler et al., 2011) and thus may measure different aspects of cognitive control. Therefore it is difficult to predict whether both will be influenced in a similar way by the affective information.

MATERIALS AND METHOD

PARTICIPANTS

Sixty students of Ghent University participated for course requirements. Sixteen were excluded from analysis because they exceeded the threshold of 80% task repetitions (for a similar cut-off procedure see Arrington and Logan, 2004, 2005). All 44 participants (23 females) had normal or corrected-to-normal vision and were naïve to the purpose of the experiment.

MATERIALS

The target stimuli were the symbols "#" and "%" presented in the colors purple and green. Participants either categorized the identity of the symbol (symbol task) or the color (color task) of the symbol. Participants performed both tasks manually and responded on a QWERTY keyboard. Both tasks were assigned to a different hand. The symbol task was performed with the left hand with the response keys "D" for symbol "#" and "F" for symbol "%." The color task was performed with the right hand with the response keys "J" for purple symbols and "K" for green symbols. Fifteen pictures (448/336 pixels) were selected from the IAPS. Because previous studies found sex differences in the ratings for the IAPS pictures (e.g., Bradley et al., 2001), this was done for male and female participants separately. These pictures were categorized in three conditions of five pictures (**Table A1** in Appendix): positive, neutral and negative. As shown in **Table A1** in Appendix the positive and negative pictures were matched in function of arousal. The mean arousal score was higher for positive and negative pictures than for neutral pictures. Important to mention is that in order to match the arousal level of the positive pictures with the level of the negative pictures we had to include erotic pictures for the male subjects. This was not the case for female subjects.

PROCEDURE

Participants were tested individually by means of a Pentium III personal computer with a 17-inch color monitor running Tscope (Stevens et al., 2006). Instructions were presented on screen and paraphrased if necessary. The instructions stated that participants were free to select which task to perform on each trial, as long as they performed each task an approximate equal number of times and the pattern of task choices was not predictable. In order to explain unpredictability, we translated the coin-flipping metaphor of Arrington and Logan's (2004, 2005) studies into Dutch. This metaphor entails that subjects sometimes will have to repeat the same task and sometimes have to switch between tasks and that they have to choose the tasks as if flipping the coin has decided which task to perform.

The experiment started with a practice block of 60 trials, followed by 10 test blocks of 60 trials. There was a short break of approximately 30 s following each block. On each trial, a task-irrelevant affective picture was presented for 900 ms and disappeared. The picture type (positive, negative, neutral) varied from trial to trial and followed an unpredictable pattern. In order to investigate the immediate influence of valence and arousal on VTS, we presented the task-irrelevant affective pictures at the beginning of each trial. Subsequently, a neutral target stimulus was presented in the center of a black screen in ARIAL font, size 108. When a response was given or a maximum presentation time of 6000 ms had elapsed, the target stimulus disappeared. For incorrect responses the word "FOUT" (error) was presented for 1000 ms before the ITI of 100 ms started (for a schematic overview see **Figure 1**).

RESULTS

First, trials were categorized according to the task that was chosen on the basis of the response hands. Next, trials were classified as a task repetition or a task switch. First trials of a block, trials following an error and trials with RTs shorter than 50 ms were excluded from analysis (data loss: 13.3%). Because we were interested in immediate effects of valence and arousal on the ability to switch tasks, we wanted to avoid that our measures were contaminated by the influence of the affective picture presented on the previous trial. Therefore, we focused on trials following trials in which a neutral picture was presented. By comparing task choice and task performance on positive pictures, neutral, and negative pictures, we tested for the influence of valence, and by comparing positive and negative pictures with neutral pictures we tested for the influence of arousal.

TASK PERFORMANCE

RTs and error rates were subjected to a 2 (task transition: task-repetition or task switch) by 3 (trial type: positive, neutral, or negative) repeated measures ANOVA with an alpha-level of 0.05. RTs and accuracies of each cell of the design are shown in **Figures 2** and **3** respectively. For RTs, the main effect of task transition was significant, $F(1,43) = 60.11$, MSE = 19031, $\eta_p^2 = 0.58$, indicating that RTs were higher on task switches ($M = 767$ ms, SE = 28) than on task repetitions ($M = 635$ ms, SE = 26). The main effect of

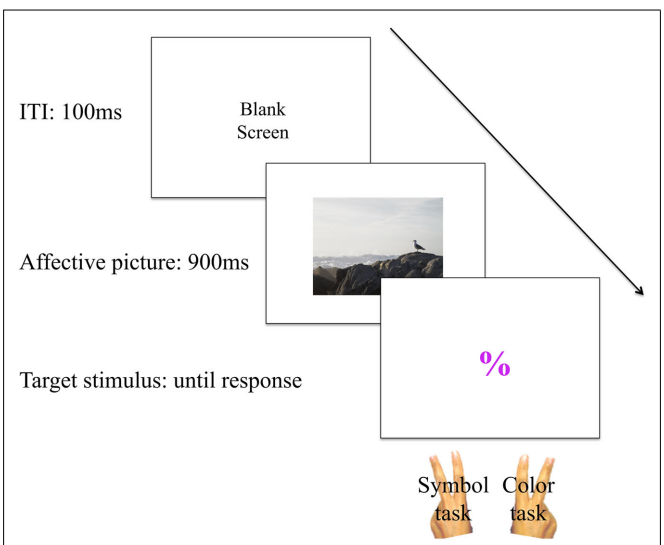

FIGURE 1 | Schematic overview of a trial.

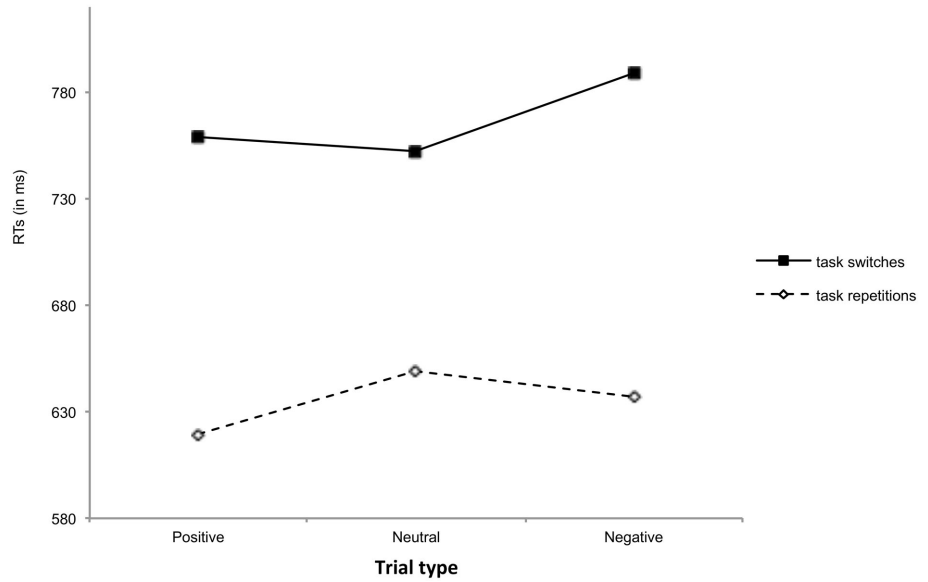

FIGURE 2 | Reaction times as a function of task transition and trial type.

trial type was significant, $F(2,86) = 2.57$, MSE = 4904, $p = 0.08$, $\eta_p^2 = 0.06$. Planned comparisons showed that RTs on positive trials ($M = 689$ ms, SE = 23) were faster than on negative trials ($M = 713$ ms, SE = 28), $F(1,43) = 5.17$, MSE = 4871, $\eta_p^2 = 0.11$, but did not differ from neutral trials ($M = 700$ ms, SE = 28), $F(1,43) = 1.14$, MSE = 4849, $\eta_p^2 = 0.03$. Also the RTs on neutral trials did not differ reliably from the RTs on negative trials, $F(1,43) = 1.42$, MSE = 4992, $\eta_p^2 = 0.03$. The difference between positive and negative trials indicates that positive valence facilitates general task performance. The interaction between task transition and trial type was significant[1], $F(2,86) = 3.12$, MSE = 4402, $\eta_p^2 = 0.07$ (see **Figure 2**). Planned comparisons showed that the switch cost was larger on positive (139 ms) than on neutral trials (104 ms), $F(1,43) = 2.85$, MSE = 4927, $p < 0.10$, $\eta_p^2 = 0.06$, but this difference was only marginally significant. Also on negative trials (152 ms) the switch cost was higher than on neutral trials, $F(1,43) = 5.48$, MSE = 4646, $\eta_p^2 = 0.11$. There was no difference between switch costs on positive and negative trials, $F < 1$, indicating that the valence of the affective picture did not affect the switch cost. In order to investigate the effect of arousal on the switch cost we collapsed both high arousal conditions (positive and negative trials) and compared the mean RTs with the mean RT of the low arousal condition (neutral trials). As the individual contrasts already suggested, this analysis showed that the switch cost was significantly larger with high-arousing pictures than with low-arousing pictures, $F(1,43) = 4.98$, MSE = 5170, $\eta_p^2 = 0.10$. Additional analyses showed that this higher switch cost was primarily caused by faster task repetitions, $F(1,43) = 4.33$, MSE = 2768, $\eta_p^2 = 0.09$, and not by slower task switches following arousing pictures, $F(1,43) = 1.88$, MSE = 7340, $p = 0.18$, $\eta_p^2 = 0.04$.

On the error rates we found that participants made more errors on task switches ($M = 0.07$, SE = 0.008) than on task repetitions ($M = 0.05$, SE = 0.004), $F(1,43) = 14.59$, MSE = 0.0025, $\eta_p^2 = 0.25$. The main effect of trial type was not significant, $F(2,86) = 1.54$, MSE = 0.0020, $p = 0.22$, $\eta_p^2 = 0.03$. The interaction between task transition and trial type was marginally significant, $F(2,86) = 2.55$, MSE = 0.0020, $p = 0.08$, $\eta_p^2 = 0.06$ (see **Figure 3**). Planned comparisons showed that the switch cost was marginally significantly larger on positive (0.027) than on neutral trials (0.016), $F(1,43) = 2.95$, MSE = 0.0017, $p < 0.10$, $\eta_p^2 = 0.06$. The switch cost on negative trials (0.047) was also higher than on neutral trials, $F(1,43) = 5.17$, MSE = 0.0020, $\eta_p^2 = 0.11$. No difference in switch cost was observed between the positive and negative trials, $F < 1$, again suggesting that the valence of the affective picture did not affect the switch cost. After collapsing positive and negative high-arousing pictures, an additional analysis showed that the switch cost was higher with high-arousing than with neutral pictures, $F(1,43) = 6.40$, MSE = 0.002, $\eta_p^2 = 0.13$. Additional analyses showed that this higher switch cost was caused by more errors during task switches, $F(1,43) = 6.88$, MSE = 0.002, $\eta_p^2 = 0.09$, and not by fewer errors during task-repetitions, $F < 1$. In order to differentiate between the arousal and the attention hypothesis it is important to mention that global performance, both on RTs and error rates, was never impaired following high-arousing pictures compared to neutral low-arousing pictures.

TASK CHOICE

In addition, the task choices were analyzed. For each trial type, the proportion of task repetitions and switches was calculated. Because the proportion of repetitions and switches are complementary, namely p(switches) = 1-p(repetitions), we only focused on the proportion of task repetitions. On these proportions we

[1]An additional analysis showed that gender did not modulate the interaction between trial type and task transition, $F(2,84) = 1.07$, $p = 0.34$. This also indicates that the use of erotic pictures for the male subjects did not play an important role and can thus be excluded as an alternative explanation for the absence of an influence of valence on the switch cost.

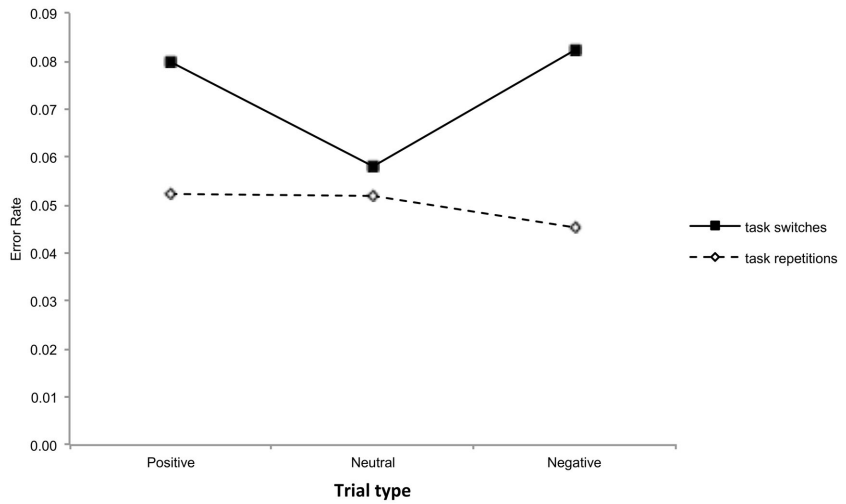

FIGURE 3 | Error rates as a function of task transition and trial type.

conducted a repeated measures ANOVA with trial type (positive, neutral, or negative) as single factor. The main effect of trial type was not significant, $F(2,86) = 1.40$, $MSE = 0.0035$, $\eta_p{}^2 = 0.03$, indicating that the proportion of task repetitions did not differ between positive ($M = 0.603$, $SE = 0.020$), neutral ($M = 0.595$, $SE = 0.020$), and negative trials ($M = 0.616$, $SE = 0.018$). After collapsing positive and negative high-arousing pictures, an additional analysis showed that the task-repetition bias did not differ between high-arousing and neutral pictures, $F(1,43) = 1.70$, $MSE = 0.004$, $\eta_p{}^2 = 0.04$.

DISCUSSION

In the present study we focused on the relation between emotional and cognitive control by investigating the influence of the valence and arousal of task-irrelevant pictures on cognitive flexibility during VTS. It was found that arousal specifically affected the switch cost and that valence affected global performance but did not affect the switch cost. The preference to repeat or switch was not affected by the arousal neither by the valence of the affective pictures.

The results support the arousal hypothesis as it showed that the switch cost increased following arousing stimuli. Both findings of a larger switch cost caused by facilitated task repetitions, as found for the response latencies, and a larger switch cost caused by impaired task switches, as found for the error rates, are in line with the predictions of the arousal hypothesis (Verguts and Notebaert, 2009). According to Verguts and Notebaert (2009) high-arousing pictures lead to larger switch costs because the stimulus-response associations related to the previously executed task-set become strengthened through increased Hebbian learning. As such, switching toward the alternative task becomes more difficult, while repeating the same task is facilitated (see also **Figure 4** for a schematic presentation). The pattern of results of the present study is remarkably similar to the findings of Braem et al. (2011) that showed that switch costs increased following conflict trials. The present finding that arousal induced by affective pictures can have a similar influence on the switch cost can be considered as

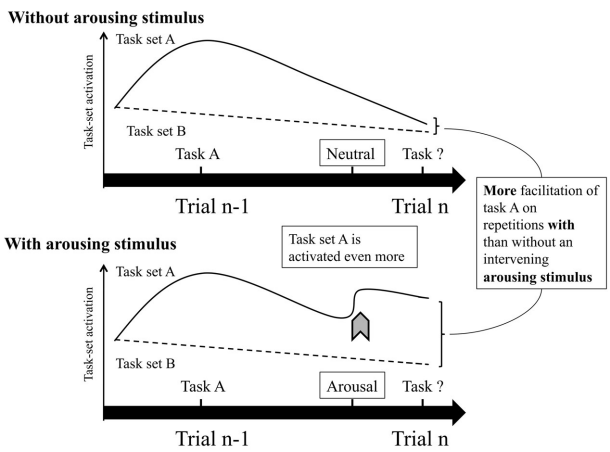

FIGURE 4 | Scheme of the application of the noradrenalin account of Verguts and Notebaert (2009) on the switch cost in VTS.

indirect support for the hypothesis of Braem et al. (2011) stating that reductions in switch costs following conflict trials are related to the level of arousal triggered by the experience of a response conflict, facilitating task repetitions, and impairing task switches.

At first sight, the finding that the switch cost was higher following arousing stimuli also seems to be in line with the attention hypothesis as proposed by Schimmack (2005) and De Houwer and colleagues (Verbruggen and De Houwer, 2007; De Houwer and Tibboel, 2010). These authors argued that arousal interferes with cognitively controlled behaviour, because arousal occupies the necessary cognitive resources. In the context of task switching this hypothesis entails that processes such as task-reconfiguration processes or processes necessary for interference control, which are especially important on task switches, suffer from a lack of available attentional resources caused by the arousing stimulus. As a consequence this hypothesis predicts that especially the

ability to switch tasks should suffer, leading to higher switch costs. In addition, this hypothesis entails that performance on task repetitions also should suffer because during task repetitions in a task-switching context, cognitive control is also important, although not as important as during task switches (e.g., Braver et al., 2003). However, the results showed that task repetitions following high-arousing pictures were never impaired, not on the response latencies and error rates. The results even showed that the higher switch costs observed on the response latencies were driven mainly by facilitated task repetitions. Both these findings cannot be accounted for by the attention hypothesis, since this hypothesis only predicts impaired performance in cognitively demanding situations (e.g., De Houwer and Tibboel, 2010). It is important to mention that, although the present data support the arousal hypothesis, we do not state that competition for attentional resources did not take place, since this effect is widely accepted and replicated in a large amount of studies (e.g., Fox et al., 2001; Schimmack, 2005; Wyble et al., 2008). We simply argue that the attention hypothesis cannot account for the observed facilitation on task repetitions following arousing pictures and that an additional mechanism, possibly related to the LC–NA system, also played an important role in the observed interaction between emotional and cognitive control.

In addition, we found that pictures with a positive valence improved global performance, but we did not observe that valence had an influence on the ability to switch tasks, both on the switch cost and the task-repetition bias. As already mentioned in the introduction, this finding does not correspond with a large amount of studies in which was reported that positive information affects cognitive flexibility (e.g., Isen and Daubman, 1984; Greene and Noice, 1988; Isen et al., 1992; Kuhl and Kazén, 1999; Bolte et al., 2003; Dreisbach and Goschke, 2004; Dreisbach, 2006). However, we believe it is difficult to compare the findings of these studies directly with the results of the present study, since in most of these studies the effects of arousal were not controlled for. In addition, as Cools et al. (2001) already pointed out, the effects of DA modulations also strongly depend on the task demands. In fact, most effects of valence have been reported in studies in which subjects were not asked to switch between tasks and cognitive flexibility was measured with different paradigms. In the single study that was designed to dissociate the effects of valence and arousal of affective information on the ability to switch tasks, performed by Dreisbach and Goschke (2004), was found that subjects were more able to adapt to a task switch when positive information was presented. However, the procedure used in that study differed strongly from the currently used procedure in three important aspects. First, they used the so-called intermittent instruction procedure, in which subjects had no free task choice and tasks were only switched occasionally (once in each block). Second, they did not investigate the impact of affective pictures on the switch cost directly, but they investigated the difference in performing the five tasks before a task switch and the five tasks following a task switch, which was indicated by a task cue. Therefore, we think it is possible that the study of Dreisbach and Goschke (2004) taps on a different component of cognitive control, not on the ability to switch tasks from trial to trial, but on the ability to adapt to changing task demands over a longer period of time. We found indirect evidence for this explanation by showing that the general performance improved following positive than negative affective pictures. In addition, this finding suggests that the valence of an affective picture affects multitasking ability and cognitive control on a more general level, possibly by phasic modulations of DA (Ashby et al., 1999; Cools et al., 2009). Third, in comparison with the present study, Dreisbach and Goschke (2004) did not mix positive and negative trials with neutral trials in a single block. This difference could have caused the failure in the present study to find an influence of valence, since it is possible that mixing positive and negative affect cancels out the short-term effects of valence. In sum, the inconsistencies between findings in the study of Dreisbach and Goschke (2004) and the present study concerning the effects of emotional valence suggest that more research is necessary in order to capture the critical conditions in which emotional valence has an influence on the ability to adapt flexibly to a changing environment.

The influence of arousal on the switch cost, and the hypothesis that this effect is caused by task-set strengthening, converges with the more recent assumption that the switch cost is mainly related to interference control that is needed to cope with persisting task-set activation (Wylie and Allport, 2000; Yeung and Monsell, 2003), and not by switch-specific task-reconfiguration processes (e.g., Rogers and Monsell, 1995). Interestingly, we found that the switch cost but not the proportion of task repetitions varied with arousal. In convergence with recent research on VTS, this suggests that the selection component and the execution component in VTS are underlain by distinct sets of processes and are tapping on different aspects of cognitive control (see also Arrington and Yates, 2009; Butler et al., 2011). More importantly, this finding suggests an important feature of the interplay between emotional and cognitive control, namely that not every aspect of cognitive control is necessarily influenced by arousal. More precisely, it seems that arousal only affects those aspects of cognitive control related to behavioral stability and reduced distractibility, such as processes responsible for interference control. Arousal helps avoiding interference of irrelevant task-sets by strengthening the currently relevant task-set. This hypothesis can be related to a VTS study of Butler et al. (2011) where it was observed that individual differences in working-memory capacity affected task performance but not task choice in VTS. Based on the finding that a key feature of working-memory capacity is the ability to cope with interference of irrelevant information (Kane et al., 2001), it thus seems that task performance is affected by arousal because it comprises interference control, while task choice does not.

In conclusion, the present study was the first to investigate the impact of arousal and valence of a task-irrelevant stimulus on the ability to switch tasks voluntarily. We found that presenting affective pictures affected the ability to switch between tasks. The data showed that arousal related to an affective stimulus made it more difficult to switch between tasks. This result is in line with the binding account of Verguts and Notebaert (2009) that states that phasic modulation of NA plays an important role in the functional overlap between emotional and cognitive control because an arousing signal triggers the noradrenergic

system that reinforces the previously executed task. In addition, we observed that presenting positive pictures only improved global performance but did not affect the ability to switch between tasks compared to negative pictures. This finding suggests that positive valence affects general multitasking performance but does not have an immediate influence on the efficiency of the processes that are necessary to switch between tasks from trial to trial.

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APPENDIX

Table A1 | An overview of the selected pictures of different subsets for gender, valence and arousal (the mean valence rating and the mean arousal rating of the IAPS pictures are presented in parentheses).

Female, positive/high: 8496, 8490, 8370, 8185, 8370	**(Mean valence = 7.77; mean arousal = 6.95)**
Female, neutral/low: 7500, 7224, 5500, 7234, 7130	(Mean valence = 4.89; mean arousal = 3.04)
Female, negative/high: 6250, 3400, 3500, 6510, 6540	(Mean valence = 2.12; mean arousal = 7.06)
Male, positive/high: 4002, 4180, 4220, 4250, 4290	(Mean valence = 7.94; mean arousal = 7.09.)
Male, neutral/low: 7500, 7224, 5500, 7234, 7130	(Mean valence = 4.89; mean arousal = 3.04)
Male, negative/high: 3000, 3010, 3530, 6260, 6350	(Mean valence = 2.28; mean arousal = 7.01)

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Muscle or motivation? A stop-signal study on the effects of sequential cognitive control

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Performance on cognitive control tasks deteriorates when control tasks are performed together with other control tasks, that is, if simultaneous cognitive control is required. Surprisingly, this is also observed if control tasks are preceded by other control tasks, that is, if sequential cognitive control is required. The typical explanation for the latter finding is that previous acts of cognitive control deplete a common resource, just like a muscle becomes fatigued after repeated usage. An alternative explanation, however, is that previous acts of cognitive control reduce motivation to match allocated resources to required resources. In this paper we formalize these muscle and motivation accounts, and show that they yield differential predictions regarding the interaction between simultaneous and sequential cognitive control. These predictions were tested using a paradigm where participants had to perform multiple stop-signal tasks, which varied in their demands on simultaneous and sequential control. Results of two studies supported predictions derived from the motivation account. Therefore, we conclude that the effects of sequential cognitive control are best explained in terms of a reduction of motivation to match allocated to required resources.

Keywords: cognitive control, resource depletion, ego-depletion, motivation, stop-signal task, stimulus response compatibility, formal models, multilevel analysis

INTRODUCTION

Cognitive control is essential for optimal everyday functioning. Unfortunately, the capacity for cognitive control is limited, as is evidenced from tasks that require simultaneous use of multiple control functions. For example, stop-signal inhibition deteriorates in tasks that also require inhibition of distracting stimuli (e.g., Verbruggen et al., 2005). Surprisingly, not only simultaneous, but also sequential demands on cognitive control degrade performance (Hagger et al., 2010). For example, stop-signal inhibition deteriorates if a preceding task requires control over food intake (Muraven et al., 2006). The usual account for these effects of sequential cognitive control[1] is that sequential tasks rely on one common resource, just as simultaneous tasks do. This common resource then becomes depleted, as a muscle becomes fatigued after repeated use (Muraven and Baumeister, 2000). However, an alternative account has also been put forward, namely that sequential cognitive control reduces motivation to allocate resources required to meet task demands (Hagger et al., 2010; Robinson et al., 2010).

It is unknown whether the "muscle" or the "motivation" account provides the best explanation for the effects of sequential cognitive control (Baumeister and Vohs, 2007; Hagger et al., 2010; Robinson et al., 2010). In the present paper we therefore aim to test their relative merits. First, we show by means of simple formal modeling, that the muscle and motivation accounts yield differential predictions on the interaction between simultaneous and sequential cognitive control. Second, we test these predictions using a paradigm that varied the demands on both simultaneous and sequential cognitive control. We do this by focusing on a key aspect of cognitive control, namely the ability to inhibit prepotent motor responses, as assessed in the stop-signal task (Logan and Cowan, 1984; Logan, 1994).

In the stop-signal task, participants are instructed to make a speeded binary response to a go signal, for example participants have to respond left to a left pointing arrow, and right to a right pointing arrow. Shortly after the onset of the go-signal participants occasionally receive a stop signal that requires them to stop the go response. The stop-task yields several informative indices of performance, among which the time required to respond to the go signal (go reaction time, RT), the percentage of choice errors, i.e., responding left if a right-hand response is required and vice versa, and, most importantly, an estimate of the time required to stop the response upon presentation of the stop signal (stop signal reaction time, SSRT, cf. **Figure 1**). A prolonged SSRT is a reliable index of suboptimal inhibitory performance and thus of impaired cognitive control (Logan and Cowan, 1984; Band et al., 2003).

The effects of *simultaneous* demands on cognitive control have been investigated by pairing the stop-signal task with the Eriksen flanker task, the Simon task, and with a spatially incompatible stimulus–response task. In the Eriksen flanker task, a central go stimulus is surrounded by distracting flankers. These flankers can

[1]Note that in this paper sequential control refers to sequential tasks, and not to sequential trials within a task.

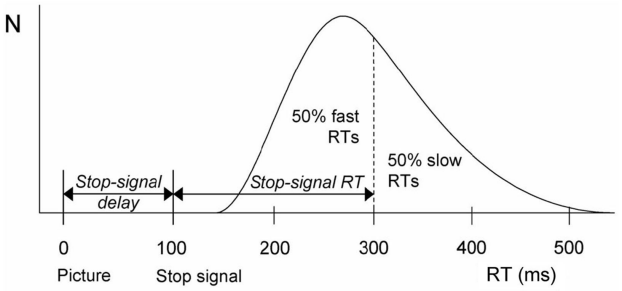

FIGURE 1 | Calculation of stop-signal RT (SSRT) according to the race model (Logan and Cowan, 1984). The black curve depicts the distribution of RTs on go trials (i.e., trials without a stop-change signal) representing the finishing times of the go process. Assuming independence of the go and stop processes, the finishing time of the stop process bisects the go RT distribution. Given that the response could not be stopped on nth percent of all stop-change trials (here at 50%), SSRT (200 ms) is calculated by subtracting the mean stop-change signal delay (100 ms) from the 50th percentile of go RT (300 ms).

be congruent with the correct response, i.e., both the central target and surrounding flankers indicate the correct response, or they can be incongruent, in which case the incorrect response signaled by the flankers needs to be inhibited (Eriksen and Eriksen, 1974). If the stop task is paired with the Eriksen flanker task, SSRT typically is increased on incongruent trials compared to congruent trials (Kramer et al., 1994; Ridderinkhof et al., 1999). In the Simon task, a go signal is presented at locations that may conflict with the location of the response (Simon, 1969). For example, a left pointing arrow requiring a left hand response is presented at the right side of the screen. If the stop task is paired with the Simon task, SSRT will increase on conflict trials (Verbruggen et al., 2005). On a spatially incompatible stimulus–response task participants have to execute a left hand response to a right pointing arrow and vice versa. Logan and Irwin (2000) observed an increased SSRT for incompatible as compared to compatible responses, but only for eye movements and not for hand movements. van den Wildenberg and van der Molen (2004a,b) also observed a prolonged SSRT for incompatible as compared to compatible responses, but only on a selective stop task, when participants had to inhibit to one stop signal and not to another. Finally, Morein-Zamir et al. (2006) showed, using an alternative paradigm, that inhibition deteriorated if stimulus–response mappings were spatially incompatible. In sum, these studies indicate that SSRT increases if the stop-signal task is paired with another task that also requires cognitive control, although there is mixed evidence for the combined effects of stopping and stimulus–response incompatibility.

The effects of *sequential* cognitive control are generally investigated by comparing performance under two conditions. In the "depletion" condition participants perform a cognitive control task that is preceded by another cognitive control task. In the "non-depletion" condition participants perform a cognitive control task that is preceded by a task that does not require cognitive control. For example, inhibition of emotional expression is assessed after participants either performed a "depleting" memory updating task or a "non-depleting" memory maintenance task (Schmeichel, 2007). A recent meta-analysis indicated that the effects of sequential cognitive control are very robust: previous acts of cognitive control diminish performance on a variety of subsequently executed cognitive control tasks (Hagger et al., 2010). It is commonly assumed that effects are limited only to subsequent tasks that require cognitive control whereas performance on subsequent tasks that do not require cognitive control is not affected (e.g., Baumeister et al., 1998). Effects are most pronounced for tasks that require "hot" control over emotional responses (Hagger et al., 2010), although a subset of studies also report effects on "cold" control, as is required in the stop-signal task (e.g., Muraven et al., 2006).

While the deleterious effects of sequential tasks requiring cognitive control are quite robust, the underlying mechanism is still unknown. Both a "muscle" and a "motivation" account have been proposed. The standard explanation, the muscle account, states that previous acts of cognitive control deplete a common resource (Muraven and Baumeister, 2000). This account provides a parsimonious explanation for the wealth of studies that find effects of sequential cognitive control. The alternative motivation account states that previous acts of cognitive control reduce motivation to allocate resources to meet current task demands (Hagger et al., 2010; Robinson et al., 2010). Consistent with this interpretation, it has been shown that increasing motivational incentives reduces the detrimental effects of sequential cognitive control (Muraven and Slessareva, 2003). In addition, these effects are reduced if motivation is primed (Martijn et al., 2007) or if the depleting task is intrinsically motivating (Muraven et al., 2008). Thus the motivation account may provide a viable alternative to the muscle account. The aim of the present paper therefore is to test the relative merits of the muscle and motivation account. In order to do so, we first derive simple formal models of these two competing accounts.

SIMPLE FORMAL MODELS OF MUSCLE AND MOTIVATION ACCOUNTS

In this section, we derive simple formal models for the muscle and motivation accounts. We do this by considering the situation in which participants have to perform two versions of the stop-signal task that vary both in their demands on simultaneous control and in their demands on sequential control. We will show that the muscle and motivation accounts yield differential predictions regarding the interaction between simultaneous and sequential cognitive control.

Demands on simultaneous control were manipulated by having participants perform a task using a compatible stimulus–response mapping and a task with an incompatible mapping; the latter task thus requires simultaneous cognitive control (Logan and Irwin, 2000; van den Wildenberg and van der Molen, 2004a,b; Morein-Zamir et al., 2006). Task order was randomized between participants. The factor Task is dummy coded as 0 (compatible task) and 1 (incompatible task). Presentation order is denoted by the factor Order, which is dummy coded as 0 (first task) and 1 (second task)[2].

[2]If the design includes more tasks, for example not only a compatible and an incompatible task but also a task with an arbitrary mapping, two dummy variables are required for Task, and two for Order.

A common assumption in resource theory (e.g., Kahneman, 1973; Sarter et al., 2006; Pessoa, 2009) is that cognitive control performance will decrease if allocated resources do not meet required resources. This assumption is supported by studies in which allocated resources were manipulated, for example by requiring simultaneous cognitive control (e.g., Kramer et al., 1994; Ridderinkhof et al., 1999; Logan and Irwin, 2000; van den Wildenberg and van der Molen, 2004a,b; Verbruggen et al., 2005; Morein-Zamir et al., 2006). We therefore assume that SSRT, an index of cognitive control, will increase if allocated resources do not meet required resources:

$$\text{SSRT} = \beta 0 + (\text{required resource} - \text{allocated resource}), \quad (1)$$

where the constant β0 denotes SSRT if there is a perfect match between required and allocated resources. Note that we assume that SSRT is a linear function of the discrepancy between required and allocated resources. We might have assumed a non-linear function *f*: SSRT = β0 + *f*(required resource-allocated resource). However, the functional form of the relationship between resources and performance is difficult to determine. More specifically, there is no evidence to suggest an adequate functional form for the relationship between resources and SSRT (e.g., Norman and Bobrow, 1975; Logan, 1997). Therefore we assume, for reasons of convenience, a linear function.

The required resource in Eq. 1 is a function of task:

$$\text{Required resource} = \beta 1 + \beta 2 * \text{Task}. \quad (2)$$

Since Task is dummy coded as 0 (compatible) and 1 (incompatible), β1 denotes the resource required for the compatible task and β2 denotes the task effect, which is expected to be positive. That is, incompatible tasks are expected to require more resources than compatible tasks.

Both the muscle and the motivation accounts share the assumptions underlying Eqs 1 and 2, yet they differ in their assumptions concerning allocated resources. The muscle model assumes that either task order or the control demands of the previous task affect allocated resources. The motivation model assumes that task order affects the motivation to match allocated resources to required resources. We will show that these assumptions yield mutually exclusive predictions on the interaction between task and order.

MUSCLE MODEL

The meta-analysis by Hagger et al. (2010) indicates that the effects of sequential cognitive control mainly originate in the fact that a cognitive control task is preceded by another cognitive control task, the control demand of this preceding task is of no influence. However, a meta-analysis is necessarily based on a between study comparison of demanding and less demanding depleting tasks. Such a between study comparison may not be very powerful, and therefore we will account for the possibility that control demands of the previous task are of influence. That is, we present two muscle models. In the first muscle model, consistent with Hagger et al., task order affects allocated resources. In the second muscle model, cognitive control demands of previous tasks affect allocated resources.

Muscle model 1: task order affects allocated resources

In the first muscle model, Order determines the amount of resources allocated to the current task:

$$\text{Allocated resource} = \beta 3 + \beta 4 * \text{Order}. \quad (3)$$

Since Order is dummy coded as 0 (first task) and 1 (second task), β3 is the allocated resource in the first task and β4 the Order effect, which is expected to be negative; that is, less resources are allocated to the second as compared to the first task. Substitution of Eqs 2 and 3 into (1) yields:

$$\text{SSRT} = (\beta 0 + \beta 1 - \beta 3) + \beta 2 * \text{Task} - \beta 4 * \text{Order}. \quad (4)$$

This model thus predicts a main effect of Task, a main effect of Order, yet no interaction. That is, Task effects do not increase or decrease with increasing Order.

Muscle model 2: control demand preceding task affects allocated resources

In the second muscle model, the control demand of the preceding task determines resources allocated to the current task. By design, a demanding task is necessarily preceded by a less demanding task, and therefore receives relatively high resources, whereas a less demanding task is preceded by a demanding ask, and thus receives relatively low resources. Consequently, this model predicts that task effects decrease with increasing Order.

More specifically, allocated resource is a function of the control demands of the previous task, therefore, we do not only have to include an effect of Order as in Eq. 3, but also an interaction effect of Order and Task:

$$\text{Allocated resource} = \beta 5 + \beta 6 * \text{Order} + \beta 7 * \text{Order} * \text{Task} \quad (5)$$

Remember, Order is dummy coded as 0 (first task) and 1(second task), Task is dummy coded as 0(compatible) and 1(incompatible). Therefore, β5, denotes resources allocated to the first task, this parameter is expected to be positive. The parameter β6 denotes the order effect (if the current task is compatible), it is expected that less resources are allocated to a second as compared to a first task, therefore this parameter is expected to be negative. The parameter β7 is the additional order effect if the current task is incompatible. Since the incompatible task is preceded by the less demanding compatible task, this parameter is expected to be positive.

Substituting of Eqs 5 and 2 into 1 yields:

$$\text{SSRT} = \beta 0 + (\beta 1 + \beta 2 * \text{Task}) - (\beta 5 + \beta 6 * \text{Order} + \beta 7 * \text{Order} * \text{Task})$$

That is:

$$\text{SSRT} = \beta 0 + \beta 1 - \beta 5 + \beta 2 * \text{Task} - \beta 6 * \text{Order} - \beta 7 * \text{Order} * \text{Task} \quad (6)$$

This model thus predicts main effects of Task and Order and an interaction between Task and Order. Since β2 is expected to be positive and (−β7) is expected to be negative, it is expected that Task effects will decrease with increasing Order.

MOTIVATION MODEL

Baumeister and Vohs (2007) indicated that motivation is required on cognitive control tasks "to achieve the goal or meet the standard." We therefore assume that a motivated individual will try to match allocated resources to required resources. More specifically, we assume that allocated resources are a proportion of required resources, and that this proportion increases with increasing motivation.

Robinson et al. (2010) argued that performing one cognitive control task may reduce motivation to allocate resources to a second cognitive control task. This is supported by the observation that the detrimental effects of sequential cognitive control are reduced in the presence of extrinsic or intrinsic motivation (Muraven and Slessareva, 2003; Muraven et al., 2008) or if motivation is primed (Martijn et al., 2007). We therefore assume that motivation to allocate resources decreases with increasing task order.

Given these assumptions, allocated resources are a fraction of required resources, where this fraction decreases with increasing Order. More specifically:

$$\text{Allocated resource} = \beta 8 * \text{required resource} + \beta 9 * \text{required resource} * \text{Order} \quad (7)$$

Since Order is dummy coded as 0 (first task) and 1 (second task), β8 denotes the fraction of required resource allocated to the first task, which is between zero and one, and β9 denotes the Order effect on this fraction, which is expected to be between −1 and 0. Required resource is defined in Eq. 2, its substitution into Eq. 7 yields:

$$\text{Allocated resource} = \beta 8 * (\beta 1 + \beta 2 * \text{Task}) + \beta 9 * (\beta 1 + \beta 2 * \text{Task}) * \text{Order} \quad (8)$$

Substitution of Eqs 2 and 8 into 1 yields:

$$\text{SSRT} = (\beta 0 + \beta 1 - \beta 8 * \beta 1) + \beta 2 * (1 - \beta 8) * \text{Task} - \beta 9 * \beta 1 * \text{Order} - \beta 9 * \beta 2 * \text{Task} * \text{Order} \quad (9)$$

That is, this model predicts a main effect of Task, a main effect of Order, and an interaction between Task and Order. Since all coefficients are expected to be positive except for β9, which is expected to be negative, Task effects are predicted to be most pronounced for the second task. That is, the motivation model predicts an interaction between Task and Order: Task effects increase with increasing Order.

STUDY 1

Although all models predict main effects of Task and Order, they differ in their predictions concerning the interaction of Task and Order. The first muscle model predicts that an interaction is absent, the second muscle model predicts that Task effects decrease with increasing Order, whereas the motivation model predicts that Task effects increase with increasing Order.

In study 1 we tested these predictions by having participants perform on three consecutive stop-signal tasks that varied in their demands on simultaneous cognitive control; a compatible stop task, an incompatible stop task, and a stop task in which the mapping between stimulus and response is arbitrary. The latter task was included to increase variation in both task and order and, thus, to increase the power of tests of their main and interaction effects.

STUDY 1 – METHODS

Participants

Thirty-four young healthy adults (23 women and 11 men, *M* Age = 21.10 SD = 3.78 years) participated in this experiment for course credit or a financial reward. All participants provided informed consent. The study was approved by the local ethics committee.

Stop-signal task

The experimental task was programmed using Presentation® software (Neurobehavioral Systems, www.neurobs.com). Participants were instructed to respond quickly and accurately in response to the identity of white, 8 mm high, go stimuli that were centrally presented against a gray screen background (go trials). Manual responses were made by pressing the "z" or "?" keys of the QWERTY computer keyboard with the left and right index finger, respectively.

Study 1 consisted of a 1-h 30 session in which participants completed three versions of the stop-signal paradigm (Logan and Cowan, 1984; Logan, 1994). Under the compatible and incompatible conditions, the go stimuli consisted of left and right pointing brackets ("<" and ">"), whereas the arbitrary go task incorporated X and O stimuli. Participants were instructed to respond with the hand indicated by the bracket (compatible task), or to respond with the other hand (incompatible task). In the arbitrary task, subjects pressed left to an O and right to an X (or vice versa). Order of the three conditions was counterbalanced between participants.

Go stimuli were presented pseudo-randomly, with the constraint that they signaled left- and right-hand responses equally often. The presentation of go signals was response-terminated or presented with a maximum of 1975 ms. Intervals between subsequent go signals varied randomly but equiprobably, from 1525 to 1975 ms in steps of 50 ms. During these inter-stimulus intervals, a white fixation cross (3 mm in diameter) was presented.

Unpredictably, the white go-signal changed to red on 25% of the trials, upon which the response had to be inhibited (stop trials). A staircase-tracking procedure dynamically adjusted the delay between the onset of the stop signal to control inhibition probability (Levitt, 1971). That is, after a successfully inhibited stop trial, stop-signal delay on the next stop trial increased by 50 ms, whereas the stop-signal delay decreased by 50 ms on the next stop trial when the participant was unable to stop. The initial stop-signal delay was set to 200 ms, the minimum and maximal delay were 50 and 950 ms respectively. This algorithm ensured that responses were successfully inhibited in about half of the stop trials.

Stop signal reaction time was estimated using the integration method (Logan, 1994; Band et al., 2003, p. 215; cf. **Figure 1**). Estimation of SSRT relies on the race model which assumes independence between going and stopping processes (Logan and Cowan, 1984). If the latency of go-signal processing is affected by stop-signal processing, then the assumption of "functional" or "context"

independence is violated (e.g., Logan, 1994; Ridderinkhof et al., 1999; Band et al., 2003). Simulation studies by Band et al. (2003) showed that the race model yields reliable estimates of SSRT despite context dependence between stopping and going (cf., Ridderinkhof et al., 1999). The premise of "stochastic" independence seems more critical (Logan and Cowan, 1984; Logan, 1994). Stochastic independence refers to the condition that stopping and going are not correlated (i.e., go RT and SSRT are independent random variables). Again, extensive simulation studies performed by Band et al. (2003) demonstrated that the race model is quite robust, even against violations of stochastic independence.

Each of the three stop-task versions consisted of five blocks of 96 trials, the first of which served as a practice block to obtain stable performance. This is especially important to avoid negative carry-over effects between compatible and non-compatible tasks. After each block, mean go RT, SD of mean go RT, percentage correct responses and percentage unsuccessful inhibitions were presented on screen. To ensure that the tracking algorithm worked properly, participants were given additional feedback when the percentage of failed inhibition was below 30% or above 70%. In case failed inhibits were below 30% participants were instructed to react as fast as possible to go stimuli. In case of failed inhibits above 70% participants were instructed to keep reacting as fast as possible to the go stimuli, but also to try and withhold their response to the stop stimuli.

Data analysis

The dependent variable of primary interest was SSRT. In addition we analyzed go RT and the percentage of choice errors. The latter two dependent variables were included to test the assumption that effects of order are limited to a decline in cognitive control processes and do not extend to more basic processes like responding to the go stimulus (Baumeister et al., 1998). Task and Order served as independent variables. The design cannot be analyzed using a regular factorial repeated measures ANOVA since each task is only administered once, i.e., there would be many missing values. This design can be analyzed, however, in a straightforward manner by a multilevel analysis (e.g., Snijders and Bosker, 1999) as implemented in SPSS MIXED[3]. All statistical tests were two-tailed.

STUDY 1 – RESULTS

No SSRT outliers (below or above 3 SD's of the mean) were present and therefore all data were analyzed. Analyses included the factors Task and Order and their two-way interaction.

Figure 2, left hand panel, shows SSRT as a function of Task and Order. There was a main effect of Task [$F(2, 24.5) = 3.57$, $p = 0.04$]. SSRT was prolonged on arbitrary ($p = 0.03$) and incompatible tasks ($p = 0.02$) as compared to the compatible task, with no further differences between tasks. There was a main effect of Order [$F(2, 66.34) = 4.36$, $p = 0.02$], SSRT was increased for order 3 ($p < 0.01$) as compared to order 1, with no further significant differences. The crucial interaction between Task and Order was not significant [$F(4, 62.54) = 1.04$, $p = 0.39$]. However, follow-up tests for each level of Order, separately, indicated that a Task effect was only present at order 3 [$F(2, 21.24) = 4.87$, $p = 0.02$][4]. That is, for order 3 SSRT was increased for the incompatible task ($p = 0.02$) and the arbitrary task ($p = 0.04$) as compared to the compatible task, with no further differences between tasks (cf. **Figure 2**). In sum, these results indicate that SSRT lengthened on arbitrary and incompatible tasks (Task effect) and that SSRT was enhanced for the final task relative to the preceding tasks (Order effect). The results indicate that the Task effect was only present for order 3, although the omnibus Task∗Order interaction effect was not significant.

[3]The error covariance structure was left unspecified, just as in a regular multivariate approach to a repeated measures ANOVA. That is no sphericity or other restrictive assumptions were imposed on the Task covariance structure. Estimates were derived by Maximum Likelihood.

[4]Analyses at each level of Order separately may yield unstable estimates of the Task covariance structure. Therefore, all such analyses were repeated with a diagonal covariance matrix, which yielded comparable results.

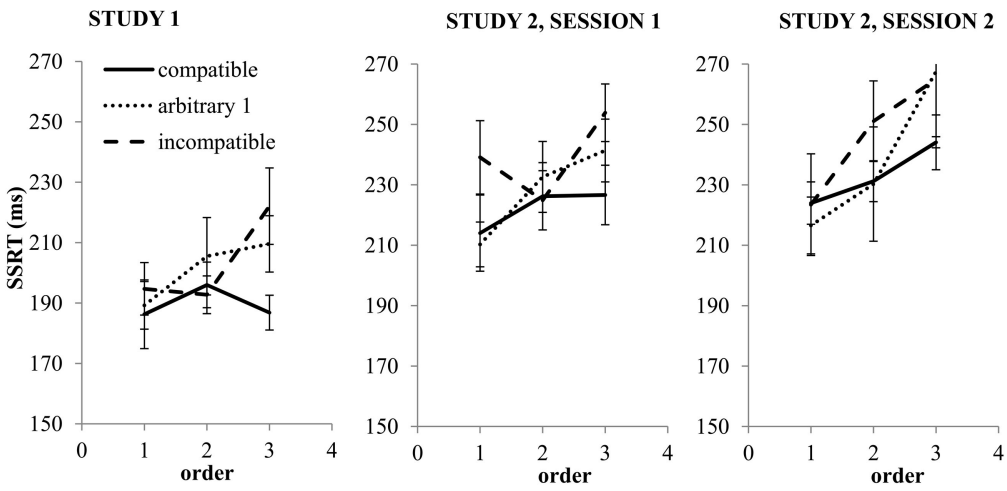

FIGURE 2 | Mean SSRT (error bars denote ± 1 SEM) as a function of Task (compatible, arbitrary 1 or incompatible) and as a function of task order (either first, second, or third task). The left hand panel concerns study 1, the middle panel study 2, session 1 and the right-hand panel study 2, session 2.

There was a main effect of Task on go RT [$F(2, 32.59) = 12.11$, $p < 0.01$]. Go RT was longer for the arbitrary task, as compared to the compatible ($p < 0.01$) and incompatible ($p < 0.01$) task, with no further differences between tasks. There was a main effect of Order [$F(2, 62.26) = 5.69$, $p < 0.01$], Go RT was shorter for order 3, as compared to order 2 ($p < 0.01$) and order 1 ($p = 0.02$), while the latter two did not differ. The interaction between Task and Order was not significant [$F(4, 56.57) = 1.81$, $p = 0.14$]. Follow-up tests for each level of Order separately, yielded no effects of Task.

There was a main effect of Task on percentage of choice errors [$F(2, 33.77) = 6.03$, $p = 0.01$], this percentage was higher for arbitrary ($p = 0.01$) and incompatible ($p < 0.01$) tasks as compared to the compatible task, with no further differences between tasks. There was no main effect of Order [$F(2, 39.91) = 1.40$, $p = 0.26$], nor an interaction between Task and Order [$F(4, 55.71) = 0.07$, $p = 0.99$].

In sum, the results on go RT and percentage of choice errors indicate that the basic go process is not affected by increasing Order. On the contrary, go RT was shortest on the final task while accuracy was maintained.

STUDY 1 – DISCUSSION

The two muscle models and the motivation model yield differential predictions on the interaction between Task and Order. The first muscle model, assuming that allocated resources depend on task order, does not predict such an interaction. The second muscle model, assuming that allocated resources depend on the cognitive control demands of the previous task, predicts that task effects decrease with increasing order. The motivation model, in which task order determines the motivation to match allocated resources to required resources, predicts that task effects increase with increasing order.

The results of Study 1 do not support the second muscle model. Consistent with the meta-analytic results of Hagger et al. (2010), the effects of sequential cognitive control do not depend on the cognitive control demands of the previous task. The results of Study 1 do not allow for a decisive conclusion regarding the relative merits of the first muscle model and the motivation model. As predicted by the first muscle model, the omnibus interaction between Task and Order was absent. However, as predicted by the motivation model, follow-up analyses did indicate that Task effects on SSRT were most pronounced, and actually were only present, for the third task.

Stop signal reaction time did not only lengthen if the stimulus response mapping was incompatible as compared to compatible, but also when the mapping was arbitrary (i.e., respond left to O and respond right to X). Two explanations have been put forward for the observation that arbitrary mappings are more difficult to inhibit than compatible mappings (Huizenga et al., 2009). First, an arbitrary mapping may initially activate competing responses associated with both hands, responses that need to be inhibited until the appropriate mapping is determined. On stop trials, this inhibitory mechanism then competes with stop-signal inhibition for common resources. Second, maintenance of an arbitrary stimulus–response rule in memory may require cognitive control resources in addition to the resources needed for stopping. Maintenance of an arbitrary rule then competes with inhibitory functioning for common resources. Note however, that maintenance of information in memory is generally not considered as a component of cognitive control, in contrast to the manipulation of information in memory. For example, digit span forward, which only requires maintenance, is not considered as a cognitive control task, whereas digit span backward, which requires manipulation, is considered as a cognitive control task (e.g., Schmeichel, 2007). Therefore the second interpretation offered above is not very plausible. Notwithstanding this theoretical argument, it still is necessary to test these two explanations empirically.

STUDY 2

The first study did not yield conclusive evidence supporting the first muscle model vs. the motivation model. That is, an omnibus interaction effect of Task and Order was lacking, supporting the first muscle model, whereas follow-up tests did indicate that Task effects were only present for order 3, supporting the motivation model. The absence of an omnibus interaction effect on SSRT might be due to the fact that our compatibility manipulation was not very effective. Participants were required to respond to the direction indicated by centrally presented arrows (i.e., symbolic mapping manipulation) rather than the location of the stimulus itself; e.g., left- vs. right-positioned stimuli requiring a left vs. right response, or vice versa (i.e., spatial mapping manipulation). A spatial mapping manipulation might yield more pronounced differences between compatible and incompatible tasks (cf. Logan and Irwin, 2000, eye movement condition).Therefore, in our second study we used a spatial mapping manipulation. If this stronger manipulation does not yield an interaction between Task and Order, this will provide evidence for the first muscle model. If it does yield an interaction, more specifically, if Task effects increase with increasing Order, this will yield evidence for the motivation model.

The first study indicated that arbitrary stimulus response mappings were more difficult to inhibit than compatible mappings. This might be due to the fact that arbitrary mappings require cognitive control resources to inhibit competing responses until the mapping is determined. Alternatively, it might be due to the fact that cognitive control resources are required to maintain a mapping in memory. In order to further investigate this issue, we employed in our second study an additional manipulation that increased memory load but did not increase response competition. That is, the arbitrary condition was now extended to include three levels in which either one, two, or three characters were mapped to each response hand. If this manipulation does not affect SSRT, it provides evidence for the response competition explanation. If this manipulation would affect SSRT, it provides support for the memory explanation.

Our second study thus featured five stop-signal tasks: a spatial compatible task, a spatial incompatible task and three arbitrary tasks that differed in memory load. These tasks were presented in two sessions on separate days. Inclusion of two sessions allows for an additional test of the muscle vs. motivation models. According to muscle models, resources would be replenished at the beginning of the second session, since people had the opportunity to rest (Tyler and Burns, 2008) and to eat (Gailliot et al., 2007). Therefore a session effect, more specifically, a decrease in cognitive control

performance in the second session is inconsistent with a muscle model. Such a finding would, however, not necessarily be inconsistent with the motivation model, since motivation may still decrease between sessions.

In sum, in our second study we addressed three questions. First, we investigated whether task effects on SSRT increase with increasing order. If so, this would provide evidence for the motivation model. Second, we investigated whether arbitrary memory load affects SSRT. If so, this would provide evidence for the memory maintenance, instead of response competition, explanation of difficulties in inhibiting arbitrary mappings. Finally, we determined whether session affects SSRT. If so, this will provide additional evidence for the motivation model.

STUDY 2 – METHODS

Participants

Forty-one young healthy adults (25 women and 16 men, *M* Age = 22.95 SD = 6.01 years) participated in this experiment for course credit or a financial reward, they did not participate in Study 1. All participants provided informed consent. The study was approved by the local ethics committee.

Stop-signal task

Task characteristics are similar to those of Study 1, except for the following modifications. Participants performed five stop-signal tasks. In the spatially compatible task, go stimuli consisted of a + sign presented either 1.6 cm left or right of fixation point, and participants were instructed to execute a spatially compatible response. The same stimuli were used in the spatially incompatible task but participants were then instructed to execute a spatially incompatible response. In the arbitrary task with a memory load of 1, an X or O was presented, were each character was mapped on one response hand. In the arbitrary task with a memory load of 2, an H and S were mapped on one response hand and a W and T on the other response hand. In the arbitrary task with a memory load of 3 an M, Y, and U were mapped on one response hand and an A, I, and V on the other. All characters were symmetrical along the vertical axis, and thus unrelated to response side.

Stop-task version (compatible, arbitrary 1, arbitrary 2, arbitrary 3 and incompatible) was counterbalanced between subjects. The experiment consisted of two sessions on separate days. Participants were free to choose whether they would perform two or three tasks during the first session.

Data analysis

The data analysis proceeded along the same lines as in Study 1. Again we did not only analyze SSRT, but also go RT and percentage of choice errors, to determine whether effects of sequential cognitive control were confined to cognitive control processes (SSRT) or extended to more primary processes (Go RT and percentage of choice errors).

STUDY 2 – RESULTS

Data of three participants were removed because SSRT exceeded 3 SD around the mean[5]. The analysis included effects of Task (five levels), Order (three levels), Session (two levels), and their two and three way interactions.

Figure 2, middle and right-hand panel, depicts SSRT as a function of Task, Order, and Session. There was a main effect of Task on SSRT [$F(4, 47.69) = 3.91$, $p < 0.01$]. SSRT was longer for the incompatible task, as compared to the compatible task ($p < 0.01$), the arbitrary 2 task ($p = 0.02$), and the arbitrary 3 task ($p < 0.01$). All other differences between tasks were non-significant. More specifically, and important for our second question, the arbitrary tasks did not differ significantly from each other (all *p*-values >0.1). There was a main effect of Order [$F(2, 99.10) = 16.54$, $p < 0.01$]. SSRT tended to be longer for Order 2 than Order 1 ($p = 0.09$) and was longer for Order 3 as compared to Order 2 ($p < 0.01$) and Order 1 ($p < 0.01$). Important for our third question, there was a Session effect; SSRT was longer for the second as compared to the first session [$F(1, 119.14) = 26.29$, $p < 0.01$]. Crucially for our first question, there was an interaction effect between Task and Order [$F(8, 68.74) = 3.11$, $p < 0.01$]. Follow-up tests for each level of Order, separately, indicated that there was no Task effect associated with Order 1 [$F(4, 10.88) = 1.86$, $p = 0.19$] nor for Order 2 [$F(4, 10.27) = 2.36$, $p = 0.12$], but there was a nearly significant Task effect for Order 3 [$F(4, 14.39) = 2.87$, $p = 0.06$]. Follow-up tests for Order 3 indicated that SSRT associated with the incompatible task was significantly lengthened as compared to the compatible task ($p = 0.04$) and the arbitrary 2 ($p < 0.01$) and arbitrary 3 ($p = 0.02$) tasks. In addition, SSRT on the arbitrary 1 task was significantly longer as compared to the arbitrary 2 task ($p = 0.04$), with no further differences between tasks. All other interactions were non-significant.

To summarize, these results indicate that Task effects on SSRT increase with increasing Order, yielding support for the motivation model. Second, arbitrary memory load does not affect SSRT, yielding support for the response competition explanation of prolonged SSRT for arbitrary mappings. Third, there was a pronounced session effect, which provides further evidence for the motivation model.

Obviously, there was a main effect of Task on go RT [$F(4, 44.22) = 52.80$, $p < 0.01$]. Go RT was longer on the incompatible task than on the compatible task ($p < 0.01$), and was longer on arbitrary tasks than on the incompatible and compatible tasks (all *p*s < 0.01). Go RT increased with arbitrary memory load (all *p*s < 0.01), except for the difference between the arbitrary 2 and arbitrary 3 condition, which was not significant. The main effect of Order did not reach significance [$F(2, 105.66) = 2.61$, $p = 0.08$]. Follow-up tests indicated, however, that Go RT was significantly longer on the first as compared to the third task ($p < 0.01$). Go RT was also significantly longer in the first as compared to the second session [$F(1, 113.27) = 8.90$, $p < 0.01$]. All other interactions were not significant.

Obviously, there was a main effect of Task on the percentage of choice errors [$F(4, 45.37) = 17.81$, $p < 0.01$]. Percentage of errors was higher on the arbitrary than on the compatible and incompatible tasks (all *p*s < 0.01) and increased with arbitrary memory load

[5]One participant had short SSRT in the arbitrary 2 task (third task, first session). One participant had long SSRT in compatible (first task first session) and incompatible (second task second session) tasks. One participant had short SSRT in the compatible task (first task first session). In these cases data were removed not only from the SSRT analysis, but also from the Go RT and percentage errors analysis.

(all $ps < 0.05$), except for the arbitrary 2 and arbitrary 3 condition, which did not differ significantly. There were no significant main effects of Order nor of Session. There was, however, a significant interaction between Task and Order [$F(8, 70.04) = 2.59, p = 0.02$]. Follow-up tests for each level of Order, separately, indicated that task effects were present at each level. All other interactions were not significant.

In sum, the results for go RT and percentage of choice errors indicate that repeated sequential control, either within or between sessions, does not degrade Go Task performance. On the contrary, go RT decreased from Order 1 to Order 3, and from Session 1 to Session 2. This speeding of responses was not accompanied by an increase in the percentage of choice errors.

GENERAL DISCUSSION

Two explanations have been put forward for the recurrent finding that performance on cognitive control tasks is degraded if such tasks are preceded by other tasks that also require cognitive control. The muscle account states that a common resource is depleted, just as a muscle becomes fatigued, after repeated usage. The motivation account states that repeated acts of cognitive control reduce motivation to match allocated resources to required resources.

The current study demonstrates that these two accounts can be tested using a paradigm where the demands on simultaneous and sequential cognitive control are varied systematically, that is, where both the factor task and the factor order are manipulated. We have formulated two muscle models, one in which allocated resources depend on order and one in which allocated resources depend on the cognitive control demand of previous tasks. In addition we have formulated a motivation model in which the motivation to match allocated resources to required resources is affected by order. We have shown that these three models yield differential predictions on the interaction between task and order. More specifically, the first muscle model does not predict an interaction, the second muscle model predicts that task effects decrease with increasing order, and the motivation model predicts that task effects increase with increasing order. Thus this formalization offered the possibility to test the relative merits of muscle and motivation accounts.

In addition, we have argued that the muscle and motivation account yield different predictions on the effects of cognitive control tasks performed in two separate sessions. A decrease in cognitive control performance over sessions would be inconsistent with the muscle account, whereas it would not necessarily be inconsistent with the motivation account, thus offering an additional test of the relative merits of these two accounts.

These predictions were tested in two studies where participants had to perform a series of stop-signal tasks varying in their demands on simultaneous cognitive control. The advantage of the stop-signal task over other cognitive control tasks is that it does not only yield an index of cognitive control functioning (SSRT), but also yields indices of more basic processes (go RT and percentage of errors). In this manner it is possible to assess whether the effects of sequential cognitive control are confined to control processes or extend also to more basic go processes.

The first study did not yield a significant task by order interaction on SSRT, supporting the muscle account, yet it did indicate that task effects were most pronounced on the final task, supporting the motivation account. The task by order interaction was present in the second study: as predicted by the motivation account, task effects increased with increasing order. In addition, the second study allowed for a test of session effects, which indicated that SSRT was prolonged during the second as compared to the first session, which provides additional evidence for the motivation account.

Collectively, these results suggest that the effects of sequential cognitive control are better explained by the motivation account than by the muscle account. This is in line with studies observing negligible effects of sequential cognitive control under conditions where motivation was enhanced or primed (Muraven and Slessareva, 2003; Martijn et al., 2007; Muraven et al., 2008) see also (Hagger et al., 2010; Robinson et al., 2010). Together these results suggest that it is more likely that the effects of sequential cognitive control originate in a depletion of motivation to match allocated resources to required resources, than in a mere depletion of resources itself.

It might be argued that the effects of order on inhibition are not due to the effects of sequential cognitive control, but rather to carry-over effects between sequential compatible and incompatible tasks. However, this alternative explanation is not very plausible. First, we trained participants before each task, to rule out carry-over effects. Second, if such negative carry-over effects would be present, this should be evidenced by a lengthening of go RT with increasing task order, which was not the case. Third, effects of order were present also for the arbitrary task, which cannot be affected by negative carry-over effects of stimulus–response mapping rules.

It might also be argued that the effects of order on inhibitory performance are related to automatization of primary task performance, as go RT decreased with order. That is, repeated presentation of a stimulus–response mapping can result in automatic associations that may be more difficult to inhibit (Schneider and Shiffrin, 1977). However, it has been shown that automaticity of stimulus–response mappings does not interfere with response inhibition as assessed by SSRT (Logan, 1982; Cohen and Poldrack, 2008). In addition, in our studies, stimulus–response mappings differ between tasks, therefore it is not very likely that associations became automatic. Therefore we do not consider it very likely that this alternative explanation in terms of automaticity holds.

In addition to our main finding, four other findings are worth mentioning. First, the results from both studies indicate that the detrimental effects of order and session on cognitive control performance (SSRT) are not paralleled by effects on more basic processes as indexed by go RT and error proportion. This provides evidence for the common assumption that the effects of sequential cognitive control are confined to cognitive control processes and do not extend to more basic processes (e.g., Baumeister et al., 1998).

Second, we tested two explanations for the Study 1 finding that arbitrary mappings were more difficult to inhibit than compatible mappings. First, an arbitrary mapping may require cognitive control resources to maintain a mapping in memory. Second, an arbitrary mapping may require cognitive control resources to inhibit both response hands until the mapping is determined. The

results support the second explanation since SSRT was not affected by a manipulation of memory load. Note, however, that in our second study, employing a spatial instead of a symbolic manipulation of compatibility, arbitrary mappings were not more difficult to inhibit than compatible mappings. This suggests that stopping responses to spatially compatible stimuli takes longer than stopping responses to symbolically compatible stimuli, a suggestion, which should be addressed in future empirical studies.

Third, SSRT was longer on incompatible as compared to compatible tasks. In previous studies, effects of stimulus response compatibility on SSRT were only found in relatively demanding situations, such as for inhibition of eye and not for hand movements (Logan and Irwin, 2000) and for selective and not for non-selective inhibition (van den Wildenberg and van der Molen, 2004a,b). In the present study we only observed significant effects of compatibility in the third task. This finding together with previous findings indicates that effects of stimulus response compatibility on SSRT only become evident in demanding situations.

Fourth, although the task, order, and interaction effects in the first and second study are qualitatively similar, SSRT seems to be prolonged in the second study, even in its first session (cf. **Figure 2**, compare left and middle panel). Since studies differed in the number of tasks, a potential explanation for this finding is that participants may allocate less resources, yielding a prolonged SSRT, if the number of prospective tasks increases (cf. Muraven et al., 2006; Tyler and Burns, 2009).

The current results have several broader implications. First, the present studies indicate that effects of sequential cognitive control can be found in a relatively "cold" cognitive control task, the stop-signal task. The effects are therefore not limited to tasks that are commonly used in resource depletion studies, i.e., "hot" tasks that require control over emotional responses. Second, the present results indicate that order is an important factor that should be considered in experimental studies that employ a within subjects design. That is, order may introduce variation that, if not accounted for in the analysis, may lower the power of statistical tests. The present study demonstrates that it is very easy to account for order, provided that one is willing to switch from a regular repeated measurements ANOVA to a multilevel analysis. Third, the finding that order has such a profound effect on cognitive control performance, has important implications for neuropsychological assessment, where patients often have to perform a battery of cognitive control tasks. It seems worthwhile to tailor statistical procedures for neuropsychological assessment (i.e. Huizenga et al., 2007) to account for such order effects. Finally, the important role of motivation warrants future experimental studies in which motivation is manipulated and prompts future studies in populations characterized by motivational deficits, for example in children with ADHD (e.g., Slusarek et al., 2001).

To conclude, two explanations have been proposed for the robust finding that repeated acts of cognitive control degrade performance. The muscle account states that repeated acts of cognitive control deplete resources, whereas the motivation account states that repeated acts of cognitive control reduce motivation to match allocated to required resources. Using a simple formal modeling approach, we obtained more evidence for the motivation account than for the muscle account. Therefore, we conclude that it is likely that the effects of sequential cognitive control originate in a reduced motivation to match allocated resources to required resources.

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Individual differences in heart rate variability predict the degree of slowing during response inhibition and initiation in the presence of emotional stimuli

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Response inhibition is a hallmark of executive control and crucial to support flexible behavior in a constantly changing environment. Recently, it has been shown that response inhibition is influenced by the presentation of emotional stimuli (Verbruggen and De Houwer, 2007). Healthy individuals typically differ in the degree to which they are able to regulate their emotional state, but it remains unknown whether individual differences in emotion regulation (ER) may alter the interplay between emotion and response inhibition. Here we address this issue by testing healthy volunteers who were equally divided in groups with high and low heart rate variability (HRV) during rest, a physiological measure that serves as proxy of ER. Both groups performed an emotional stop-signal task, in which negative high arousing pictures served as negative emotional stimuli and neutral low arousing pictures served as neutral non-emotional stimuli. We found that individuals with high HRV activated and inhibited their responses faster compared to individuals with low HRV, but only in the presence of negative stimuli. No group differences emerged for the neutral stimuli. Thus, individuals with low HRV are more susceptible to the adverse effects of negative emotion on response initiation and inhibition. The present research corroborates the idea that the presentation of emotional stimuli may interfere with inhibition and it also adds to previous research by demonstrating that the aforementioned relationship varies for individuals differing in HRV. We suggest that focusing on individual differences in HRV and its associative ER may shed more light on the dynamic interplay between emotion and cognition.

Keywords: heart rate variability, response inhibition, individual differences, emotion regulation, stop-signal task

INTRODUCTION

A vast body of literature has underscored the effect of emotions on executive control (Pessoa, 2008, 2009). For example, response latencies for solving mathematical problems are longer when very unpleasant pictures are presented compared to when moderate or low arousing emotional pictures are shown (Schimmack, 2005). People also need more time to name the color of emotional words compared to non-emotional ones (Williams et al., 1996; Phaf and Kan, 2007) or to withhold a planned response after the presentation of emotional compared to neutral stimuli (Verbruggen and De Houwer, 2007). Nevertheless, it remains unknown whether the above outcomes vary across individuals that differ in their ability to process emotional stimuli, an ability referred to as *emotion regulation* (ER; Gross, 1998). Subsequently, it is plausible that variations in ER may lead to differences in how emotions influence ongoing non-emotional tasks. In the present paper we focus on how individual differences in heart rate variability (HRV), a measure associated with ER (Appelhans and Luecken, 2006), may translate in differences in response initiation and response stopping.

The present study builds on recent findings indicating the impact of the presentation of emotional stimuli on the production and interruption of motor responses. First, Verbruggen and De Houwer (2007) showed that the presentation of high arousing emotional pictures lead to slowing as well as slowed stopping of a motor response compared to low arousing emotional pictures. Similar findings were reported by De Houwer and Tibboel (2010), who introduced emotional pictures in a Go/No-Go task[1] and found that participants had the highest error rates in no-go trials after the presentation of highly arousing negative stimuli. Subsequently, both studies indicate that emotional stimuli disturb both the production and inhibition of motor responses compared to neutral stimuli. Nonetheless, the question whether individual differences may moderate the effects of emotion on response inhibition and initiation remains unanswered.

A plethora of studies suggest that individuals differ in how effectively they can regulate incoming emotional information, an ability referred to as ER (Gross, 1998). A physiological measure that is associated with ER is HRV, which reflects the degree of cardiac activity adjustment to meet situational demands (Ruiz-Padial et al., 2003; Appelhans and Luecken, 2006; Thayer and Sternberg, 2006; Segerstrom and Nes, 2007). Technically speaking, HRV is the measure of the interplay between the sympathetic

[1] For a discussion over the differences between the stop-signal and the go/no-go task, see Verbruggen and Logan (2008a).

and the parasympathetic systems on the heart. The former system dominates over the latter during periods of stress and physiological arousal whereas the reversed pattern is observed in periods of safety and low physiological arousal. Low HRV has been associated with limited capacity of the organism to adapt to environmental changes (Appelhans and Luecken, 2006). This is because environmental modifications are not followed by quick adjustments of the interplay between the systems of the autonomic nervous system. That can also explain why reduced HRV and low vagal tone is associated with the cardiovascular symptoms of panic attacks, poor ER and behavioral stiffness (Friedman and Thayer, 1998; Thayer and Lane, 2000; Thayer and Siegle, 2002). In addition, lower HRV during rest periods is correlated with trait and self-perceived stress-induced anxiety (Lyonfields et al., 1995; Mujica-Parodi et al., 2009; Thayer et al., 1996). On the other hand, a high HRV is associated with quicker adjustment to situational diversions (Appelhans and Luecken, 2006). In the present paper, HRV serves as proxy of ER.

Considering the aforementioned findings on HRV as well as the work by Verbruggen and De Houwer (2007), we hypothesize that (1) individuals will have prolonged response and inhibition times after the presentation of high arousing emotional stimuli compared to low arousing ones and (2) individuals with high levels of HRV will have shorter response and inhibition times compared to individuals with low levels of HRV after the presentation of high arousing emotional stimuli whereas no differences are expected between individuals with different levels of HRV after the presentation of low arousing emotional stimuli. Our first prediction is theoretically in line with previous literature underscoring the influence of emotional stimuli on attention. A robust effect described in the literature on the relation between emotional stimuli and attention indicates that the presentation of high arousing emotional stimuli lead to longer attention times compared to low arousing stimuli (Lang et al., 1993; Schimmack, 2005). Subsequently, the emission or suppression of actions is expected to be delayed after the presentation of high arousing emotional stimuli compared to low arousing stimuli due to the prolonged attention times on the former ones. However, as indicated by our second prediction, this effect is expected to differ as function of the HRV level. Our second prediction is in line with recent studies demonstrating the relation between HRV level and performance in emotional tasks. For example, Johnsen et al. (2003) used a modified Stroop task with dental-related words that was administered to dental-phobic individuals. Results showed that individuals with higher parasympathetically mediated HRV could more effectively inhibit the attentional processing of the written words. Subsequently, for our study it is expected that subjects with high HRV will trigger or refrain from executing a response faster compared to subjects with low HRV. This is because participants with high HRV can quickly regulate the load of the incoming emotional information and shift their attention toward the main task. Participants with low HRV are expected to have longer response and stopping latencies compared to subjects with high HRV due to their longer attention engagement to incoming emotional information.

In sum, the present study has two goals. First, we attempt to replicate previous findings indicating the influence of the presentation of emotional stimuli on emitting and restraining from giving a response (Verbruggen and De Houwer, 2007). To this end, an emotional stop-signal task comparable to that of Verbruggen and De Houwer (2007) will be used to measure response initiation and inhibition latencies. Secondly, we will investigate whether individual differences in HRV predict the degree of slowing in response initiation and inhibition after the presentation of emotional stimuli, when compared to neutral stimuli. Individual differences in HRV will be measured during a relaxation period (Appelhans and Luecken, 2006).

MATERIALS AND METHODS

PARTICIPANTS

Sixty-two individuals participated in this experiment. Due to technical problems or incomplete answers, data from eight participants were excluded from further analyses. Hence, data from 54 participants (44 female, mean age 21.4, range 18–29) were analyzed. All participants had normal or corrected-to-normal vision and were naive to the purpose of the experiment. The ethical committee of the University of Amsterdam approved the experiment and all procedures were in accordance with the relevant laws and ethical guidelines. As a reward, participants received either research credits or a small amount of money (€14).

STIMULI, APPARATUS, AND QUESTIONNAIRES

We selected 320 negative and neutral stimuli from the International Affective Picture System (IAPS; Lang et al., 2005). Importantly, previous research with the emotional stop-signal task has shown no reliable differences in performance after the presentation of negative and positive valence pictures (Verbruggen and De Houwer, 2007). Thus, for the current experiment low arousing pictures with neutral valence served as neutral non-emotional stimuli. High arousing pictures with negative valence served as negative emotional stimuli (see **Table 1**). A DVD with all picture stimuli is available upon request.

All pictures were 500 pixels wide and 700 pixels high. Right or left pointing black arrows served as Go stimuli. All pictures and Go stimuli were presented on the middle of a 17″ inch computer screen (1680 × 1050 pixels) against a white background. For stimuli presentation, we used the software package Presentation (Neurobehavioural Systems Inc, www.neurobs.com).

Heart rate variability data were collected using three Ag–Ag electrodes. The electrodes were fixed using adhesive patches (3M Red Dot Electrode with Micropore Tape 2239). One electrode was placed below the right clavicle and one on the left side of the chest, just below the sixth rib. The ground electrode was fixed under the left clavicle. The signal was recorded using Vsrrp98, a software program developed at the University of Amsterdam, with a sampling rate of 1000 samples per second.

Table 1 | Overview of the selected pictures.

	Negative pictures	Neutral pictures
Mean valence (SD)	2.27 (2.41)	5.97 (6.16)
Mean arousal (SD)	5.07 (5.01)	3.17 (2.84)

The values in the parentheses are the SD.

Finally, for investigating the initial emotional states of participants, the Dutch version of the Positive and Negative Affect Schedule (PANAS) was used (Watson et al., 1988; Peeters et al., 1996). The Dutch versions of Spielberger's STAI–S and STAI–T were also administered in order to investigate the potential role of state and trait anxiety (Spielberger et al., 1970; Ploeg et al., 1981). Note that previous research has shown an influence of anxiety on HRV (Fuller, 1992; Miu et al., 2009).

PROCEDURE AND TASK

Upon arrival, participants were seated on a straight, high-back chair. After reading the information brochure and signing the informed consent, participants filled in PANAS, STAI–T, and STAI–S. Next, the heart rate electrodes were attached. Heart rate signal was collected during a 10-min period while participants watched a relaxing movie and listened to relaxing music coming from closed headphones. In the present study we used the film fraction from 5:33–15:33 s of the movie "Coral Sea dreaming" (Hannan, 1999).

Next, subjects performed an emotional stop-signal task (see **Figure 1**). The emotional stop-signal task consisted of *Go* and *Stop* trials. Each trial started with a black fixation cross that remained on the screen for 500–2000 ms (steps of 500 ms, $M = 1250$ ms). Next, a neutral or a negative picture was presented for 500 ms, followed by a right or left pointing arrow. Arrows remained on the screen for 200 ms and participants had to press the right or the left response button as fast and accurately as possible within a response time window of maximally 1500 ms. Response buttons were fixed on the chair's arms and participants responded using their thumbs. On Stop trials (30%), an auditory sound was presented and subjects had to withhold their motor response. There were 224 Go and 96 Stop trials, equally divided in two blocks of 160 trials each. The presentation of neutral and negative pictures, before the arrow, were equally balanced across all Go and Stop trials.

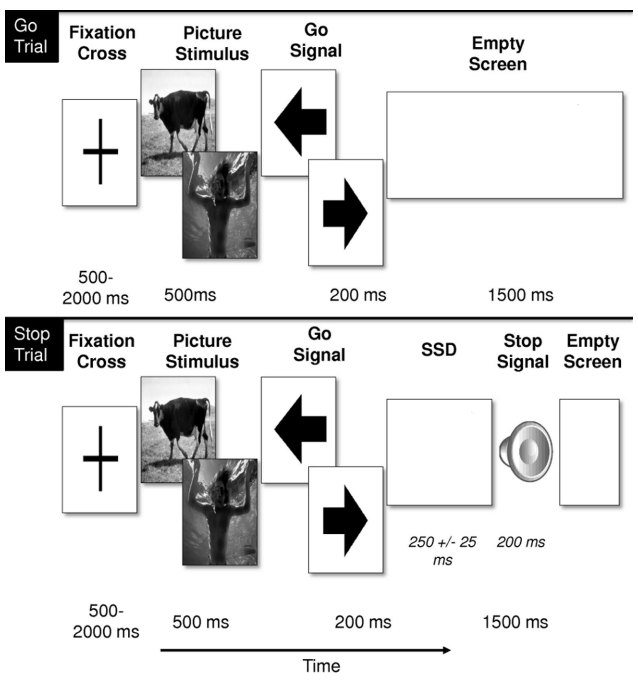

FIGURE 1 | Schematic design of the Go and Stop trials. Both trials started with a fixation cross, followed by a picture stimulus (neutral or negative). Next a left or right pointing arrow was presented (Go-signal). Each trial ended with a blank screen during which participants could give a response. On the Stop trials, the Go-signal was followed by an auditory Stop-Signal after a stop-signal delay (SSD).

The stop-signal delay (SSD) between the Go stimulus (the arrow) and the stop-signal (auditory tone) was dynamically adjusted. This was done separately for neutral and negative trials according to the staircase method and ensured convergence to P (inhibit) of 0.5. For example, if a stop-signal was presented on a neutral trial and the subject responded (Failed Stop), then the SSD for the neutral staircase was reduced by 25 ms on the subsequent neutral Stop trial; if the subject did not respond (i.e., Successful Stop), then SSD was increased by 25 ms. Initial SSD was set to 250 ms for both neutral and negative Stop trials. In order to familiarize with the task, participants had to complete a practice block consisting of 14 Go and 6 Stop trials involving neutral pictures. During the practice block, feedback for correct, incorrect, and late responses was given.

Subsequent to the emotional stop–signal task, participants rated all pictures in terms of valence and arousal using the self-assessment manikin (SAM; Lang, 1980; Bradley and Lang, 1994). Below the manikins, numbers from 1 to 9 were presented. For the valence ratings, 1 represented positive valence whereas nine represented negative valence. For the arousal ratings, 1 represented a stressed arousal state and 9 referred to a relaxed arousal state. Participants provided their ratings using the 1–9 buttons on a standard keyboard.

The experiment ended with participants filling in the PANAS scale as well as a short debriefing section.

MANIPULATION CHECK

Before performing our main analyses, we defined valence and arousal cut-off scores that would ensure that we would have enough remaining trials for the reliable computation of SSRT's (Band et al., 2003; Verbruggen and Logan, 2009) as well as having cut-off scores close to the middle of both scales (5). The only cut-off scores that fitted both criteria was that of six for valence – with scores equal or below that number indicating neutral ratings and above it negative – and four for arousal[2] – with scores equal or below that number indicating low arousal and above it high arousal. Our strategy resulted in removing 12.8% of the picture stimuli. The same picture stimuli were removed from all participants. It is noted that any other cut-off score would either result in having too few trials to compute reliable SSRT's or would result in values that would diverge more from the middle (point 5) of the valence or arousal scales.

BEHAVIORAL DATA ANALYSES

Error rates and median response times (RTs) were calculated for negative and neutral trials. All RTs above 2.5 SD from the mean

[2]It is noted that the arousal scale is reversed with 1 standing for relax ratings and 9 stressed ratings.

RT were removed from further analyses. This resulted in discarding 7.33% of the Go trials (2.15% neutral, 5.17% negative) and 3.6% (1.78% neutral, 1.84% negative) of the Stop Respond trials. For the Stop trials, SSRT was estimated separately for negative and neutral trials using the so-called "integration method" (Logan and Cowan, 1984; Verbruggen and Logan, 2009). The overall slowing effect was computed by subtracting the median RT (SSRT for Stop trials) on neutral trials from the median RT (SSRT for Stop trials) on negative trials. Paired t-tests were used to analyze the overall slowing effect on Go and Stop trials. Next, we examined the relationship between the slowing affect in Go trials and the degree of slowing in Stop trials with Pearson's correlation.

HEART RATE VARIABILITY ANALYSES

Prior to all analyses, the Electrocardiogram (ECG) signal was visually inspected and artifacts were corrected or if necessary removed. The interbeat intervals were imported to Kubios HRV Package (Tarvainen et al., 2009) and were again corrected for artifacts, using a medium artifact correction from the default options of the Kubios software. Finally, we estimated the root mean square of successive difference (RMSSD), a time domain measure of HRV (Appelhans and Luecken, 2006). Based on the RMSSD values, we performed a median split (Field, 2005) separating participants in groups with low and high HRV.

Before comparing groups' performance, we tested whether potential differences in initial emotional states diverged between groups. In this vein, we performed independent sample t-tests as well as Bayesian t-tests under the assumption of equality of variance. We specifically used Bayesian t-tests in order to test the plausibility that the two groups were equal in measures of interest. In Bayesian inference, the Bayes factor is computed which serves as an alternative to frequentist hypothesis testing. In the present study, the higher the Bayes factor the more likely the results have been occurred under the null hypothesis than the alternative hypothesis. For example, a Bayes factor of three shows that the null hypothesis is three times more likely than the alternative hypothesis[3]. We performed our computations by using the Bayesian models described in Wetzles et al. (2009) for two samples Savage Dikey t-tests with the assumption of equality of variance. The model and the R code (R Development Core Team, 2009) for computing the Bayes factor are available on http://www.ruudwetzels.com/sdtest. Our analyses showed that there were no differences between participants with low and high HRV in positive ($t_{31} = -1.29$, $p = 0.20$, $\mathrm{BF} = 2.44$) or negative ($t_{62} = 0.77$, $p = 0.44$, $\mathrm{BF}_{01} = 4.01$) mood. In addition, after correlating state and trait anxiety measures with the HRV measures for each group (Fuller, 1992; Miu et al., 2009), non-significant results emerged ($r < |0.20|$), indicating that neither state or trait anxiety differently influenced the HRV of each group. We also confirmed our results by performing Bayesian correlations (*all* $\mathrm{BF}_{01} > 3.7$). For our Bayesian correlation analyses we used the formulas provided by Jeffreys (1983) describing the comparison of a correlation coefficient to a suggested value (see Jeffreys, 1983, pp. 289–292). In our case, we compared correlation coefficients with zero (no relation).

[3]For more information on Bayesian inference and Bayes factor we refer the reader to Dienes (2011), Rouder et al. (2009), and Wetzles et al. (2009).

We then conducted a series of mixed ANOVA's with type of trial (neutral, negative) as a within-subject factor and HRV (high, low) as between subjects factor. This allowed us to examine the influence of HRV on performance in the emotional stop-signal task. In addition, two groups Bayesian t-tests were performed to test for similarity between the two groups under the assumption of equality of variance (Wetzles et al., 2009).

Linear regression analyses were also performed in order to investigate whether trait and state anxiety as well as positive and negative affect could predict response and inhibition times for each trial type. Finally, we assessed whether mood generally changed after completion of the stop-signal and whether these changes perhaps differed for our two HRV groups. This was achieved by performing mixed effects ANOVAs separately for each measure including time (first measure, second measure) as within-subject factors and HRV (high, low) as a between subjects factor.

RESULTS

THE PRESENTATION OF NEGATIVE STIMULI LEADS TO PROLONGED RESPONSE AND INHIBITION TIMES

Table 2 presents an overview of the behavioral data for Go and Stop trials. Few omissions during Go trials (combined errors, $M = 0.927\%$, $\mathrm{SD} = 1.625$) were observed. Additionally, participants gave more incorrect responses during negative trials compared to neutral trials ($t_{63} = 3.75$, $p < 0.001$), indicating that negative stimuli affect the accuracy in the stop-signal task. In line with previous findings, on Go trials participants responded slower after the presentation of negative pictures ($t_{63} = -4.1$, $p < 0.001$) compared to neutral pictures (see **Figure 2**, left panel). Moreover, negative stimuli prolonged the time needed to stop a planned response during successful Stop trials ($t_{63} = -4.8$, $p < 0.001$; see **Figure 2**, right panel), and the overall reaction time during Stop respond trials ($t_{63} = -3.8$, $p < 0.001$). The above findings provide a successful replication of the study by Verbruggen and De Houwer (2007).

Additionally, we tested the independence assumption between the Go and Stop process of the horse-race model (Logan and Cowan, 1984; Verbruggen and Logan, 2008b). Specifically, we investigated whether the slowing effect in Go trials is related to the degree of slowing during Stop trials. In accordance with the

Table 2 | Behavioral data during the emotional stop task on Go and Stop trials.

	Negative	Neutral
GO		
Median RT (ms)	500.8 (91.6)	488.7 (94.3)
Errors (%)	0.59	1.25
STOP RESPOND		
Median RT (ms)	477.8 (91.3)	461.6 (85.1)
STOP INHIBIT		
SSD (ms)	305.6 (128.3)	310.1 (131.3)
P inhibit (%)	0.55 (0.08)	0.51 (0.06)
SSRT (ms)	204.8 (79.7)	180.4 (68.5)

RT, response times; SSD, stop-signal delay; P inhibit, probability of inhibiting a response; SSRT, stop-signal reaction time.

independence assumption of the horse-race model, no relationship was found between the degree of slowing during Go and Successful Stop trials. On the other hand, when participants failed to Stop, the degree of emotional slowing was predictive for the slowing effects present on the Go trials ($t_{62} = 4.3$, $p < 0.01$).

HEART RATE VARIABILITY PREDICTS THE DEGREE OF SLOWING DURING RESPONSE INHIBITION AND INITIATION AFTER THE PRESENTATION OF NEGATIVE STIMULI

Next, we zoomed in on individual differences in HRV. To this end, we investigated whether high HRV subjects differed from low HRV subjects in the emotional stop-signal task. **Table 3** summarizes the behavioral results for each group.

First, we tested whether the high vs. low HRV group differed in their response speed in negative and neutral trials. The results showed that the two groups did not differ in the speed to initiate a response independent of the type of stimuli ($F_{1,62} = 0.015$, $p = 0.904$). There was also no difference for negative trials ($t_{62} = 0.484$, $p =$ n.s.) or neutral trials ($t_{62} = 0.313$, $p =$ n.s., $BF_{01} = 4.33$), respectively. Importantly, as shown in **Figure 3** (left panel), a strong interaction ($F_{1,62} = 11.392$, $p = 0.001$, $\eta_p^2 = 0.115$) emerged between the content of pictures presented on Go trials (i.e., neutral or negative) and HRV.

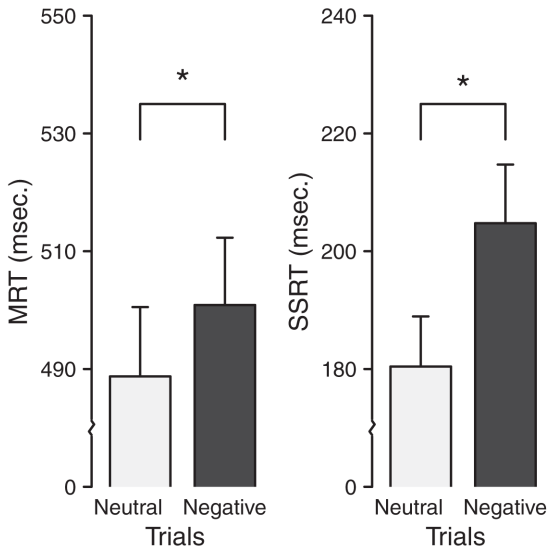

FIGURE 2 | Bar plots depicting performance in Go (left panel) and Stop (right panel) trials (Error bars represent SE). As it can be seen, participants needed more time to give but also stop their responses in the negative trials compared to neutral trials. *MRT*, median response time; *SSRT*, stop-signal reaction time. *$p < 0.001$.

Following our research questions, we investigated if the speed to inhibit a response also differed as a function of HRV. In line with the behavioral analyses reported before, participants took longer to inhibit their responses in negative trials compared to neutral ones ($F_{1,62} = 24.461$, $p < 0.001$, $\eta_p^2 = 0.283$). Notably, again this effect was different for each group as was shown from an Emotion × HRV interaction ($F_{1,62} = 5.613$, $p = 0.021$, $\eta_p^2 = 0.083$; see right panel of **Figure 3**). Specifically, subjects with low HRV compared to subjects with high HRV needed more time to inhibit their responses in negative trials ($t_{31} = 2.537$, $p = 0.014$) while no group differences were found in neutral trials ($t_{31} = 1.484$, $p =$ n.s.). The latter effect was also confirmed by our Bayesian analysis ($BF_{01} = 1.97$). Finally, participants with low HRV took longer to inhibit their responses than people with high HRV, regardless of the type of trial ($F_{1,62} = 4.486$, $p = 0.038$, $\eta_p^2 = 0.067$) with participants with high HRV being faster to inhibit than the participants with low HRV. Specifically, individuals with low HRV had shorter RTs on neutral compared to negative trials ($t_{31} = 4.675$, $p < 0.001$). The same pattern emerged for the High HRV although to a smaller extent ($t_{31} = 2.069$, $p = 0.047$), reflecting the main effect of emotion shown before. In line with our predictions, the outcomes above seem to indicate that HRV impacts the efficacy of stopping a planned response after the presentation of negative stimuli. Nevertheless, results should be interpreted with caution. As mentioned in Loftus (1978), non-crossover interactions are sensitive to the measurement scale (here SSRT's in millisecond) and can be potentially transformed away by a monotonic transformation of the scale (see also Luce and Tukey, 1964; Krantz and Tversky, 1971; Salthouse and Hedden, 2002).

Finally, we investigated whether positive and negative affect before and after the experiment was different among groups. Both groups reported a reduction in their positive affect although this reduction was bigger for the high HRV group ($t_{30} = 6.481$,

Table 3 | Behavioral data during the emotional stop task on Go and Stop trials.

	Negative – low HRV	Neutral – low HRV	Negative – high HRV	Neutral – high HRV
GO				
Median RT (ms)	506.4 (91.2)	485.03 (91.0)	495.3 (93.1)	492.5 (98.8)
Errors (%)	1.5	0.7	1.1	0.6
STOP RESPOND				
Median RT (ms)	480.8 (87.6)	458.3 (85.6)	474.8 (96.12)	464.9 (85.8)
STOP INHIBIT				
SSD (ms)	287.8 (124.7)	295.2 (125.2)	323.9 (131.3)	324.97 (137.49)
P inhibit (%)	54.91 (0.08)	0.51 (0.05)	0.54 (0.07)	0.51 (0.08)
SSRT (ms)	229.01 (88.36)	193.02 (68.89)	180.51 (62.37)	167.84 (68.87)

HRV, heart rate variability; RT, response times; SSD, stop-signal delay; P inhibit, probability of inhibiting a response; SSRT, stop-signal reaction time.

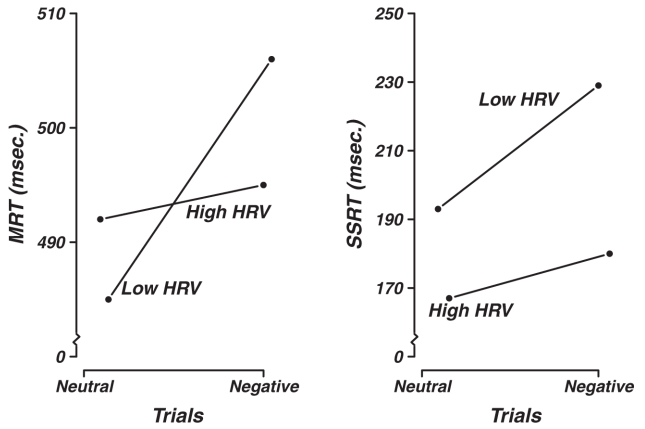

FIGURE 3 | Two 2 × 2 interactions between trial type and group. On the left panel, it is shown that participants needed more time to respond in the negative compared to neutral trials. On the right panel, the time to inhibit a planned response for each trial is depicted. Although participants needed more time to inhibit a response in negative compared to neutral trials, this increase in SSRT's is quicker for participants with low HRV. Please see text for further details on both interactions. *MRT*, median response time; *SSRT*, stop-signal reaction time, *HRV*, heart rate variability.

$p < 0.001$) rather than the low HRV group ($t_{29} = 2.785$, $p = 0.009$), as indicated by a significant cross over interaction between group and time ($F_{1,60} = 7.530$, $p = 0.008$). No differences between groups were found in positive affect for time point one ($t_{62} = -1.296$, $p = 0.200$) and two ($t_{60} = 1.084$, $p = 0.283$). Increase of negative effect was reported ($F_{1,59} = 9.034$, $p = 0.004$), although this effect did not differ as function of group ($F_{1,59} = 1.402$, $p = 0.241$).

DISCUSSION

The present study set out to investigate the influence of emotional stimuli on cognitive control processes such as the initiation or inhibition of motor responses. We also tested whether the initiation or inhibition of responses is different for participants with low and high HRV. Using an emotional stop-signal task, we found that after the presentation of high arousing negative pictures, participants needed more time to initiate and stop a planned response, compared to when neutral pictures were presented. These results are in line with previous research (Verbruggen and De Houwer, 2007) affirming a robust effect of the presentation of emotional stimuli on response initiation and inhibition. Furthermore, based on participants' HRV scores, we separated participants in groups with low and high HRV and compared the performance of the two groups in the emotional stop-signal task. In line with our predictions, performance in the emotional stop-signal task differed among participants with low and high HRV, indicating that individual differences in HRV translate into latency differences in action initiation and inhibition. While HRV is associated with ER, our findings provide evidence how performance in cognitive tasks is mediated by the perception and processing of emotional stimuli (Gross, 1998).

One explanation for the prolonged response and stopping times after the presentation of emotional stimuli is an attentional allocation toward emotional stimuli (Fox et al., 2001; Vuilleumier, 2005; Wyble et al., 2008; see also Estes and Verges, 2008 for a description of alternative explanations). In particular, emotional stimuli seem to attract and withhold attention longer compared to non-emotional stimuli (Öhman et al., 2001; Anderson, 2005). Although it remains unclear whether this effect is driven by the valence (Pratto and John, 1991; Estes and Verges, 2008) or the arousal dimension (Schimmack, 2005; Vogt et al., 2008) of each stimulus, it is assumed that the longer attentional engagement toward incoming emotional information results in slower responding in co-occurring non-emotional tasks (Lang et al., 1993; McKenna and Sharma, 1995; Buodo et al., 2002; Gronau et al., 2003). This explanation seems also to be in agreement with De Houwer and Tibboel's (2010) recent findings on a go/no-go task.

Attentional biases toward emotional stimuli can also be a potential explanation for the difference in performance between participants with low and high HRV, as previous studies have associated low HRV to poorer attentional control (Porges, 1992; Friedman and Thayer, 1998). Specifically, when RTs were investigated, individuals in the low HRV group were slower in giving a response after the presentation of negative compared to neutral pictures. This result can be attributed to the slow disengagement of attention from emotional stimuli which delayed the subsequent execution of overt responses (Buodo et al., 2002). On the other hand, people with high HRV had similar performance independently of what type of picture was presented. By taking attentional biases into account, we argue that individuals with HRV are more efficient in shifting their attention away from emotional stimuli and subsequently give a response.

In their seminal work, Logan and Cowan (1984) regarded stop-signal inhibition as a form of executive function. Accordingly, attentional biases can also be a potential explanation for the differences between groups to stop a planned response as well. Although the interaction on inhibition times can be potentially attributed to the measurement scale (Loftus, 1978; Salthouse and Hedden, 2002), our results show that people with low HRV needed more time to stop their responses compared to individuals with high HRV after the presentation of negative pictures. This finding supports the notion of an attentional bias in that individuals in the low HRV group had more difficulty in shifting their attention from emotional stimuli compared to subjects with high HRV. In support of this view, no differences between the two groups emerged after the presentation of neutral pictures.

An alternative explanation of participants' response and inhibition times could as well be that the use of negative and neutral emotionally stimuli could have resulted in some kind of negative emotional state, affecting individuals' performance[4]. Although valid, we think that this explanation contradicts the results on the PANAS, collected at the beginning and the end of the experiment. Specifically, both groups showed a decrease in positive affect as well as an increase in negative affect. Interestingly, the decrease in positive affect was steeper for the high HRV group as shown by a statistically significant interaction. This indicates that individuals

[4]We would like to thank an anonymous reviewer for suggesting this alternative explanation of the results.

with high HRV had stronger emotional reactions than the low HRV individuals in the end compared to the beginning of the experiment. At first sight, this may contrast our finding that individuals high in HRV are less affected by negative pictures during performing the stop-signal task than those displaying lower HRV. However, taken together, these findings merely show that individuals higher in HRV certainly are affected by the negative pictures, as indicated by the PANAS scores, but are nevertheless very well capable to effectively regulate those emotions and still focus on the main task demands (i.e., performing the emotional stop-signal task). This is in line with previous literature (Porges, 1992; Thayer and Lane, 2000) indicating that individuals high in HRV are not so much unaffected by certain stimuli, but display high adaptability to environmental demands (i.e., the experimental task). Nonetheless, due to the conflicting findings in the literature regarding HRV and mood states (for a review Pressman and Cohen, 2005) and the inability of our data to exactly address this question, future research is needed to investigate the exact relation between affect and HRV.

Although for both the response and stopping times, a significant interaction emerged, the pattern of the interactions was quite different. For example, no between group difference was found when participants had to initiate a response. Contrary to this finding, individuals with low compared to high HRV needed more time to inhibit a response in both neutral and negative trials, respectively. These results can be explained by the independence between the Go and Stop processes of the horse-race model (Logan and Cowan, 1984; Verbruggen and Logan, 2008a,b). Specifically, the horse-race model assumes that the Go and Stop process are stochastically independent. By measuring the level of HRV, we extend previous findings by showing that emotions differentially influence the Go and Stop process. To note, both our results and previous studies (e.g., Verbruggen and De Houwer, 2007) revealed that negative pictures lead to prolonged response and stopping times. Based on those results, it may be argued that in both cases emotional stimuli interrupt both the Go and the Stop process. The present results clearly oppose this assumption.

Nonetheless, the present study suffers from two main limitations. Firstly, although we selected our negative and neutral stimuli based on the normative IAPS ratings (Lang et al., 2005), preliminary analyses in which the mean valence and arousal ratings of the pictures were compared with participants' subjective ratings for each picture, showed that many pictures did not induce the desired emotion. Specifically, a high proportion of negative stimuli were regarded as low arousing by the participants, and a high proportion of neutral stimuli were regarded as positive valence. This led us to use less strict criteria in determining the cut-off scores in discarding pictures (see Materials and Methods). The aforementioned difference between IAPS mean ratings and participants' ratings could be attributed to the stronger content of a subset of pictures compared to others. This could have resulted in making individuals regard some negative pictures as low arousing and some neutral pictures as positively valence. Another important limitation is the low number of pictures included in the IAPS system that matched our valence and arousal ratings. As a result, we included neutral and negative pictures with a wide range of valence and arousal scores. Future studies should either use a smaller number of high arousing negative pictures or determine the final picture sets in pilot studies.

Secondly, although we used HRV during rest as an associative measure of ER (Lyonfields et al., 1995), this measure cannot be used as a direct index of ER. Future studies could overcome this shortcoming by measuring HRV during emotional situations as well and not only during resting periods.

In sum, the present study replicates the results of Verbruggen and De Houwer (2007) by showing that after the presentation of highly emotional pictures participants need more time to initiate and inhibit a response. We found that this effect interacted with the level of HRV while individuals with high HRV showed shorter response and stopping times after the presentation of emotional pictures compared to subjects with low HRV. These present results further our understanding of the intricate relation between ER and executive control functions by taking into account individual differences in HRV.

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The role of executive functions in the control of aggressive behavior

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An extensive literature suggests a link between executive functions and aggressive behavior in humans, pointing mostly to an inverse relationship, i.e., increased tendencies toward aggression in individuals scoring low on executive function tests. This literature is limited, though, in terms of the groups studied and the measures of executive functions. In this paper, we present data from two studies addressing these issues. In a first behavioral study, we asked whether high trait aggressiveness is related to reduced executive functions. A sample of over 600 students performed in an extensive behavioral test battery including paradigms addressing executive functions such as the Eriksen Flanker task, Stroop task, n-back task, and Tower of London (TOL). High trait aggressive participants were found to have a significantly reduced latency score in the TOL, indicating more impulsive behavior compared to low trait aggressive participants. No other differences were detected. In an EEG-study, we assessed neural and behavioral correlates of error monitoring and response inhibition in participants who were characterized based on their laboratory-induced aggressive behavior in a competitive reaction time task. Participants who retaliated more in the aggression paradigm and had reduced frontal activity when being provoked did not, however, show any reduction in behavioral or neural correlates of executive control compared to the less aggressive participants. Our results question a strong relationship between aggression and executive functions at least for healthy, high-functioning people.

Keywords: reactive aggression, executive functions, Eriksen Flanker task, stop-signal task, Taylor aggression paradigm, Tower of London

INTRODUCTION

There has recently been increasing interest in examining the similarities and interactions between cognitive and affective control and their underlying neural networks (Ochsner et al., 2002; Ochsner and Gross, 2005; Etkin et al., 2006; Egner et al., 2008; Chiew and Braver, 2011). Studies compare, for instance, interference control in an emotional vs. neutral context and typically find at least partly overlapping networks with a common hub in the anterior cingulate cortex (ACC; Etkin et al., 2006; Egner et al., 2008). These results lead to the critical question whether interindividual differences in the identified control "mechanisms" impact behavior in social interactions also. Here, we ask whether executive control functions relate particularly to the control of aggressive behavior.

Previous research on the relationship between executive functions and aggressive behavior in humans points mostly to an inverse relationship. This literature is limited though in some important aspects: firstly, most of this research focused on clinical groups, such as patients with personality disorders or neurological diseases (Anderson et al., 1999; Morgan and Lilienfeld, 2000; Brower and Price, 2001; Blair et al., 2006). This leaves the question open to what extent interindividual variability of aggressive behavior in psychologically and neurologically healthy people can be explained by differences in executive functions. Secondly, studies typically include only a few measures of executive functions, although executive functions comprise a range of different functions such as working memory, performance monitoring, and inhibition with only low intercorrelations between tasks designed to tap into each one of them (Miyake et al., 2000). In line with this, psychopathic patients show specific deficits in some but not all tests of executive functions (Blair et al., 2006). Lastly, only rarely did researchers assess the neural correlates of executive functions by means of functional magnetic resonance imaging (fMRI) or electroencephalography (EEG) although these measures might (i) be more sensitive to interindividual variability and (ii) provide clues about the underlying "mechanism" of the suggested interaction between executive functions and aggression. In this paper, we present behavioral and electrophysiological data addressing these issues.

Studies relating executive functions and aggression have mainly focused on psychiatric or neurological samples (Anderson et al., 1999; Raine et al., 2000; Brower and Price, 2001). Patients with antisocial personality disorder, psychopathy, or conduct disorder exhibit impaired performance in measures of executive functions (Morgan and Lilienfeld, 2000; Blair et al., 2006). In addition, evidence exists for structural alterations in prefrontal areas in patients with antisocial personality disorder, supposedly underlying these diminished executive functions (Raine, 1993; Raine et al., 2000). However, there are likely fundamental differences between psychiatric patients and individuals with increased, but non-pathological trait aggressiveness.

These clinical studies therefore cannot speak to the influence of executive functions on aggressive behavior in healthy people, which has been addressed in a couple of studies with non-clinical samples using laboratory measures of aggression (Hoaken et al., 2003; Pihl et al., 2003; Giancola, 2004). Hoaken et al. (2003), for instance, selected participants based on their performance in two executive function tasks (Spatial Conditional Association Learning Task and Self-Ordered Pointing Task), and demonstrated enhanced levels of laboratory-induced aggression in participants with lower executive functions. A different approach was chosen by Giancola (2004), who derived a composite score of executive functions from a neuropsychological test battery and related this to participants' behavior in the Taylor aggression paradigm (TAP). Although the hypothesis of less aggressive behavior in people with high executive functions was confirmed, the data also showed interactions with alcohol consumption and gender, such that the largest difference related to executive functioning was observed in intoxicated men, while aggressive behavior shown by women was independent of executive functions.

The approach of Giancola (2004), linking aggressive behavior to a global measure of executive functions, is questioned both by evidence for the diversity of executive functions (Duncan et al., 1997; Miyake et al., 2000; Friedman and Miyake, 2004; Braver and Ruge, 2006) and by studies demonstrating an influence of specific components of executive functions on aggressiveness. Blair et al. (2006) for instance, could differentiate the link between prefrontal functions and violence by showing that the performance of psychopaths is impaired particularly in tests sensitive for orbitofrontal dysfunctions (object alternation task), but unimpaired in tasks probing dorsolateral prefrontal cortex (spatial alternation task), or cingulate cortex functions (number-Stroop reading). Moreover, a common explanation for the relationship of executive functions and violence is related particularly to the concept of impulsivity (Hoaken et al., 2003; Krakowski, 2003), such that people with low executive functions are thought to be unable to inhibit aggressive response tendencies. This is supported by studies directly investigating the link between aggression and impulsivity in healthy humans. LeMarquand et al. (1998), for instance, reported performance differences in a Go/No-go task between low and high trait aggressive adolescents, such that more aggressive boys made also more commission errors. This raises the question whether the relationship between aggressiveness and executive functions can be explained with the concept of impulsivity only or, as suggested by Giancola (2004), goes beyond an impairment in inhibitory functions.

The present study thus aimed at extending the approach of Blair et al. (2007) by examining mentally healthy people and by administering a larger test battery for executive functions. Hence, we tested effects of participant's trait aggressiveness on performance in a range of neuropsychological tests. We largely followed the approach by Miyake et al. (2000), dissociating updating, shifting, and inhibtion as executive functions. We thus administered tests tapping the different functions updating (n-back), shifting (verbal fluency, trail-making test, task switching), and inhibition (Eriksen Flanker task, Stroop) and also included more complex executive functions tests, such as the Tower of London (TOL) and Wisconsin Card Sorting Test (WCST; Miyake et al., 2000). This enabled us to clarify the relationship between trait aggressiveness and specific components of executive functions.

Aggressiveness as assessed with trait questionnaires captures the interindividual variability in aggressive behavior only partly, however. In the second study, we therefore focused on participants who were characterized based on their laboratory-induced aggressive behavior in the TAP and examined neural and behavioral correlates of error monitoring and response inhibition. Neural measures of executive functions might be more sensitive for interindividual differences (Krämer et al., 2007) and can moreover reveal the underlying "mechanism" by which executive functions are linked to the control of aggression. Specifically, we and others recently provided evidence for differences in frontal activity related to interindividual variability in reactive aggression (Lotze et al., 2007; Krämer et al., 2008; Krämer et al., 2009). Participants who scored high in trait aggressiveness but largely refrained from retaliation in response to provocation showed increased frontal activity when being provoked (Krämer et al., 2008; Krämer et al., 2009). This was suggested to reflect inhibitory control functions contributing to the regulation of reactive aggression. The respective correlates of frontal activity (increased frontolateral negativity, higher frontal theta response) showed similar spatiotemporal dynamics compared with neural correlates of "cold" executive functions. It is thus tempting to assume that participants who show increased frontal activity when being provoked in a social interaction also demonstrate higher frontal activity in cognitively demanding situations. With the present study, we wanted to test this assumption. To this end, we assessed differences in neural correlates of executive functions (performance monitoring, response inhibition) between groups of participants showing low or high experimentally induced aggression in the TAP. As reported previously (Krämer et al., 2009), these participants differed also in their frontal activity in response to the provocation.

To sum up, we investigated the link between executive functions and aggressive behavior by (i) comparing high and low trait aggressive participants' performance in an extensive executive function test battery and by (ii) assessing neural and behavioral correlates of executive functions in participants characterized by their behavior in an aggressive social interaction. As measures of executive functions, we focused on the error-related negativity (ERN) and the inhibition-related N2 as these are well-established correlates of performance monitoring (Falkenstein et al., 1990; Gehring et al., 1993) and response inhibition (Ramautar et al., 2004; Schmajuk et al., 2006) and sensitive for interindividual differences (Pliszka et al., 2000; Krämer et al., 2007). The ERN is a frontocentral negativity, maximal around 60 ms after an erroneous response, which is believed to emanate from dorsal ACC. The stop-N2 is maximal over central (Ramautar et al., 2004) or right frontal areas (Pliszka et al., 2000) and assumed to be generated in the ACC or right prefrontal cortex. Based on the existing evidence, we expect less aggressive people to show better performance in executive functions tasks and an increased ERN and stop-N2.

BEHAVIORAL STUDY

MATERIALS AND METHODS

All procedures were cleared by the ethical review boards of the University of Magdeburg and the University of Barcelona. The study was conducted in compliance with the *Declaration of Helsinki*.

Participants

The data were assessed from 655 students from the University of Barcelona (491 women; age range from 18 to 39, mean = 21.7 ± 3.2). The participants underwent a neuropsychological test battery and filled out a range of personality and health questionnaires.

Questionnaire

As a measure of aggressiveness, we used the Aggression Questionnaire (Buss and Perry, 1992) in its Spanish version (Andreu Rodríguez et al., 2002). The Aggression Questionnaire, which has been used extensively in many studies of aggressive behavior, is a valid self-report technique to assess the different subtraits of aggressiveness: physical and verbal aggression, which reflect the instrumental and motor components of aggression; anger, which is related to the affective component of aggression; and finally hostility, assessing feelings of ill will and injustice and thus representing the cognitive component of aggressiveness. A total score (AQ) as well as scores for the subscales Physical Aggression (AQ-PA), Verbal Aggression (AQ-VA), Anger (AQ-A), and Hostility (AQ-H) can be derived. The questionnaire has been shown to have good reliability as well as good convergent and discriminative validity (Buss and Perry, 1992; Andreu Rodríguez et al., 2002; Tremblay and Ewart, 2005; Vigil-Colet et al., 2005). To control for possible effects of impulsivity, we also administered the I7 Impulsivity Questionnaire (Eysenck et al., 1985).

Behavioral test battery

Flanker task. We applied a modified variant of the Eriksen flanker task (Eriksen and Eriksen, 1974), that required the participants to respond to the central arrow in an array of five arrows (with the right hand following a right-directed arrow and vice versa). The four surrounding arrows were either compatible or incompatible to the central arrow, favoring performance errors. We presented 38.5% of compatible and 38.5% of incompatible trials. In 11.5% of trials we included no-go-trials as in the stop-signal paradigm (Band et al., 2003). In these trials, the central green arrow changed to red after a variable delay, indicating participants to inhibit the response in these trials. Two different stop-signal delays were applied (with equal probability), one yielding a low inhibitory rate (180 ms), and one yielding a high inhibitory rate (70 ms). In the remaining 11.5% of trials we included change trials, in which the central arrow changed its direction after 50 ms, indicating the subject to react with the other hand. Each stimulus array was presented in the middle of the screen. Stimulus duration was 300 ms and the stimulus onset asynchrony (SOA) was between 900 and 1100 ms (rectangular distribution). Participants received 10 training trials to get acquainted to the task. They were encouraged to correct their errors in the go-trials as fast as possible. The experiment was divided into three blocks, each comprising 208 trials, resulting in a total of 624 trials. We derived several parameters from this task, reflecting inhibitory functions, stimulus–response interference, and performance monitoring: incongruency effect on reaction time (reaction time of correct responses in incompatible trials – compatible trials) and percentage of errors (errors in incompatible trials – compatible trials), percentage of inhibited trials, stop-signal reaction time (SSRT; see Band et al., 2003) for the computation; we used the easy stop-trials for computation of the SSRT) and percentage of correctly changed trials.

Stroop. We used a computerized version of the classical Stroop task (Stroop, 1935), presenting the words "blue," "green," and "red" in either the congruent or incongruent color, requiring the participants to press the button that was associated with the color of the ink. One hundred twenty-one trials were presented (50% incongruent), with 10 training trials in the beginning. Stimulus duration was 500 ms and the SOA varied randomly between 1500 and 2500 ms. We computed the incongruency effect on the reaction time (reaction time of correct responses in incongruent trials – congruent trials) and the percentage of errors (errors in incongruent trials – congruent trials).

N-back working memory task. Letters were presented one by one centered on a video monitor, requiring the subject to press a button, whenever the letter was identical to the one shown two letters before. Letters were depicted in upper- and lowercase with the case to be ignored for the task, encouraging the participant to verbally encode and rehearse the letter. One-third of the trials were target trials. Stimulus duration was 500 ms with an SOA of 2500 ms. We computed the percentage of correct trials and the reaction times of correct responses as measures of working memory performance.

Task switching. We used the task switching paradigm from a German test battery for attention assessment (Zimmermann and Fimm, 1994). In every trial one letter and one digit were presented. The participants were asked to pay attention to the letter or the digit in alternating trials and to press a button on the respective side (right or left). Each trial thus required a switch of the attention focus (letter or digit). However, in some trials a switch of the response hand was needed, while in others no switch was needed. Switching of the response hand has been shown to be easier, reflected in a shorter reaction time (Zimmermann and Fimm, 1994). Note that the switch costs are thus reversed in the current task, as they do not refer to the switch of the attention focus, but to the switch of the response hand. One hundred trials were presented, each trial ended with the subject's response. We computed the mean reaction time of correct switch responses and the difference between reaction times in switching and non-switching trials.

WAIS matrices. We used the matrices subtest of the WAIS III (Wechsler, 1997) to assess participants' non-verbal reasoning abilities. It consists of 29 designs, requiring the participant to fill in a missing design from a number of choices. A computerized version was used with a time restriction of 25 s for each design. The total number of solved problems was scored.

Verbal fluency. We used a semantic verbal fluency task. Participants were required to write down as many animals as possible, within 2 min. Dependent variable was the number of generated words.

Trail-making-test. This test (Reitan, 1958) has two parts. Trail-making-test (TMT) A requires the participant to connect a sequence of numbers (1–25) distributed over a whole page as fast as possible without lifting the pen from the paper. This part measures visuomotor tracking. In TMT B, the subject has to alternate between numbers and letters (1–A–2–B–3…), which necessitates more attention and conceptual tracking. The time to complete parts

A and B was measured and the difference between A and B was taken as test parameter, which removes the simple differences in motor functions.

Wisconsin Card Sorting Test. The Nelson version of the WCST (Nelson, 1976) comprises a series of 48 cards, which show printed colored geometrical figures, that differ in one or more dimensions: shape, color, or number. Participants were asked to discover the rule and sort each card by matching it to one of the four stimulus cards, with the rule changing after six consecutive correct sorts. The task was presented as a computerized version. We used the number of perseverative errors (two successive card assignments to an incorrect dimension) as parameter from this test.

Tower of London. We used the TOL – Drexel version (Culbertson and Zillmer, 1998), comprising 10 test items with 4–7 required movements each. Participants are instructed to replicate different patterns of cylinders on three pegs in as few moves as possible. Two rules have to be adhered to: only one cylinder at a time can be moved and more cylinders cannot be placed on a peg than the peg can accommodate. For each item 120 s are given to solve the task. Measured parameters are the time to start the first move (latency), and the number of moves exceeding the necessary number of moves.

Data analysis

We examined differences in executive functions between high and low trait aggressive participants. To this end, we compared participants within the lowest and the highest quartile of the aggression questionnaire (general score) in their performance in the executive functions test battery with univariate analyses of variance (ANOVAs). To examine possible differential effects of trait aggressiveness on executive functions (Hoaken and Pihl, 2000), we added sex as second factor to test for sex by AQ interactions.

RESULTS

All executive functions parameters of interest were available from 91 participants in the lowest aggression quartile and from 90 participants in the highest aggression quartile. Participants in the low trait aggressive group (70 women) had a maximum aggression score of 53 (mean = 46.1 ± 5.2), while high trait aggressive participants (66 women) had a minimum score of 74 (mean = 83.9 ± 8.0).

The univariate ANOVAs for the different executive functions parameters yielded a significant difference between high and low trait aggressive participants in the latency score of the TOL only ($F_{1,179} = 11.41$, $p_{\text{uncorrected}} = 0.001$; $p_{\text{Bonferroni}} < 0.05$). This was due to a shorter latency in high than in low trait aggressive participants (see **Table 1**; **Figure 1**). A trend toward group differences was seen in the Stroop incompatibility effect on the error rate, with a higher incompatibility induced increase of the error rate in low trait aggressive people, but did not survive the correction for multiple comparisons ($F_{1,179} = 4.18$, $p_{\text{uncorrected}} = 0.045$, $p_{\text{Bonferroni}} > 0.1$). No other group differences were observed (**Table 1**). Adding sex as a second factor did not change the results substantially and, importantly, no interactions of sex by AQ were detected (all $p > 0.1$). We additionally performed regression analysis testing for effects of the Aggression Questionnaire score on the executive functions measures in the complete sample. This approach yielded similar results,

Table 1 | Results of executive functions test battery (Study 1).

		LT	HT
Flanker	RT (difference)	34 (15)	36 (13)
	% Errors (difference)	5.4 (4.5)	5.3 (4.8)
	% Inhibition	54.9 (18.0)	52.5 (17.5)
	SSRT	293 (24)	295 (30)
	% Correct change	74.9 (15.8)	74.1 (19.1)
Stroop	RT (difference)	64 (56)	61 (54)
	% Errors (difference)	3.6 (4.3)	2.3 (4.4)
n-back	RT	578 (121)	578 (100)
	% Errors	5.4 (4.5)	5.3 (4.8)
TS	RT (correct)	1085 (242)	1157 (260)
	RT (difference)	170 (212)	151 (157)
Fluency	*n* (Animals)	29.2 (7.8)	29.7 (6.5)
WCST	*n* (Perseverations)	2.1 (2.2)	2.4 (2.5)
ToL	**Latency (s)**	**60 (43)**	**42 (21)**
	Exceed. moves	34.2 (17)	35.6 (15)
WAIS	*n* (Correct)	18.2 (2.6)	18 (2.5)

Behavioral results in the executive functions test battery for low trait (LT) and high trait (HT) aggressive participants. Significant differences are highlighted in bold. RT, reaction time (ms); TS, task switching; WCST, Wisconsin Card Sorting Task; ToL, Tower of London; WAIS, Wechsler Adult Intelligence Scale – Matrices; SSRT, stop-signal reaction time. Difference in Flanker and Stroop refers to incompatible – compatible and difference in task switching refers to non-switching – switching. Values in brackets are SD.

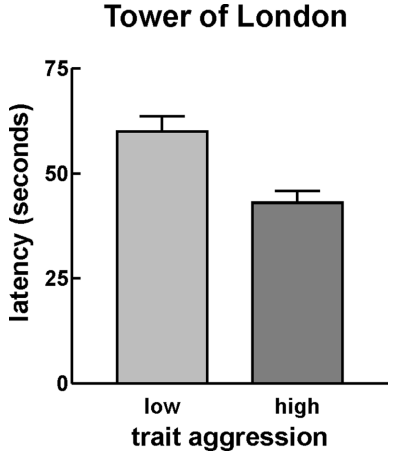


FIGURE 1 | Average latency (seconds) in the Tower of London task separately for low (left) and high (right) trait aggressive participants. High trait aggressive participants showed a reduced latency of the first move in the ToL paradigm. Error bars reflect SE.

since only the effect on the TOL latency measure was statistically significant after correction for multiple comparisons ($\beta = -0.120$, $p_{\text{uncorrected}} = 0.003$; $p_{\text{Bonferroni}} < 0.05$). As performance in the TOL has been related to inhibitory functions, the reduced latency in high trait aggressive participants might reflect higher impulsivity. To test this, we finally performed a regression analysis with both the I7 Impulsivity scale and the Aggression Questionnaire as predictors of the TOL latency measure. The I7 Impulsivity score significantly predicted the TOL performance ($\beta = -0.109$, $p = 0.015$), but the

AQ did not yield significance anymore ($\beta = -0.072$, $p = 0.106$). This suggests that the reduced latency in high trait participants reflects a higher tendency for impulsive behavior.

EEG-STUDY

MATERIALS AND METHODS

Participants

Note that the behavioral and EEG results of the aggression paradigm in this study have been reported previously (Krämer et al., 2009). Here, we only summarize the relevant results of the aggression paradigm and focus on the results of the Flanker experiment.

Participants were selected from two larger groups of students from the University of Magdeburg on the basis of their scores on an aggression questionnaire (see below). The first sample comprised 231 economy students (129 women; mean age 22.6, SD 1.9) and the second sample consisted of 520 students from engineering, economics, medicine, and humanities (286 women; mean age 22.9 years). From these groups, we selected participants with high values in the aggressiveness score (see below for further explanation).

Thirty-two students (17 women, mean age = 24.7) participated in the EEG-study after giving informed consent. All were free of neurological or psychiatric disorders and had normal or corrected-to-normal vision. Four participants were excluded from further analyses because they were deemed not to have been completely deceived in the aggression paradigm ($n = 2$) or because of excessive eye movement artifacts. Thus, 28 participants (15 women; mean age = 24.7 years) were included in the analyses. One participant had a very low inhibition rate of 4% in the stop-task (see below) and was thus excluded from analysis of the stop-trials. Participants received money as compensation.

Questionnaire

Participants were selected based on their trait aggressiveness assessed with a German inventory for the assessment of factors of aggression (FAF, Fragebogen zur Erfassung von Aggressivitätsfaktoren; Hampel and Selg, 1975). With this questionnaire, five subscales (spontaneous aggression, reactive aggression, impulsiveness, autoaggression, aggression inhibition) and a control scale (openness) can be obtained. Spontaneous aggression (19 items) refers to unrestrained verbal or physical aggression. A typical item is "I sometimes like to tantalize others." Items of the reactive aggression scale (13 items) ask for aggressive reactions to some kind of provocation or unfairness, such as "If someone provokes me, I want to punish him badly." Items of the impulsivity scale (13 items) deal with the affective component of aggression, as "I flare up quickly, but get over it quickly." The sum of the scales "spontaneous aggression," "reactive aggression," and "impulsiveness" gives a reliable measure for outwardly directed aggression (internal consistency Cronbach's alpha = 0.85) and was thus used for selection of high trait aggressive participants. The sum score has been shown to be significantly different between both adolescent and adult violent criminals on the one hand and non-violent controls on the other hand (Hampel and Selg, 1975), providing evidence for its external validity.

Task and procedure

All participants performed first in the aggression paradigm and afterward in the Eriksen Flanker task. Aggression was elicited and assessed using a modified version of the TAP (Taylor, 1967). Participants were instructed that they were playing successive competitive reaction time trials against one of two opponents in alternating trials (Krämer et al., 2009). The opponents (one man, one woman), confederates of the experimenters, met the participant prior to the experiment to jointly listen to the instructions. They were told that whoever lost would be punished by the opponent with a mildly painful electric shock. The severity of the punishment, that is the intensity of the shock, had to be selected for each trial on a range from 1 to 8. In fact, selections of the putative opponents and outcome of the trials (50% winning and losing trials for each opponent) were under control of the experimenter. The experiment comprised seven blocks of 40 trials each, yielding a total of 280 trials. Participants were told that the opponents would play in alternating trials and rest during the others. At the end of the experiment participants were completely debriefed about the deception and the experiment's motivation.

We applied a modified variant of the Eriksen Flanker task (Eriksen and Eriksen, 1974), which was highly similar to the paradigm used for the behavioral study (see above and Krämer et al., 2007). In contrast to the behavioral study, the delay of the stop-signal was adapted to participants' behavior by means of a staircase-tracking algorithm (Band and van Boxtel, 1999) as follows. The stop-signal delay was set to 140 ms initially. After a successful inhibition, the stop-signal delay was increased by 10 ms (making the inhibition harder). After a failure in inhibition, the stop-signal delay was reduced by 10 ms (making the inhibition easier). This procedure was applied to yield an inhibition rate of about 50%. We computed the SSRT (Band et al., 2003) by subtracting the participant's mean stop-signal delay from the median reaction time of correct go responses. No change trials were included, yielding thus 33% of stop-trials. Participants received 20 training trials to get acquainted to the task. They were encouraged to correct their errors in the go-trials as fast as possible. The experiment was divided in eight blocks, each comprising 240 trials, resulting in a total of 1920 trials. Duration of the experiment including electrode preparation was 2.5 h.

EEG recordings

The electroencephalogram (EEG) was recorded from 27 tin electrodes mounted in an elastic cap (Easycap™; positions: Fp1/2, F3/4, C3/4, P3/4, O1/2, F7/8, T7/8, CP1/2, PO3/4, FC1/2, FC5/6, CP5/6, Fz, Cz, Pz) with reference electrodes placed on the right and left mastoid. During recording, all scalp electrodes were referenced against an average reference and offline re-referenced against the algebraic mean of the activity at the two mastoid electrodes. Electrode impedances were kept below 5 kΩ. To monitor horizontal eye movements, electrodes were placed on the outer canthus of the right and left eye. Vertical eye movements and blinks were monitored by electrodes placed below and above the right eye. EEG and EOG were recorded continuously with a bandpass of 0.01–70 Hz and digitized with a sampling rate of 250 Hz.

Data analysis

Stimulus- and response-locked averages were obtained for errors and stop-trials. For stimulus-locked ERPs, the 100-ms preceding the stimulus were considered as baseline, for response-locked ERPs, baseline was defined as the 50-ms before the button press

(Rodriguez-Fornells et al., 2002; Krämer et al., 2007). All artifact-free error trials were included irrespective of a following corrective response. To account for the overlap between go- and stop-ERPs in the stop-signal-locked data, we computed difference waves following previously published methods (Ramautar et al., 2004; Ramautar et al., 2006; Krämer et al., in press). Specifically, we shifted go ERPs from the respective reaction time distribution (fast reactions for errors and slow reactions for successful inhibitions) across the range of individuals' stop-/change-signal delays weighted by the actual occurrence of that delay and averaged them. These "virtual" go ERPs were then subtracted from successful and failed inhibitions.

For statistical analyses, mean amplitudes (unless otherwise stated) were subjected to a repeated measures ANOVA with the between-subject factors of experimentally induced aggression (factor Group, HE vs. LE) and the within-subject factors condition and electrode position, dependent on the particular component (as stated below). For all statistical effects involving more than one degree of freedom in the numerator, the Huynh–Feldt correction was applied to correct for possible violations of the sphericity assumption (Huynh and Feldt, 1976). The corrected probabilities are reported.

RESULTS

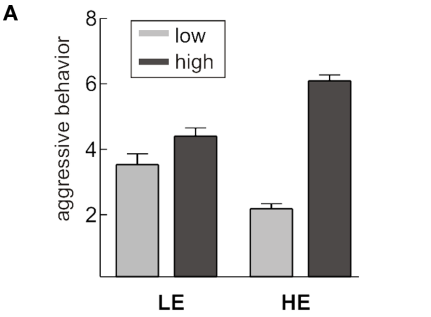
Aggression paradigm

Participants selected higher punishments for the highly provoking opponent (mean 5.25 ± 1.16) compared to the non-provocative opponent (2.86 ± 1.16; $t_{27} = 6.90$, $p < 0.001$). The average time to make the selection under high provocation was 1060 ms (±467), which did not differ from the time taken to select the punishment under low provocation (1059 ± 446 ms). Participants' mean reaction time to the target was 202 ms (±39).

For the present study, we wanted to examine differences in executive functions between participants responding aggressively to the provocation (high experimentally induced aggressiveness, HE) and those that did not (LE). We accordingly divided the sample into two groups based on their average selection under high provocation (median split; **Figure 2A**). Although the provocation effect was evident in both groups, it was clearly higher in the HE group ($t_{13} = -20.40$, $p < 0.001$) than in the LE group ($t_{13} = -2.7$, $p = 0.019$). The two groups did not differ with respect to their mean FAF-score ($t_{26} = 1.32$, $p = 0.20$) or regarding gender distribution ($\chi^2 = 0.337$, $p = 0.561$).

Flanker paradigm

Participants were faster (385 ± 36 ms) and more accurate (error rate 6.2 ± 5.6 %) in compatible than in incompatible (409 ± 39 ms; 12.9 ± 8.1 %) trials (reaction times: $F_{1,26} = 105.9$, $p < 0.001$; accuracy: $F_{1,26} = 70.8$, $p < 0.001$). The staircase-tracking algorithm was successful, such that the average inhibition rate was 49.1% (±4.5). The average SSRT was estimated to be 271 ms (±41). As typically observed in the Eriksen Flanker task, participants slowed down after making errors, both after errors in go-trials (post-error-slowing: 26 ± 31 ms) and after stop-errors (14 ± 31 ms). **Table 2** shows the behavioral data separately for the two groups. We did not detect significant group differences in any of the behavioral measures.



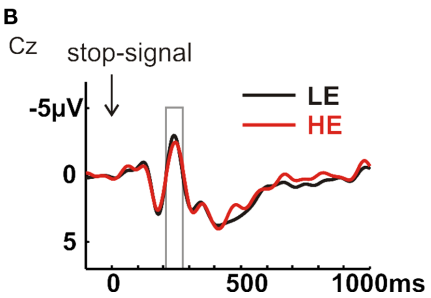


FIGURE 2 | (A) Depicted are the mean selections under low (light gray) and high (dark gray) provocation separately for the two groups LE (low experimentally induced aggression) and HE (high experimentally induced aggression; right panel) in the Taylor Aggression Paradigm. Error bars reflect SE. **(B)** Event-related potentials (ERPs), stimulus-locked to the stop-stimulus in successfully inhibited trials, separately for the groups LE (black line) and HE (red line). The time-window of interest for the stop-N2 (200–280 ms) is indicated with a gray box.

Table 2 |Behavioral results of modified Flanker task (Study 2).

	LE	HE
RT (compatible)	379 (33)	390 (39)
RT (incompatible)	403 (40)	415 (39)
RT difference	24 (14)	24 (11)
% Go-errors	10.6 (6.9)	9.4 (7.1)
Post-error-slowing	30 (31)	23 (32)
Post-non-inhibition-slowing	19 (30)	9 (33)
Stop-signal-delay	124 (40)	118 (45)
SSRT	269 (43)	274 (41)

Behavioral results in the modified Eriksen flanker task for low (LE) and high experimentally induced aggression (HE) participants. SSRT, stop-signal reaction time; difference refers to incompatible – compatible. Values in brackets refer to the SD.

As neural correlates of performance monitoring and response inhibition, we assessed the ERN and the stop-signal related N2, respectively (Krämer et al., 2007). Note that one outlier (in the LE group) was excluded from the analysis of the stop-trials because of an inhibition rate below 5% (see Materials and Method). In stop-trials, both inhibited and non-inhibited trials elicited an N2 distributed over central and parietal areas, as had been reported previously (Pliszka et al., 2000; Schmajuk et al., 2006; Krämer et al., 2007). The N2 was maximal around 230 ms after the stop-signal.

To examine group differences in inhibitory control, we compared average amplitude values in the N2 time-window (200–280 ms) at midline electrodes with a repeated measures ANOVA with the within-subject factors Inhibition (inhibited vs. non-inhibited) and Electrode (Fz, Cz, Pz) and the between-subject factor Group (LE vs. HE). Errors showed a larger negativity over central electrodes (Inhibition × Electrode: $F_{2,50} = 4.80$, $p < 0.019$). No main effects or interactions with the factor Group were significant (all $F < 1$; **Figure 2B**), suggesting comparable inhibitory functions in participants showing low or high experimentally induced aggression. This was further supported when testing for correlations between aggressive behavior and the stop-N2 amplitude at Cz across the whole sample. The stop-N2 amplitude for inhibited trials did not correlate with aggressive behavior ($p > 0.1$).

A clear ERN was detectable in the group averages, which peaked around 60 ms and had the typical frontocentral maximum (**Figure 3**). Based on previous literature (Rodriguez-Fornells et al., 2002; Krämer et al., 2007), we submitted average amplitude values of the time-window 30–80 ms to a repeated measures ANOVA with the within-subject factors Accuracy (error vs. correct) and Electrode (Fz, Cz, Pz) and the between-subject factor Group (LE vs. HE). The ANOVA confirmed the enlarged negativity in error compared to correct trials at frontocentral electrodes (Accuracy: $F_{1,26} = 39.60$, $p < 0.001$; Accuracy × Electrode: $F_{2,52} = 39.70$, $p < 0.001$). In contrast to our hypothesis, the highly aggressive group showed a tendency for a larger ERN, but this trend did not even yield marginal significance (Accuracy × Group: $p > 0.1$). The correlation between ERN amplitude at Fz and aggressive behavior across the whole sample was not significant either ($p > 0.1$).

DISCUSSION

In the present study, we examined the relationship between executive functions and aggressive behavior using a combined behavioral and electrophysiological approach. We assessed performance differences on an extensive test battery of executive functions between high and low trait aggressive participants and found a prolonged latency of the first move in the TOL in low trait aggressive people. Additionally, a separate group of participants differing in reactive aggressive behavior as measured by the TAP performed a modified Eriksen Flanker task aimed to tap into performance monitoring and response inhibition aspects of executive functions. The groups did not differ, however, in behavioral or neural correlates of cognitive control. The results question a strong link between executive functions and control of aggressive behavior.

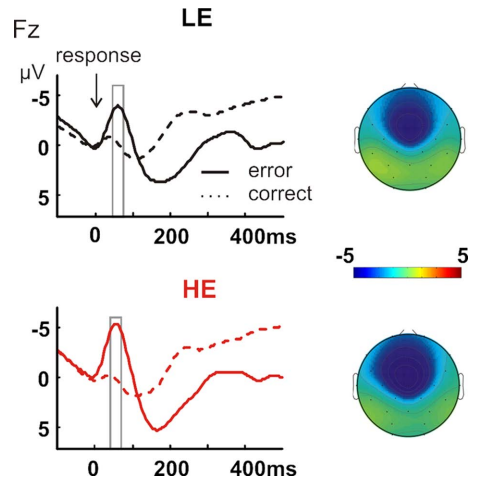


FIGURE 3 | Response-locked ERPs for error (solid line) and correct (dashed line) trials, separately for the LE (upper row, black lines) and HE group (lower row, red lines). The time-window of interest for the error-related negativity (30–80 ms) is indicated with a gray box and the respective topographical maps of the average amplitude in the time-window of interest is shown separately for the two groups (upper map: LE; lower map: HE).

BEHAVIORAL STUDY

High and low trait aggressive participants differed regarding their executive functions only in the latency parameter of the TOL, with a shorter latency in highly aggressive people. No other differences were observed, arguing against a general impairment in executive functions associated with high trait aggressiveness (Giancola, 2004). Moreover, regression analysis on the whole sample showed a significant relationship between trait aggressiveness and the TOL latency measure only. Regarding the TOL, different parameters are usually measured to quantify participants' performance in this task, supposedly reflecting the different cognitive processes involved (i.e., working memory, planning, and inhibition). The number of exceeding movements has been shown to correlate with fluid intelligence (matrix reasoning; Zook et al., 2004), but the case is less clear for the latency parameter. However, as both the TOL and the similar Tower of Hanoi depend on inhibitory functions (Goel and Grafman, 1995; Welsh et al., 1999; Miyake et al., 2000), this factor might be in fact best captured by the time to initiate the first movement, (i.e., the time taken to consider the best solution). Shorter latencies in highly aggressive participants might therefore reflect their reduced inhibition abilities and tendency for impulsive, imprudent behavior. This interpretation is further strengthened by the observation of a significant correlation between the I7 Impulsivity score and the TOL latency measure, which could largely account for the effect of trait aggressiveness on the TOL performance in a regression analysis.

We did not find any differences in other parameters of inhibitory functions, however (e.g., the Stroop incompatibility effect or the percentage of inhibited trials in the Flanker task). Two reasons could have accounted for this: first, the inhibitory functions in the Stroop or Flanker task have a stronger motor component and thereby differ from the inhibition required for the TOL. However, this argument is challenged by reports of significant correlations between Stroop performance and TOL or Tower of Hanoi performance (Welsh et al., 1999; Miyake et al., 2000). Alternatively, the TOL latency score might be a more sensitive parameter, while ceiling effects accounted for indistinguishable performance in the Stroop or Flanker. It should be noted, that the same argument applies for the other parameters. As all participants were young and healthy, it might be that the applied tasks were not sensitive enough to detect subtle interindividual differences in executive functions. This can most likely be ruled out, since the different parameters presented a considerable interindividual variance and previous studies have shown subtle genotype effects with these tasks (Egan et al., 2001; Goldberg et al., 2003).

This result extends previous findings regarding the relationship of inhibitory abilities and aggressive behavior. Observations in both psychiatric and neurological patients hint at probable prefrontal alterations causing an impaired response control, which is likely underlying their higher proneness to violence (Raine, 1993; Kiehl et al., 2000; Munro et al., 2007a). Kiehl et al. (2000) for instance could demonstrate diminished neurophysiological correlates of response inhibition in psychopaths, supposedly associated with their inability to refrain from aggressive outbursts. However, these patients may possibly have extensive structural and functional abnormalities, which limits the conclusions that can be drawn about specific causes of their behavioral deficits. Laboratory studies in healthy samples could provide additional evidence for the relationship between aggressive tendencies and inhibitory abilities, as highly aggressive participants also show more impulsive behavior (Cleare and Bond, 1995; LeMarquand et al., 1998). Also Sellbom and Verona (2007) reported significant correlations between a self-report measure of psychopathic traits and a response inhibition composite score in a student sample. Our behavioral data support the notion of a role of inhibitory functions in the regulation of aggressive behavior and question a general, non-specific influence of executive functions on aggression (Giancola, 2004).

EEG-STUDY ON EXECUTIVE FUNCTIONS

One might argue that trait questionnaires are only part of the story and that aggressive behavior elicited in response to provocation in the lab might be a better measure to distinguish groups of low or high aggressiveness. In fact, EEG findings related to laboratory-induced reactive aggression suggested enhanced prefrontal activity in those with high trait aggressiveness, who were able to refrain from retaliation after provocation (Krämer et al., 2008, 2009). This suggests that executive functions might particularly impact violent behavior in more challenging situations that involve interpersonal provocation, for example. Based on this reasoning, we compared participants who differed in their aggressive response to provocation with respect to behavioral and neural measures of executive functions. However, although the two groups of high and low experimentally induced aggression clearly differed in their neural response to provocation in the aggression paradigm (Krämer et al., 2009), we did not observe any evidence for diminished executive functions in the highly aggressive group. In fact, this group showed a tendency for a higher ERN, and thus, better performance monitoring (i.e., better executive functions), compared to the less aggressive group. Additionally, correlations between aggressive behavior and the ERN and stop-N2 amplitude did not yield significance.

Previous results regarding the ERN or stop-N2 in psychiatric samples are not fully conclusive, with a few studies showing diminished error monitoring or inhibitory control in psychopaths or persons with antisocial personality disorder and others finding no difference (Kiehl et al., 2000; Munro et al., 2007a,b; Brazil et al., 2009, 2011; von Borries et al., 2010). Evidence points specifically to altered error monitoring in an affective context (Munro et al., 2007a) or in learning tasks (von Borries et al., 2010). The present results are consistent with a view that there is no direct link between aggressive behavior and a general impairment in executive control functions, such as performance monitoring or response inhibition. In fact, the results showed a tendency for a higher ERN in highly aggressive participants. Since personality variables, such as anxiety, are known to influence the ERN (Hajcak et al., 2003; Aarts and Pourtois, 2010), one might speculate that differences in emotional responsivity were underlying the increased reactive aggression and the tendency for a higher ERN in this group.

The small sample size of 14 participants per group might have precluded significant group differences. This is rather unlikely, however, as the groups significantly differed in their neural response to provocation in the aggression paradigm (Krämer et al., 2009). In addition, we used a median split to analyze our data, which leads to rather similar groups. Comparing extreme groups may have lead to significant differences in neural correlates of cognitive control, as well.

CONCLUSION

There has been an increasing interest in the relationship between control of emotions vs. cognition in recent years, raising the question whether interindividual differences in executive control functions impact behavior in social interactions. The present study suggests that cognitive control as assessed with neural and behavioral measures of executive functions does not directly relate to aggressive behavior in affective social interactions. This brings into question a strong link between aggression and executive functions in healthy high-functioning individuals.

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The feeling of action tendencies: on the emotional regulation of goal-directed behavior

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In this article, we review the nature of the functional and causal relationship between neurophysiologically/psychologically generated states of emotional *feeling* and *action tendencies* and extrapolate a novel perspective. Emotion theory, over the past century and beyond, has tended to regard feeling and action tendency as *independent* phenomena: attempts to outline the functional and causal relationship that exists between them have been framed therein. Classically, such relationships have been viewed as unidirectional, but an argument for bidirectionality rooted in a dynamic systems perspective has gained strength in recent years whereby the feeling–action tendency relationship is viewed as a composite whole. On the basis of our review of somatic–visceral theories of feelings, we argue that feelings are grounded upon neural-dynamic representations (elevated and stable activation patterns) of action tendency. Such representations amount to predictions updated by cognitive and bodily feedback. Specifically, we view emotional feelings as *minimalist* predictions of the action tendency (what the agent is physiologically and cognitively primed to do) in a given situation. The essence of this point is captured by our exposition of action tendency prediction–feedback loops which we consider, above all, in the context of emotion regulation, and in particular, of emotional regulation of goal-directed behavior. The perspective outlined may be of use to emotion theorists, computational modelers, and roboticists.

Keywords: feeling, action tendency, prediction, feedback, neural-dynamic representations, reinforcement, homeostasis, goal-directed behavior

HISTORICAL BACKGROUND – ON FEELINGS AND ACTIONS

> "[c]ommon sense says, we lose our fortune, are sorry and weep; we meet a bear, are frightened and run; we are insulted by a rival, are angry and strike . . . this order of sequence is incorrect . . . the more rational statement is that we feel sorry *because* we cry, angry *because* we strike, afraid *because* we tremble,"
>
> (James, 1890, p. 449, James' italics).

The above view of the pioneering emotions researcher William James provided a landmark in the understanding of the role of bodily *feedback* in feelings and decision making in the context of overt behavior. It has been considered counter-intuitive to the psychological interpretation of emotional experience[1]. The Jamesian view has since been conceived as a pioneering *somatic theory* of emotion, contrasting with the more abundant *cognitive theories* of contemporary emotion research, since bodily changes were considered compositional to, rather than independent of (and secondary to), the emotion. The Jamesian perspective can also be considered a "(somatic) feeling theory of emotion" (Prinz, 2004, p. 5) where feelings are comprised of bodily changes that follow "perception of the exciting fact." The purported *causal* role of action-instigated bodily feedback in feeling together with the concept of "feeling as the emotion" had at least two important implications for the *functional* role of emotions: firstly, if the emotion follows action, what useful role, if any, does it have in higher cognitive and behavioral activity, e.g., decision making? Secondly, if there is no role for cognition in triggering the bodily changes that comprise the emotional state, what is the trigger mechanism?

Following the behaviorist and cognitivist revolution of the early to mid twentieth century, interest in affective and emotional processes and their role in conscious experience and behavior waned (cf. LeDoux, 1996) as they were not considered functional to classically conceived *rational* cognitive processes, e.g., planning, decision making, attention, learning. Nevertheless, James had seeded a prospective debate that would be embarked on in the second half of the twentieth century concerning the causal and functional relationship existing between emotional feelings and their associated actions. From the 1960s, interest in emotion *per se* was galvanized, and a popular conception viewed them as part and parcel of functional behavioral activity in the context of cognitive appraisals (Arnold, 1960). Early "appraisal theory," however, still had a very much cognitivist flavor whereby emotions were considered *hot* action responses or tendencies triggered by, and independent from (secondary to), *cold* cognitive perceptual judgments of the significance of stimuli to the well-being of the organism (effective triggers).

In recent years, appraisal theory has evolved to incorporate a bidirectional perspective on the relationship between (cognitive)

[1]Though for a review on the cultural-constructivist nature of the "intuitive" alternative perspective see Laird (2007).

appraisal and emotion rooted in the dynamics and neural representation of action tendency. Such views include: (1) *embodied appraisals* (cf. Prinz, 2004, 2005), and (2) *process models* of appraisal (cf. Scherer, 1984, 2000, 2009; Ellsworth and Scherer, 2003). In the case of perspective (1), neural patterns reflective of changes in the internal milieu and skeletomusculature serve as embodied appraisals (perceptions and feelings). These appraisals track the relevance of an external stimulus to the well-being of the organism thereby establishing organism–environment *relations* – "core relational themes" (Lazarus, 1991) – and are informative insofar as they register bodily changes that constitute *action tendencies* (see Lowe et al., 2007 for discussion of this perspective). In the case of (2), affective action tendencies contribute to ongoing and context-elaborated appraisals. These appraisals manifest according to a dynamic relationship (reciprocal modulation) among the constitutive components that include "monitoring/feeling state" and "motivation/action tendencies." Such process models are, nevertheless, purely cognitive regarding the early stages of stimulus appraisal processing. Recently, Lewis (2005) also posited a dynamic systems (DS) approach whereby feeling and action tendencies are intricately interwoven[2], producing synchronized and stable global-orienting states (emotion–appraisal amalgams) the "substrate" on which higher emotional–cognitive activity (e.g., attentional orientation, learning) operates. In this view, no causal precedence for either purely cognitive or emotion processes exists.

Mirroring the development of embodied/somatic and DS appraisal theoretic accounts of emotional–cognitive activity, much research into the neurophysiology underlying affective phenomena has emphasized the complex and integrated role of feelings and action tendencies. In the spirit of James, *somatic* emotion theorists have identified feelings as being developmentally grounded in bodily changes that prefigure behavior. Some of these theorists have emphasized the significance of skeletomuscular feedback to the generation of feeling. In line with James' central argument, Bem (1972), Ekman (1972, 2003), Laird (2007) have argued for the *sufficiency* to feeling generation of feedback from facial, vocal, and postural expression. In the view of Laird, error-based feedback can provide a critical "cybernetics control process" function for "shaping" behavior. Damasio (1994, 1999) and Bechara (2004), however, have been the chief architects of re-establishing the Jamesian-somatic (or, perhaps more accurately, somatic–visceral) feeling perspective in psychology and neuroscience. Their perspective converges with that of the above-mentioned theorists. Nevertheless, (somatic–visceral) bodily feedback is not considered *necessary* for feeling generation. In their view, consistent with the *feedback–feeling sufficiency* argument, bodily changes to the organism (above all internal milieu and skeletomusculature), *in some circumstances,* precede registrations in the brain that correlate with emotional experience (feelings) – Damasio termed this stimulus processing route the "body loop" (Damasio, 1994). This loop is enacted in circumstances of uncertainty or prior to learning stimulus significance. Critically, however, Damasio posited that emotional feeling states can occur *prior to* such bodily changes *following* learning. In this case, brain areas implicated in providing the neural substrate for feelings are activated in parallel with (or in the absence of) the slower dynamics of bodily change activated and fed back (to the central nervous system) through the conduit of the peripheral nervous system. Damasio termed this processing route the "as-if body loop" (Damasio, 1994, 1999, 2003, 2010; also see Bechara, 2004). In the Damasio/Bechara perspective, therefore, emotional feelings can be both primary, and secondary, to somatic–visceral bodily changes.

On the basis of the evidence outlined above and in contradistinction to James, emotional feelings may be derived from both *feedback* and *anticipation* of bodily change involved in overt behavioral–expressive activity. In line with these findings, contemporary researchers of emotion regulation, have considered both feedback and anticipation as mechanisms critical to context-appropriate behavior and expression (particularly with respect to social context). For example, Baumeister et al. (2007) have suggested that emotion serves as an anticipatory-feedback system the primary role of which is to facilitate learning through feedback since "feed-forward" bodily emotional activity operates on too slow a time scale to be of cognitive/informational value to online behavior. Emotional feelings have also been seen, consistent with Baumeister et al., as a means for anticipating other emotions – Mellers et al. (1999), Anderson (2003), Krueger et al. (2005), as cited in Gross (2007) – where actions are chosen that are expected to promote the onset of positive emotions and reduce the likelihood of negative emotions. This agrees with views of emotions researchers that hold that "emotions are motivating" (Rolls, 1999), and that "[e]motions are closely and intimately related to action by way of their nature as motivational states" (Frijda, 2004, p. 159). As a general mechanism, anticipation may also go some way to offsetting the above-mentioned latency of the emotional (action tendency) response (cf. Gross, 2007). Leventhal (1980) has viewed emotion in terms of the generation of prediction–feedback mismatch "errors." He posits the existence of emotion schemata that are "integrations of separate perceptual codes of the visual, auditory, somesthetic, expressive, and autonomic reactions that are reliably associated with emotional experiences" (p. 171) and that "man and other primates clearly respond emotionally to the disconfirmation of schematic expectations. The violation of schemata is a critical source of affective experiences and reactions" (p. 187).

If such perceptual schemata exist that permit predictive–feedback processing, however, their computational dynamics and underlying neural representation have yet to be identified. Notwithstanding, dynamic control processes have been viewed as fundamental to emotion regulation functionally realized through predictive/primary-responses and feedback/secondary-responses (e.g., Koole, 2009). In the case of the former response, emotion elicitation sensitivity may be malleable to experience while in the latter response, emotion states are regulated. In this sense, emotion regulation comprises an initial estimate of emotion relevance and also down- or up-regulated emotional activity contingent on the monitored feedback that precipitates the secondary response.

The present state of the art of emotion science, summarized briefly above, therefore, is such that, *contra* James, emotional feeling is viewed as *both* preceding and following action or action

[2]Here "cognitive" components *perception*, *attention*, *reflection,* and *evaluation* reciprocally interact with "emotion" components *arousal*, *action tendency,* and *feeling tone.*

tendency as it affords online behavior and learning. In the remainder of this article, we discuss the relationship between emotional *feeling* and *action tendency*. We investigate the extent to which they can be viewed in terms of a composite whole according to a dynamic bidirectional relationship with the neural-dynamic substrate of the former state affording a prediction of the latter state in the context of a particular emotional event. The functional and causal relationship between feeling and action tendency is discussed, above all, in relation to homeostatic- and event-based contingencies that impact on goal-directed (or more generally reinforcement–contingent) behavior (cf. Simon, 1967; Frijda, 1986, 1995, 2004, 2007, 2010; Rolls, 1986, 1999, 2005; Oatley and Johnson-Laird, 1987; Dickinson and Balleine, 1994; Cañamero, 2003; Koole, 2009; Boureau and Dayan, 2010). Complementary to James, such contingencies or junctures provide scope for addressing the question of the nature and form of non-cognitive triggers in emotional episodes.

The rest of the article breaks down as follows: in Section "The Relationship Between Emotional Feeling and Action Tendency: A review," we review classical and contemporary perspectives on *action tendencies, feelings,* and the *feeling–action tendency* relation according to existing evidence gleaned from research in neurophysiology and psychology. In Section "Emotional Feelings as Predictions of Action Tendency: A Position," we discuss the feeling–action tendency relation through the lens of our postulation that emotional feelings function as *predictions* of action tendency. We expound our notion of action tendency prediction–feedback loops (ATPFL) that regulate emotion episodes according to a neural-dynamic stable representational feeling substrate. In Section "ATPFL in Goal-Directed Behavior: An Application," we examine the role of ATPFL according to ongoing and prospective goal-directed behavior with respect to triggers rooted in reinforcement contingencies (or goal junctures, GJs) and in the context of homeostatic regulation of an existing goal/need set. In this section we make specific reference to work undertaken in AI and robotics that has utilized emotion-like mechanisms in the service of adaptive goal-regulated behavior. Finally, in Section "Conclusion," we offer some final remarks.

THE RELATIONSHIP BETWEEN EMOTIONAL FEELING AND ACTION TENDENCY: A REVIEW

Broadly, theories of emotion can be categorized according to their emphasis on one or more of *triggers*, *action and action tendencies*, and perceptual states that regard the body and may or may not integrate information from the outside world – we may broadly label such states as *feelings*. Emotion theories may focus more or less on a given constituent.

Certain theorists focus on the primacy of pre-conscious *triggers* in the emotion episode, often in the context of appraised dimensions (cf. Arnold, 1960; Zajonc, 1980, 1984; Lazarus, 1984, 1991; Scherer, 1984, 2009; Frijda, 1986, 2007) or alternatively in terms of reinforcement or/and goal-directed behaviors (Simon, 1967; Frijda, 1987, 2010, Oatley and Johnson-Laird, 1987; LeDoux, 1996; Rolls, 1999, 2005; Kreibig et al., 2010), that may relate to a "primary reinforcing stimulus" i.e., that has some pain or pleasure value, or a secondarily reinforcing stimulus, i.e., that is predictive of the primary reinforcer. Other theorists place strong emphasis on *action and action tendency* where individual emotions are either considered relatively prescribed and rigid action programs (cf. Damasio, 1994, 2010; Panksepp, 1998, 2000, 2007) of ancestral survival relevance (Tooby and Cosmides, 2008) and may involve an expressive–communicative component (cf. Ekman, 1972, 2003) or alternatively considered motivating and preparatory to, though not determining, action (cf. Frijda, 1986, 2004, 2010; Rolls, 1999, 2005; Bradley and Lang, 2000; Lang and Bradley, 2010). Finally, some theorists emphasize the role of *feelings* in emotions at subconscious and conscious perceptual levels and that may or may not concern bodily feedback (James, 1884; Damasio, 1994, 1999, 2003, 2010; Prinz, 2004, 2005; Laird, 2007; Friedman, 2010).

In the remainder of this section we will attempt, according to a broad review of (embodied; cf. Ziemke, 2008; Ziemke and Lowe, 2009) emotions, to clarify better the *feeling* and *action tendency* components of emotion as well as their temporal and functional relationship in the context of a given emotion episode that is triggered by a nebulus emotionally competent stimulus (ECS; Damasio, 1994)[3]. We reserve discussion of triggers, in the context of goal-directed and reinforcement–contingent behaviors, to Section "ATPFL in Goal-Directed Behavior: An Application."

WHAT IS AN (EMOTIONAL) ACTION TENDENCY AND WHAT IS ITS RELATION TO (OVERT) ACTION?

Discrete action programs versus motivation-grounded action tendencies

The link between emotion and action or the "impulses" to action has been apprehended since the time of Aristotle (cf. Aristotle, 1984, also see Oatley et al., 2006, pp. 11–13, for discussion). A key area of debate among emotion theorists is whether bodily activity that precipitates overt emotional behavior can be characterized according to one of two main perspectives. The first of these holds that discrete action- (or affect-) programs exist with relatively detailed and stereotyped autonomic nervous system (ANS) profiles. For simplicity, we will label this the *discrete action program* perspective. The second perspective declares that bodily activity is expressed in relatively unspecified ANS states whose influence on behavior is structurally and functionally grounded upon, and constrained by, motivational and environmental contextual factors. We label this latter view the *motivation-grounded* perspective.

As a stereotypical description, the *discrete action program* perspective, endorsed by protagonists such as Ekman (1972), Levenson et al. (1992), Levenson (2003, 2011), Panksepp (1998, 2000, 2007), Friedman (2010), Stephens et al. (2010), holds that emotions: are "hard-wired," "natural kinds," have discrete ANS-specified profiles, have evolved to enable organisms to cope with specific survival challenges, directly map/output to behavioral and expressive states, are identifiable as subjectively reported feelings (see Levenson, 2011, for summary of discrete basic emotion

[3] Scherer (2004) has criticized Damasio for his focus on the ECS as an innate emotion differentiator. Damasio (2004), however, has suggested that an ECS is underdetermined by evolution – it may acquire the ECS status over the organism's lifetime and may or may not be viewed in cognitive appraisal theoretic terms. For convenience, it is to this broad definition that we subscribe for the bulk of this article – but see Section "ATPFL in Goal-Directed Behavior: An Application."

properties). Levenson (2011) has indicated the existence of, and requisite need for (*for* basic emotion theory to hold), a *central organizing mechanism* that "(searches) continuously for meaningful patterns in incoming sensory information, recognizing survival-critical situations, and activating the appropriate emotion, which recruits and orchestrates the optimal behavioral and physiological responses". This "executive system" is conceived as an affect program (cf. Tomkins, 1962). Criticism of the *discrete action program* perspective has focused on the relatively weak evidence concerning reliable somatic–visceral profiles of activity demarcating even the basic emotions and that a (low) dimension perspective, e.g., concerning valence and arousal, offers a more reliable indicator of ANS–emotion relations (see Cacioppo et al., 2000; Feldman-Barrett, 2006; Larsen et al., 2008; Koole, 2009; Mauss and Robinson, 2009 for critical reviews). Further criticism, important for the discussion in the rest of this section as it concerns the action–action tendency relation, is that affective/emotional activity can only be understood in terms of the dynamic interchange of behavioral, physiological, and experiential components (cf. Mauss and Robinson, 2009; Lang and Bradley, 2010) rather than with recourse to a centralized system.

In contrast to the *discrete action program* position, the *motivation-grounded* position posits that emotional systems structurally (in terms of neural circuitry) and functionally build upon networks for "appetitive" and "aversive/defensive" responding (cf. Frijda, 2010). These two systems function as (tree-like) cascade networks (e.g., see Frijda, 2010; Lang and Bradley, 2010) that afford approach- and withdrawal-action possibilities. The path along this cascade may be more or less habitually traversed or require deliberation (Daw et al., 2005; Frijda, 2010, Gläscher et al., 2010). The applicable motivational states may be considered, above all, "extrinsic" as opposed to "intrinsic" (cf. Schmidhuber, 1991; Oudeyer and Kaplan, 2007, Baldassarre, 2011) where the former type is evoked by homeostatic imbalances, as a form of "negative feedback control" (Frijda, 2010, p. 573), and the latter concerns learning for the sake of learning (e.g., artificial curiosity – Schmidhuber, 2010; Luciw et al., 2011) where no specific aim or goal is necessary. The "tendency" to act is often equated with the emotion itself: "emotions are often dispositions to act rather than the actions themselves: when a stimulus of threat or appetite prompts the execution of an action procedure, preparatory metabolic changes occur in muscles and glands" (Bradley and Lang, 2000, p. 244), and "[e]motion, by its very nature, is change in action readiness to maintain or change one's relationship to an object or event" (Frijda, 2004, p. 158). The tendency, fundamentally, is rooted in a "striving" following obstructions or facilitations to goal- or need-directed behaviors (cf. Frijda, 1986, 2010; Cacioppo et al., 2000; Kreibig et al., 2010).

Attempts have been made to reconcile the *discrete action program* and *motivation-grounded* perspectives on emotional action and action tendency. Christie and Friedman (2004), for example, found that the "basic"[4] emotions (anger, fear, sadness, amusement, contentment, disgust) mapped onto the continuous dimensions of valence and activation in the case of subjective reports of emotion following presentation of emotion-inducing film clips. They found that these basic emotions, however, mapped significantly better onto the dimensions of approach–withdrawal and activation with respect to ANS activity. This result indicates that low-dimensional ANS specificity or/and neural representation may exist for arousal/activation, affective valence, and action tendencies (approach–withdrawal orientation). Mauss and Robinson (2009) also point out that discrete emotions might be envisioned according to combinations of two or more dimensions, e.g., anger = negative valence, high arousal, and high approach tendency, whereas fear = negative valence, high arousal, and high withdrawal tendency. More discussion of the dimensional perspective on emotions and action tendency will be provided in Section "Emotional Feelings: The Neural-Dynamic Representation of Action Tendency."

From the alternative angle, a given theoretical position, naturally, may not easily fit into the *discrete action program* or *motivation-grounded* emotion camp. The position of Damasio, for example, while suggesting that emotions are hierarchically grounded upon homeostatic and motivation-based neurophysiological "machinery" (Damasio, 2003), nevertheless describes emotions as action programs:

> "[e]motions are complex, largely automated programs of *actions* concocted by evolution. The actions are complemented by a cognitive program that includes certain ideas and modes of cognition, but the world of emotions is largely one of actions carried out in our bodies, from facial expressions and postures to changes in viscera and internal milieu," (Damasio, 2010, p. 109).

This tightly coupled emotion–behavior relation is, however, one to which the *motivation-grounded* advocates object. Frijda (2010) suggests that differences in autonomic activity may not so much reflect automated emotional action patterns but constitute *contingent* patterns of action readiness. With reference to Kreibig et al. (2010), Frijda points out that many different behaviors can manifest subsequent to a given emotion-relevant event. A threat stimulus, for example, commensurate with a fear state, may elicit fight or flight tendencies. Furthermore, a given behavior may be elicited according to different emotional events though its expression may alternately owe to enaction of the appetitive or defensive systems. Fight (or aggressive approach), for example, may be triggered in *either* a defensive, *or* a non-threatening appetitive, context. Following Van Hoof (1972), a cascade of broad appetitive and defensive behaviors has been identified by Frijda (2010) as providing *elaborated* states of action tendency. Appetitive behaviors include *approach*, *watch*, *open up*, *body contact*; defensive behaviors include *withdraw*, *go against*, *submit*, *detach* the latter in turn branching to subsets of possibilities – *physical*, *verbal*, or *turn back*.

The above-mentioned perspective on the role of ANS activation in the constitution of the emotion state can be understood with reference to Cacioppo et al.'s (1992, 2000) terms of *tactical*

[4]The basic emotions identified by Ekman (1972) and Levenson et al. (1992) are: anger, fear, happiness, disgust, sadness, and *surprise*. The latter emotion, omitted by Christie and Friedman (2004), has perhaps been most often contended regarding its status (e.g., Oatley and Johnson-Laird, 1987). In the case of the Christie and Friedman study, the basic emotions were derived from a pattern classification analysis of a number of emotion-relevant ANS-activating physiological variables.

versus *strategic* emotions, made with reference to Bradley (2000). A *Strategy* pertains to "underlying (ANS) organizations that direct actions in the pursuit of broad end goals," p. 183. They are centered around appetitive and aversive systems and the chief responsibility of the ANS is to "mobilize metabolic resources in response to hostile and hospitable stimuli [. . .] crucial to survival," p. 183. *Tactics*, on the other hand, refer to "specific context-bound patterns of actions." Broadly, *tactical* emotions are those advocated by the *discrete action program* position whereas *strategic* emotions are those purported by the *motivation-grounded* position. For the former position, however, and similar to Frijda, Cacioppo et al. suggest that ANS specificity may be compromised as the same emotion may relate to many behaviors. From the *motivation-grounded* perspective, the *discrete action program* position confuses *tactics*-based patterns of action with the broad, strategic emotional *biasing* of branches of cascaded behavioral possibilities. On this basis, ANS activity relevant to particular branched subsets of behaviors is not synonymous with the underlying emotion.

The exact nature of appetitive–defensive motivation systems is complex but appears to be rooted in diffusive neuromodulatory projections involving, above all, dopamine and serotonin. Gray (1982; see also Gray and McNaughton, 2000; McNaughton and Gray, 2000) proposed two types of neural system for dealing with appetitive and defensive survival challenges, the behavioral activation system (BAS) and the behavioral inhibition system (BIS), respectively. The BAS is largely composed of mesolimbic dopaminergic projections from the ventral tegmentum area to the ventral striatum, the BIS is composed of serotonergic projections in amygdala and septohippocampal regions (also see Boureau and Dayan, 2010 for perspective). The systems are seen to work largely independently and in opposition to each other where the BIS is implicated more in withdrawal tendencies and the BAS in approach tendencies. However, the extent of the existence of opponency in such systems is contended. The separate appetitive and aversive quantifying systems may work in opposition (Daw et al., 2002; Cools et al., 2010) but also in co-activation (cf. Norris et al., 2010) augmenting behavioral arousal.

Attempts to map motivational valence onto action tendencies has led to criticism of the validity of the *motivation-grounded* perspective on emotion. Stemmler et al. (2007) found somatovisceral activation ("structural") independence concerning anger–fear states and approach–withdrawal tendencies and concluded functional independence between motivation and emotion-based systems. There are several comments that can be made regarding Stemmler et al.'s conclusions: (1) as mentioned, appetitive–defensive systems do not map directly (linearly or monotonically) to approach–withdrawal systems. Defensive systems may incline withdrawal responses but can nevertheless activate fight-like approach defensive responses (cf. Boureau and Dayan, 2010; Frijda, 2010). On this basis, it may be challenging to establish an exact relationship between anger–fear and approach–withdrawal based on ANS profiles of activity; (2) the finding of only weak somatovisceral activation somewhat undermines the relevance of the results. Furthermore, even if structural independence is the case, it does not preclude functional interdependence. Emotions may have evolved for several functions (see Koole, 2009 for review of core emotion functions) and therefore some structural independence in the multi-serving emotion systems may be expected.

The dynamics of action and action tendency

The dynamic mapping of action tendency to overt action/behavior highlights difficulties in sustaining a strong *discrete action program* position. Actions, and tendencies to act, invariably involve sequences of responding which may be qualitatively different regarding profiles of physiological activation and/or approach–withdrawal tendency. Fanselow (1994) identified three classes of behavior, structured according to a cascade of possibilities, appropriate to a defensive behavior system – pre-encounter, post-encounter, circa-strike. The pre-encounter class pertains to changes in behavioral organization affecting homeostasis, e.g., feeding habits, and behaviors that may pre-empt hostile encounters, e.g., protective nest maintenance. The post-encounter class amounts to immediate threat management where BIS-like activity is enacted (e.g., freezing, potentiated startle). In the circa-strike phase animals choose from a behavioral repertoire pertinent to active coping (similar to BAS employment). Fight, flight, and display behaviors can be utilized at this threat engagement stage.

The dynamic flow concerning action tendencies, as physiological (ANS-governed) states, and overt actions has been investigated in depth by Bradley, Lang, and co-workers (cf. Bradley and Lang, 1994; Lang, 1995; Bradley and Lang, 2000, 2007; Löw et al., 2008; Lang and Bradley, 2010). Lang and Bradley (2010) illuminated that the dynamic flow of emotional action tendency and behavior is so complex as to render "impossible" the ANS specificity of emotions and action programs as retroactively attributed, as a position, to James. The *defense cascade model* (Lang et al., 1997) demonstrates the importance of viewing ANS activity preparatory to action as a dynamic process. The model (**Figure 1**) consists of three distinct phases, similar to those identified by Fanselow (1994), that map the relation between the organism and the ECS according to action – pre-encounter, post-encounter, overt behavior. However, the mapping of the physiological variables that capture preparatory aspects to a behavioral response (e.g., sweat gland activity, heart rate, startle reflex) have a non-monotonic relation to each other and to overt behavior: heart rate, for example, initially slows as the organism attends to the encountered stimulus (presumably to evaluate threat relevance) prior to an acceleration preparatory to a fight–flight response.

More specifically, the adapted diagram of the Lang et al. (1997) model (**Figure 1**) highlights the non-monotonicity of the *arousal constituents* of the motive state (emotional response). The manner in which such tendency components: constitute emotions in themselves or/and instigate overt behaviors, however, comprise issues subject to ongoing debate. Mauss and Robinson (2009), in their extensive meta-analysis of ANS specificity of emotion, concluded that of the many physiological components proffered to distinguish among basic emotions cross-study correlations between emotion experience and *individual components* are weak. Instead, a clearer relationship is apparent between *sets of physiological variables* comprising a particular affect dimension, e.g., arousal, and a given emotion.

Similar to the action tendency dynamic flow, the nature of the emotional action tendencies and how they map to overt behavioral

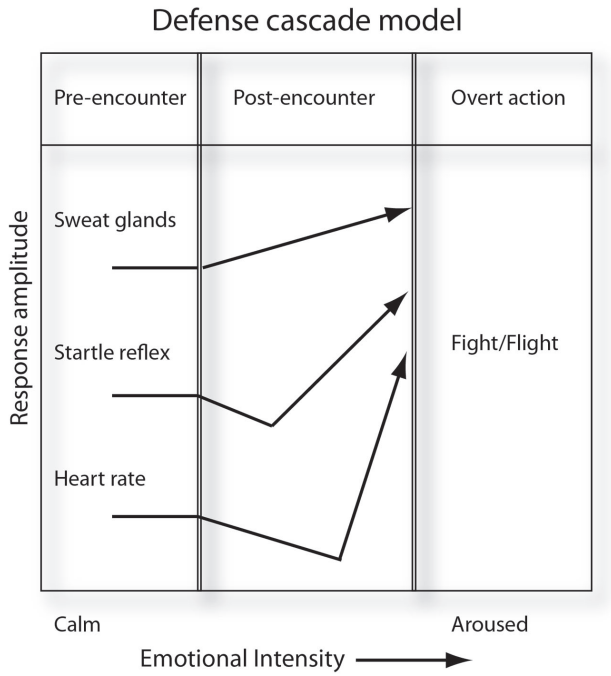


FIGURE 1 | Action as a function of action tendency variables – the defense cascade model. The relationship between bodily variable activity (arousal-relevant) antecedent to action is non-monotonic. The action tendency therefore is dynamic and changes as a function of time and/or perception of the emotion eliciting stimulus. Adapted from Bradley and Lang (2000).

response is not merely a "feed-forward" relation but is instead of a complex dynamic nature. Schauer and Elbert (2010), for example, have produced a six stage *action–action tendency* model according to fear activity that evolves into a traumatic stress response. The model is conceived according to a cascade of "Freeze–Flight–Fight–Fright–Flag–Faint" behaviors (**Figure 2**) that functions to provide the best chance for an organism to overcome a severe survival threat.

Both **Figures 1** and **2** demonstrate stages of inhibition (BIS activation) and active coping (cf. Waldstein et al., 1997; BAS activation). It is clear however, that even within this action–action tendency dynamic the BIS and BAS systems may be alternately activated during the threat episode that requires the emotional response. We may schematize the relation between action tendency and overt behavior in **Figure 3** where the gray arrows overlaying a feed-forward model serve to implement the more dynamic perspective that best fits the *action–action tendency* relation according to the above-discussed research.

While correlations in neurophysiological states and particular subjective ratings of emotions may exist, it is not obvious how the individual *action and action tendency* components cohere into a global categorizable state that is greater than the sum of its parts. Such a gestalt would help rebuff the accusation of emotions as being epiphenomenal. This lack of componential coherence has been claimed to be a problem for the *discrete action program* position (Mauss and Robinson, 2009). However, it is also of concern for the *motivation-grounded* position. Bradley and Lang (2000) acknowledge that "no system can be defined by a single subsystem measure" (p. 245) where subsystem refers to a given single dimension of behavior–physiology-language emotional *action and action tendency* space but where "the three response systems have no obvious common metric." For Prinz (2004), this represents an example of the "problem of plenty" in emotion theory: "if all parts [emotion constituents] are essential, how do they hang together into a coherent whole? . . . the Problem of Plenty asks for an essential function of emotions in virtue of which they may have several essential components" (p. 18).

Naturally, a relative lack of tractability does not preclude the existence of coherence which may necessarily only be conceived in complex dynamic terms, e.g., pertinent to stabilizations and transitions between sequenced sensorimotor components. Lewis (2005) has posited a (psychoneurophysiological) dynamic systems (DS) account of emotional states, incorporating action tendency components that stabilize through their interaction with appraisal constituents culminating in global attention orienting states ("emotion–appraisal amalgams"). On this basis, appraisal is considered the end point of the emotional episode that, through stabilized neural activity, enables learning of enduring action (e.g., goal-directed) plans (cf. Lewis, 2005, p. 177). Carver and Scheier (1998) – see also Carver (2006) and Carver and Harmon-Jones (2009) – have proposed a *motivation-grounded* DS perspective on emotional activation. Carver (2005) criticized Lewis' assertion of the existence of a psychoneurophysiological stabilization mechanism for emotion. In the Carver and Scheier perspective, emotions and motivations serve as control (feedback) systems whose activations stabilize but in accordance with *behavioral* as well as (psycho-) neurophysiological activity.

Coherence among action tendency components may also occur through a process of development and learning which may constrain self-organized activation patterns. Camras (2011), for example, claims that peripheral motor system components may become associated following learning (also see Berkowitz, 2000). For Camras affective (facial) expressions are considered motor coordinated structures – the coincident activity of groups of muscles – that via hebb-like processes, come to recruit/entrain activity in other muscles or muscle groups such that a coordinated expression develops. Furthermore, according to Laird, who also refers to the same three *action and action tendency* systems as Bradley and Lang (2000; i.e., behavior, physiology, and language), the deployment of these systems may have been coordinated as a consequence of both learning and Darwinian evolutionary pressures.

Dynamic neural activity may, therefore, enable coherence in the physiological domain as it concerns action tendency components across a sequence of behavioral phases constitutive of an emotion episode. The specific dynamic processes that enable this coherence offer a means of providing a firm mechanistic and functional foundation to a *motivation-grounded* perspective of emotion. Another fundamental means of component synchronization may be the *regulation* of emotion. This is the major topic of Sections "Emotional Feelings as Predictions of Action Tendency: A Position" and "ATPFL in Goal-Directed Behavior: An Application."

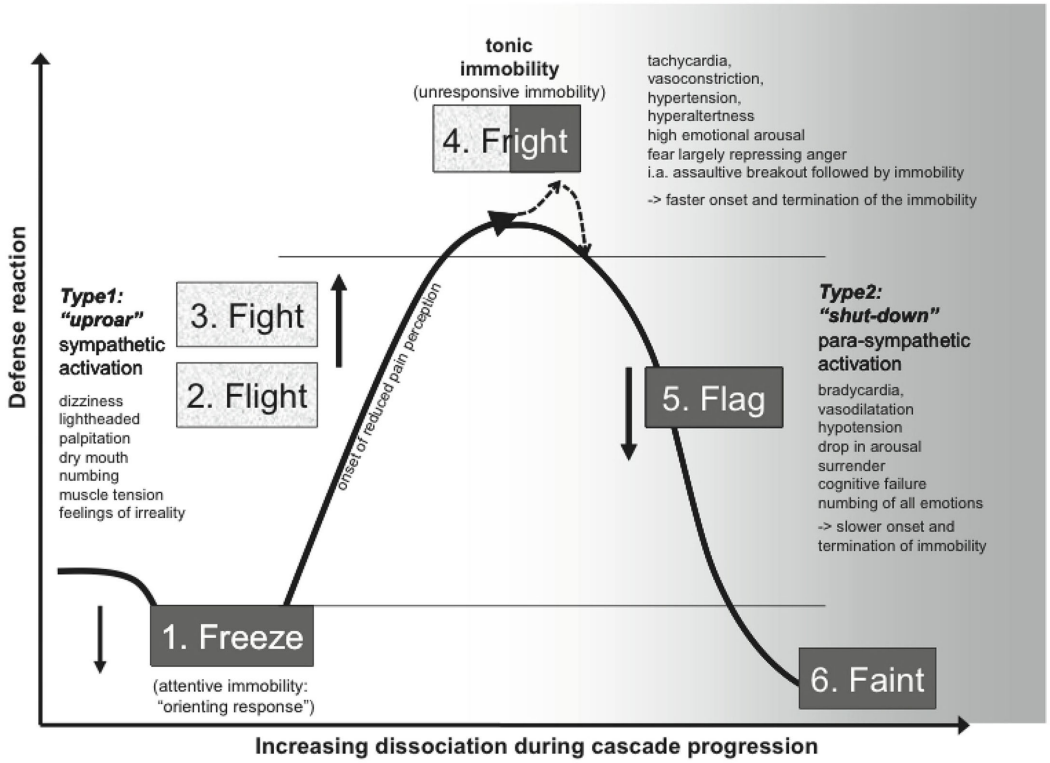


FIGURE 2 | Action and action tendency mapping in fear–trauma responding. Here, is provided an example of the complex non-linear relationship between bodily variables constitutive of the evolving action tendencies and the concomitant overt behaviors. Taken from Schauer and Elbert (2010). Reprinted with permission.

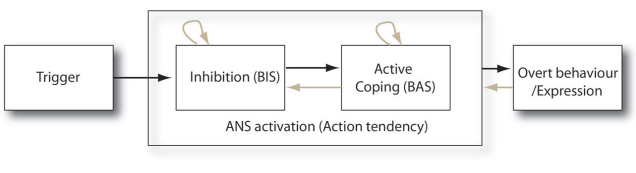


FIGURE 3 | The relation between action tendency and action. The gray arrows superimpose a dynamic perspective onto an otherwise conventional feed-forward model of emotional behavior. Only the "dynamic" model is in line with the *motivation-grounded* perspective.

WHAT IS AN (EMOTIONAL) FEELING AND WHAT IS ITS RELATION TO ACTION TENDENCY?

Emotions and *feelings* are popularly conflated terms in everyday parlance. James put forward the counter-intuitive perspective of feelings as *following* rather than *preceding* actions. Nevertheless, feelings, for James, are perceptions of body states that are *synonymous* with the emotion states. One of the most oft-cited perspectives on feelings is offered by James:

> "[o]ur natural way of thinking about these standard emotions is that the mental perception of some fact excites the mental affection called the emotion, and that this latter state of mind gives rise to the bodily expression. My thesis on the contrary is that the bodily changes follow directly the PERCEPTION of the exciting fact, and that our feeling of the . . . same changes as they occur IS the emotion"
>
> (James, 1884, p. 191).

The perception of bodily changes preparatory to action is at the core of somatic–visceral theories of emotion and emotional feeling as pioneered by James. A century on from James, a number of somatic–visceral theorists that broadly fall into the *discrete action program* and *motivation-grounded* camps have put forward different accounts of the relation between feeling and: (a) conscious or unconscious perception, (b) emotions, and (c) action tendency. The precise causal and functional nature of the relation between (conscious or unconscious) emotional feelings and actions and action tendencies, however, requires recourse to the underlying neural dynamics that may permit a representational substrate on which such feelings can manifest.

Emotional feelings: the perception of action tendency

Extending the James' (1884)[5] position, Damasio (1994, 2010), Prinz (2004, 2005), and Laird (2007; see also Ekman, 1972,

[5]James did not advocate that feelings are captured by, or synonymous with, particular neural patterns.

2003 and Friedman, 2010) are among the key *discrete action program* theorists that posit that emotional feelings are (neurally) registered states capturing somatovisceral changes comprising action states/patterns. However, depending on the theorist in question, feelings may be thought of as conscious or unconscious, and may be synonymous with, or separable from, emotions. The Jamesian view on feelings is that they are synonyms for emotions and thus not separable (see Prinz, 2004 for discussion); they are also consciously experienced. Damasio's perspective on feelings and emotions, however, differs in both respects. Firstly, Damasio views feelings and emotions as separable, distinct phenomena. Simply, emotions are about action programs (see previous sub-section). Emotional feelings, on the other hand, are primarily perceptions of action commands or programs:

> "[f]eelings of emotion [...] are composite perceptions of what happens in our body and mind when we are emoting. As far as the body is concerned, feelings are images of actions rather than actions themselves; the world of feelings is one of perceptions executed in brain maps"
>
> (Damasio, 2010, p. 110).

Both Damasio and Prinz, as somatic/behavior emotion theorists, do not view emotional perceptions as being necessarily consciously experienced. James, contrarily, uses labels for internal bodily "perceptions," "feelings," and (self-) "consciousness" interchangeably. However, whilst Damasio does not contend that feelings need be conscious, Prinz views them as experiential. Finally, for Prinz, similar to James, perceptions *are* emotions which, as stated above, differs from Damasio:

> "[w]hen emotions are felt, the feeling is the emotion: the emotion is a conscious perception of a patterned change in the body. But emotions can go unfelt: they can be unconscious perceptions of patterned changes in the body"
>
> (Prinz, 2005, p. 17).

Laird (2007), on the other hand, distinguishes between feelings, as types of "self-perception" and consciousness in his somatic/behavioral feeling theory: "Self-perception theory assumes that feelings like these (happiness, hunger) are higher order integrations of various kinds of cues. As with depth perception, the process of detecting and integrating these cues is automatic and occurs outside consciousness," (p. 10).

Notwithstanding these contentions, a perspective that unites *discrete action program* theorists is that emotional feelings can be engendered and modulated by bodily feedback. Emphases of modern theorists, however, diverge with respect to the extent to which proprioceptive and interoceptive processes are involved. According to Damasio (2010), proprioceptive processing entails "images of specific body components such as joints, striated musculature, some viscera" and interoceptive processing entails neural activation "mapping" of "the functional condition of body tissues such as the degree of contraction/distension of smooth musculature; parameters of internal milieu state," (p. 76). Ekman has focused on the role of proprioceptive feedback[6] (initially embraced by James but later subordinated to interoceptive feedback) especially in facial expression: forcing oneself to smile can literally make one happier. Laird (2007) has similarly pushed a proprioceptive feedback – feeling theory: "self-perception theory." On this account, emotional feelings are primarily caused by bodily feedback. Prinz (2004, 2005), on the other hand, emphasizes interoception as a means of somatic feeling constitution. Similar to James (and Laird), Prinz views the bodily state changes intrinsic to the emotion as preceding the feeling state which are then registered and represented in neural states.

While Damasio (1994, 1999, 2003, 2010), also holds a "strong" Jamesian-somatic, i.e., predominantly interoceptive, view of the primacy of body in emotional feelings, in his view, feeling states need not *always* be subsequent to the bodily changes underlying emotional activity. According to Damasio (2010), there are three means by which feelings can be constructed: (1) by registrations of bodily changes induced by ECS in the brain-stem and cerebral cortex, (2) by an "as-if" body loop (Damasio, 1994; Bechara, 2004), (3) by misrepresentative "hallucinations" of bodily changes. In the case of (2), "after emotions have been expressed and experienced at least once" (Bechara, 2004, p. 38), those brain areas that ordinarily trigger bodily changes constitutive of the emotional feeling become able to activate parts of the brain responsible for registering bodily changes. This "simulation" of bodily change may happen (a) prior to, but in parallel with, those bodily changes, (b) in the absence of the bodily changes. In the case of (3), Damasio suggests in certain instances the brain can be duped into misrepresenting the body and ECS-induced changes therein. Examples of this phenomenon include cases in which analgesic medicine is provided to temporarily alleviate pain "fooling" the brain into a feeling percept of non-bodily irritation or where, in the service of promoting survival related behaviors, the brainstem may "disengage" from the body regarding the representation of pain.

Many *motivation-grounded* emotion theorists have considered the relation among action, action tendency and feeling emotional components. Frijda suggests, *contra* Damasio, that *tendency* to act rather than acting *per se* is at the heart of emotional experience:

> "emotional feeling is to a very large extent awareness, not of the body, but of the body striving, and not merely of the body striving, but the body striving in the world ... [e]motional experience is, to a large extent, experienced action tendency or experienced state of action readiness,"
>
> (Frijda, 2004, p. 161).

Cacioppo et al. (1992, 2000) – also see Larsen et al. (2008) – have argued that the relation between action tendency (as manifested in ANS patterns of activity) and experiential feeling states can be understood as a function of cognitive elaboration/disambiguation of ANS activation patterns. The researchers' "Somatovisceral Afference Model of Emotion" (SAME) describes three (historically)

[6]Ekman and colleagues have, however, investigated physiological (interoceptively processed) correlates of emotion – see, for example, Levenson et al. (1992).

prototypical cases: (1) "unambiguous" patterns where a hypothetical highly specified ANS pattern for a discrete emotion may be experienced without cognitive deliberation beyond pattern recognition (the pro-James perspective); (2) "general arousal" which requires a cognitive extraction of context for emotional experience to occur (essentially the proposal of Schachter and Singer, 1962); (3) "ambiguous" which requires a combination of cognitive "priming" and (ANS) pattern recognition in order for emotion experience to ensue. In the case of (3), an ANS-activated pattern of somatic and visceral change may be ambiguous, i.e., consisting of only a few physiological dimensions/variables, affective "valence" being primary. The ambiguity may be resolved by recourse to cognitive processing or biasing ("priming" relating to context) giving rise to the differentiated emotional experience. This experience is considered a "somatovisceral illusion" in that a fuller somatovisceral state that may map to a given subjectively experienced/reported discrete emotion may not initially exist and the full gamut of physiological responses associated with discrete emotional states may in fact only be galvanized by the experiential feeling following cognitive discernment. This notion is somewhat similar to Damasio's "as-if body loop" insofar as feeling can precede full emotion-relevant somatovisceral activation.

Emotional feelings: the neural-dynamic representation of action tendency

In relation to the previous sub-section, it is incumbent on this review to illuminate how the felt action tendency corresponds to neural-dynamic activity. A key question, in line with the above-mentioned perspective of Prinz (2004), is: to what extent can neural-dynamic activity be considered *representative* of the underlying action tendency? According to the philosopher Dretske (1981, 1986) for a phenomenon to count as representational it must be fallible. Prinz (2004), with reference to Dretske's philosophy, offers the example of a dog representation that might, on occasion, mistake a wolf, for example, for a dog. The fundamental point is that the "dog concept is a mental state that is reliably caused by dogs *and* was acquired for that purpose [. . .] After that state is formed, it carries information about dogs, foxes, and wolves, because all these things can cause it to activate, but it only *represents* dogs, because it was set up as a result of dog encounters" [authors' italics]. The point made here is that for a neural pattern to constitute a representation of action tendency that is a substrate for feelings, it should have the *purpose* of (e.g., have evolved for) representing action tendency. Essentially, the feeling neural pattern would comprise an effective and fallible prediction of what action tendency will occur following emotion event triggers. From the point of view of *minimizing* error, a parsimonious representation of action tendency is desirable where it is sufficient to distinguish functional (i.e., emotional) states from non-functional states; that is, states that are of informational benefit to the organism. Appeal to parsimony also respects the need for swift and efficient processing in the face of dynamic environmental threats or/and appetitive opportunities. Naturally, a corollary of holding this perspective is that the *discrete action program* position postulating relatively high dimensional ANS specificity for basic emotions may be subject to great potential for misclassification of somatovisceral patterns[7].

An alternative view of emotions is that they can indeed be captured according to just a few dimensions. As mentioned in the previous section, it has been suggested that basic emotions may be mapped onto a low-dimensional space where affective valence, arousal/activation, and approach–withdrawal tendency are at the core (cf. Christie and Friedman, 2004). Russell (1980, 2003) has provided a model of "core affect" whereby affective valence and arousal account for basic emotions according to a circumplex. However, it has been noted that two dimensions may be insufficient to account for differences in particular negative emotions, e.g., fear and anger. The approach–withdrawal dimension has been championed by Davidson (1993), Watson et al. (1999), Carver (2006), Mauss and Robinson (2009), Koole (2009) such that anger–fear emotions may be at least *partially* discerned by ANS activity – diastolic blood pressure difference (see Sinha et al., 1992) – or by differential left–right brain hemispheric EEG activity (Mauss and Robinson, 2009). The qualitatively similar dimension of dominance has also been posited (Russell and Mehrabian, 1977; Mehrabian, 1996, 1997) – this dimension has otherwise been conceived as "potency" (Osgood et al., 1957). The three dimensions of Russell and Mehrabian (1977) – pleasantness, arousal, dominance – referred to as PAD, is a convenient acronym for referencing valence, arousal, and orientation (approach–withdrawal) dimensions. The fact that these three dimensions, that appear necessary and sufficient to discriminate among the basic emotions, have been extrapolated according to factor analyses using self-report measures is indicative of their importance in emotion and, more speculatively, in (conscious or unconscious) feeling.

As mentioned in the previous sub-section with reference to the work of Cacioppo et al., it may also be possible that emotional feeling states are grounded in low-dimensional neural-dynamic activity that stimulate more elaborated somatovisceral responses. Insofar as these neural-dynamic patterns should be considered representational, the feelings would *come to represent* somatovisceral states over the emotion episode. Whether such a dynamic process should be considered representational, however, is arguable given the purported brain–body transformative nature of the somatovisceral state. It may be more confidently asserted that the nascent feeling state represents valenced motive states (cf. Frijda, 2010) or core affective phenomena that is then elaborated into fuller emotional bodily and feeling states the constituent activity of which being distributed across brain and body. On the other hand, whether or not a feeling state comes to represent somatovisceral activity may depend on whether a self-organized process, such as that inherent to Cacioppo et al.'s view, culminates in *stable activity*, i.e., elevated (above baseline) neural activation levels persistent in the face of perturbations (e.g., noise). As previously mentioned, Lewis (2005) has posited such an emotion episode whereby following an emotion trigger,

[7]Mauss and Robinson (2009) identify at least seven dimensions regularly cited according to *discrete action program* sympathizers where only a comparatively narrow range of values for each dimension is permissible for a given emotion categorization.

emotion–cognition components integrate in a self-amplification process culminating in a stable state. Stability may thereafter allow for persistent action orientations and availability of learning affordances.

In this view, stability is a critical *pre-condition* to higher cognitive and behavioral activity. It may also be a requisite feature of a *full* or *functional* emotional response. Stable activation patterns may not just be a self-organized *outcome* of emotion–cognition component integration but a *pre-condition* to further integration of those somatovisceral afferents whose temporal dynamics manifest on a slower time scale to emotion-relevant neural–cognitive processing. It is such stability that may lay the foundation for functional representations – in the sense of Dretske – of action tendency.

The DS perspective of Scherer, broadly, exemplifies the above view. Scherer (1984, 2004, 2009) suggests feelings can simultaneously monitor and trigger emotional change whilst registering integrated inputs from motor expressive, cognitive, physiological, and action tendency systems: "I propose to view the feeling component of emotion as a monitoring system that consists of a central representation of the response organization, including the underlying cognitive processes in an emotion episode . . . it integrates the representation of changes in the other components during the duration of an emotion episode" (Scherer, 2004, p. 137). Furthermore, for Scherer: "[t]he feeling component has a special status in the emotion process, as it integrates and regulates the component process" (Scherer, 2004, p. 138). In this sense, action tendency and feelings have a bidirectional relation in the emotion episode but feeling simultaneously monitors and participates in the full emotion episode.

Of what might the neurocomputational properties of stabilization dynamics[8] be comprised? Stabilization dynamics have been mathematically and computationally formalized through the differential equations of Amari (1977) initially deployed to model the topographic spatial representations in the visual cortex according to neural fields. Dynamic field theory (DFT) has since been particularly noteworthy in capturing infant cognitive–behavioral phenomena (cf. Thelen et al., 2001). However, it has also been posited to be of relevance to modeling emotional phenomena in the context of Bechara et al.'s (1994) Iowa gambling task (Lowe and Ziemke, 2010; Lowe et al., 2010b). The DFT approach has spawned a perspective on representations in the brain that map cognitive phenomena to continuous dimensions. Stabilization occurs where activation on a given site on the field exceeds a given threshold consequent to local excitation the degree of which being determined by an interaction kernel. Schöner (2008) has listed the different types of stable attractor dynamics that neural field theory permits. Fundamentally, *self-stable states* occur contingent on the presence of input. In this sense, there is a bistable attractor dynamic since activation will gravitate toward the stable suprathreshold level or to a baseline of activation as a function of input. On the other hand, *self-sustainable states* require initial input to achieve suprathreshold activity but stability is thereafter resistant to the withdrawal of the stimulus. It is only the sites on a field that have suprathreshold activation that are considered units of representation (cf. Schöner, 2008; Sandamirskaya et al., 2011) which may also be considered units of cognition to the extent that they are then capable of impacting on the activity of sites in different fields. Field activation is non-representational and non-cognitive when subthreshold since it does not influence other field site activation (it is not "for" anything at such a stage). Nevertheless, activated sites on a field may be more or less subthreshold depending on memory inputs that effectively prime or predispose activation at particular sites.

Since emotional states are generally postulated to require an ECS (or event) trigger whose offset (withdrawal) is antecedent to emotional dampening the more reasonable stabilization dynamic for emotional feelings is the *self-stable state*. The onset and offset dynamics (gain/slope) of the feeling state, however, could be modulated consequent to experience, personality, and the degree of above-threshold activation following ECS input. Experience of a *self-sustainable state* might pertain to one of a number of pathological conditions where following ECS input the agent fails to destabilize emotional feeling (e.g., in the case of "sham rage"). One could imagine an ontogenetically emergent attractor landscape that is comprised of multi- (low-) dimensional fields (e.g., representing core affect or perhaps PAD space). The landscape might predispose particular sites on the field to be active (to produce self-stable states) according to personality-dependent past experience, while low-level somatovisceral inputs would predispose (suprathreshold) emotional feeling states following ECS trigger onset constituting something like "moods." Through the provision of a non-smooth/non-linear continuous space an observable mapping from dimensional to discrete emotion space is thereby afforded. This attractor landscape could thereby suggest a mechanism to explain the Christie and Friedman (2004) result of continuous dimension–discrete emotion mapping. The computational investigation of stability dynamics in the context of an emotion episode may unveil an important bridge between dimension theory and discrete emotion theory.

Finally, at a neural–anatomic level of description, Lewis has cited the existence of interacting *object evaluation*, *monitoring*, and *action* loops centering on key hub neural structures, e.g., amygdala, prefrontal cortex, and brain-stem, that permit stability dynamics. These areas are also critical to Damasio's account of emotion feeling generation and are oft-cited in theories of emotion (cf. LeDoux, 1996; Rolls, 1999, 2005; Davidson et al., 2007; Quirk, 2007; Pessoa, 2008, 2010). Damasio has identified convergence–divergence zones (CDZs) in the brain for neural exteroceptive, interoceptive, and proprioceptive maps (the feeling substrate within the brain). At the top levels of the hierarchy, CDZs are said to represent "dispositions" of how to *construct stable representations*. Through a mechanism of "time-locked retroactivation" (Damasio, 2010) coincidence of activity in brain sites activating during the mapping of particular features enables learning and recalling of complex representational

[8]The term *stabilization dynamics* may appear as a contradiction in terms; however, it captures the neural-dynamic essence of a non-static stabilization–destabilization phenomenon based on the existence of attractors. A stable state will resist perturbatory inputs but strong inputs may destabilize activity shifting the state into another region (e.g., another attractor).

contexts. Another key feature for promoting stabilization processes is phase synchronization[9]. Damasio has suggested that this can be observed in sub-cortical regions that might be responsible for "primordial" feelings which consist of coarse mappings of body changes: "the superior colliculus (a sub-cortical convergence zone station for visual, auditory, and body states inputs) is the only brain region outside the cerebral cortex known to exhibit gamma-range oscillations," (Damasio, 2010, p. 86).

EMOTIONAL FEELINGS AS PREDICTIONS OF ACTION TENDENCY: A POSITION

Based on the brief review in Section "The Relationship Between Emotional Feeling and Action Tendency: A review" of emotional: (1) actions and action tendencies, (2) feelings, and (3) feelings–action (tendencies) causal relations, in this section we discuss a possible role for emotional feelings of action tendencies as predictive states that serve homeostatic emotion regulation. We start the section by providing a context of *prediction as core to function of whole brain activity* which thereby may generalize to emotional activity as captured by the action tendency – feeling relation.

PREDICTION IN THE BRAIN

Some recent theories of core brain (above all neocortical) functioning have highlighted *prediction* as underlying neural and psychological processing. Hawkins (2004), for example, when addressing his theory of human intelligence being rooted in a (neo-) cortical hierarchy, suggests "(prediction) is the primary function of the neocortex and the foundation of intelligence" (p. 89). Hawkins suggests that ("invariant representational") memory provides a critical substrate for thinking through the mechanism of prediction: "What we perceive is a combination of what we sense and of our brains' memory derived predictions" (p. 87). One reason why prediction based on memory is crucial is simply processing time and thus "educated guesses" are a requisite feature of neural processing. Classical Artificial Intelligence methods have invoked listing all attributes of scenes in lookup tables which affords a processing technique – searching through the table of properties to find a match or optimal solution – which is not amenable to the processing of neurons, it would take far too long. Another reason for the necessity of prediction is functional. Tracking the trajectory of a ball, either purely visually or with respect to attempting to position oneself for catching, based solely on (sensory) feedback ensures failure at the task. This is essentially an exteroceptive problem of the delay inherent in neural processing time with respect to the real-time dynamics of world events[10]. Downing (2009) also points out that basic locomotion requires prediction and that reliance on control theoretic techniques in engineering based only on sensory feedback ensures a mismatch between motor capabilities and processing speed.

The focus of Hawkins, following Mountcastle (1978, 1998), has been on sensory and motor (primarily exteroceptive and proprioceptive) processing. However, Hawkins argues that *all* neocortical brain function deals in the currency of prediction:

> "[a]ll regions of your neocortex are simultaneously trying to predict what their next experience will be. Visual areas make predictions about edges, shapes, objects, locations, and motions. Auditory areas make predictions about tones, direction to source, and patterns of sound. Somatosensory areas make predictions about touch, texture, contour, and temperature,"
>
> (Hawkins, 2004, p. 88/89).

Hawkins has proposed that hierarchically structured layers of cortical columns receive a combination of bottom-up, top-down, and lateral inputs. The latter inputs are critical to producing a type of attentional winner-take-all effect whilst the top-down versus bottom-up processing establishes a comparison between a high-level (invariant) prediction of the contextual nature of the stimulus processing at a particular level of abstraction (relative to the hierarchy) which is compared against bottom-up inputs from a number of cortical column inputs that feed back specific details relevant to the stimulus processing. Predictive error signals can occur where the processed details do not meet with the high-level contextual expectations and such signals are then sent up the hierarchy (with the hippocampus sitting atop of the cortical hierarchy as the predictive beacon) in order to establish whether the input detail can fit within a broader (higher level) context.

Downing (2009) has identified a number of structures that partake in predictive processing: cerebellum, basal ganglia, and thalamocortical loops, the latter of which he suggests is a mechanism by which Hawkins' theory of error signals relaying up the cortical hierarchy may be achieved. Thalamocortical loops are understood to be a key means by which such predictions can occur and be updated through ongoing sensorial feedback and allow for predictions of *sequences* of states (cf. Rodriguez et al., 2004; Granger, 2006, Sherman and Guillery, 2006; Downing, 2009). Hesslow (2002), has argued, that sequences of sensory perceptual states and also motoric activations involve internal *simulation* of overt behavior that constitute ever more distal forms of prediction of consequences of sensory and motoric activity as an organism relates to its environment (see also Jeannerod, 1994). In a similar vein to Damasio (1994), regarding simulation of the interoceptively processed body, motor structures may be activated in the absence of overt expression as may sensory cortex in the absence of external sensory stimulation.

Simulation has also been invoked as a concept to explain predicting sequences of sensory states according to a dynamic systems (DS) perspective (Friston and Kiebel, 2009) and in the context of predicting social situations (inferring intentionality of others from behaviors). The predictive coding hypothesis (Friston, 2002, 2003; see also Kilner et al., 2007) proposes the existence of hierarchies of DS in the brain (cortical hierarchy) that implement an empirical Bayesian inferencing network. Within this framework it is proposed that states (e.g., goal states of others) can be inferred through a process of prediction error (PE) minimization at all levels of the cortical hierarchy. The focus on PE minimization, through reciprocal or/and recurrent interactions between levels of the hierarchy, distinguishes the predictive coding perspective

[9]This is somewhat abstractly captured by the population dynamics (local excitation/global inhibition) of DFT.

[10]Damasio (2010) refers to exteroceptive neural "maps" in the brain which track external events a given object of which "engages a sensory probe such as the retina, the cochlea, or the mechanoreceptors of the skin" (p. 76).

from that of Hawkins (2004). Another difference is that predictive coding does not signify forecasting (i.e., of future events) which is the property of *prospective* coding (cf. Prinz, 2007) but rather refers to predictions of current states based on present information. Similar to Hawkins, on the other hand, is the notion that higher levels of the cortical hierarchy represent, or *come to represent,* predictions in lower levels of the hierarchy. In the predictive coding framework, predictions from lower levels of the hierarchy can feed back to higher levels and update existing predictions. This self-organization prediction–feedback process only stabilizes at the point at which PE minimization is achieved and probable (goal) state can be inferred from (action) input.

Prediction error minimization appears to be a key mechanism for all forms of learning. Theoretically well grounded learning rules have been established providing normative models (i.e., that guide functional understanding) in the context of motivational systems such as the Rescorla and Wagner (1972) model and a mathematically similar model that accounts for temporal dynamics – the temporal difference (TD) learning algorithm (Sutton and Barto, 1990, 1998). TD learning can explain much animal neurobehavioral data (e.g., Suri and Schultz, 1998; Suri, 2002; Roesch et al., 2007) but also higher order learning in humans (cf. Seymour et al., 2004). The predictive dynamics of appetitive and aversive/defensive networks in the brain are thought to be organized around key neuromodulators emanating from brain-stem regions that act in accordance with TD learning (cf. Wörgötter and Porr, 2005; Niv, 2009; Samson et al., 2010 for reviews). Above all, dopamine (DA) is implicated in reward and action signaling (cf. Schultz, 1998, 2007) and serotonin (5HT) in punisher and inhibition/withdrawal signaling. It has been suggested that the two types of signal may provide the key outputs of an opponent process system (e.g., Daw et al., 2002; Dayan and Huys, 2008; Cools et al., 2010; Norris et al., 2010) critical to arbitrating appetitive and defensive behaviors. It might be speculated that these signals parsimoniously provide compressed information to feeling representational networks concerning affective state as rooted in reinforcement contingencies. For example: a pleasantness dimension ("P") may simply imply a *negation* of dopamine and serotonin signals where DA negative PEs and high 5HT will tend toward an overall representation of negative P; an arousal dimension ("A") may imply a *conjunction* of dopamine and serotonin magnitude signals reflecting overall strength of activity of the appetitive and defensive networks; an approach–withdrawal or dominance ("D") dimension may imply a *negation* of signal magnitudes since DA positive and negative prediction errors *statistically* concern appetitive contexts relevant to approach behaviors even though the relation between such action orientation tendencies and appetitive–defensive systems is not absolute (as mentioned with respect to Frijda, 2010).

Other recent theory in neuroscience and physiology indicates that interoceptive (somatovisceral/motivation-based) processing may also be of a predictive nature insofar as it concerns homeostatic activity. The notion of allostasis (Eyer and Sterling, 1977; Schulkin, 2003; Sterling, 2004; Woods and Ramsay, 2007) concerns a rethink of the classical control theoretic perspective on homeostasis revolving around feedback loops respecting set points that demarcate "ideal" states. According to Sterling (2004), allostasis can be conceived in terms of prediction where brain areas implicated in planning and decision making (for Sterling, above all prefrontal cortex and amygdala) are viewed as supplying inputs that may override other inputs that signal errors from ideal homeostatic balance. Such overriding of "basal" homeostasis operates in the service of supplying the organism with the resources previously learned to be necessary to meet predicted environmental pressures. Sterling considers allostasis as a means of permitting adaptive bodily regulation according to "stability through change" which accounts for both internal needs and external pressures (or opportunities) and compares to the Bernard notion of "stability through constancy."

EMOTIONAL FEELINGS: ACTION TENDENCY PREDICTION–FEEDBACK LOOPS

In this section, we make explicit a potential role for prediction in the emotional feeling of action tendency. The position we put forward is extrapolated according to the review provided in the previous sections. We postulate that somatovisceral prediction–feedback loops are essential for ongoing learning and online behavior across an emotion episode. Stable (suprathreshold) activation in a neural-dynamic substrate is suggested to provide a critical foundation on which prediction–feedback loops can operate. Subthreshold activation may *come to represent* (through self-stabilization) action tendency following a) strong predictions of action tendency, or b) in the absence of relative certainty, according to bodily feedback given appropriate priming at neural activity registration sites. This process can enable efficient emotion regulation where mismatch between neural representations and actual action tendencies can be down-regulated by bodily feedback and relatively strong action tendencies may up-regulate or instigate suprathreshold activation in the "feeling" neural-dynamic substrate. This is the raw essence of the ATPFL perspective. We will discuss it in relation to emotion regulation in the remainder of this section. Section "ATPFL in Goal-Directed Behavior: An Application" focuses specifically on how the theoretical position we put forward can explain regulation of goal (or need) directed behavior. From this point, we will use the acronym ATPFL in place of "action tendency prediction–feedback loop(s)."

The "As-if body loop" as an ATPFL

To what extent can emotional feelings be viewed as being based on predictions of action and action tendency? The idea of emotional feelings predicting behaviors is implicit in many perspectives. Frijda (1986, 2004, 2007), for example, advocates that emotional feelings are concerned, above all, with action tendencies that anticipate actions though he also notes that the relationship between action tendency and action is complex: "The link between emotion and action is intimate; yet it is weak. Anger has intimate links to aggression, but few angers actually go that far" (Frijda, 2004, p. 163).

The Damasio (1994) and Bechara (2004) perspective on emotions and emotional feelings also may be interpreted in terms of prediction. In regard to Section "Emotional Feelings: The Perception of Action Tendency," we might extract from this view the following functions that underlie emotional feelings: (1) the as-if body loop *predicts* how the body will be affected by a given ECS;

(2) the body loop provides *feedback* that may confirm or disconfirm the effective as-if body loop prediction; (3) the hallucinatory aspect of feeling is, partially, a side effect of attention filtering whereby those body changes predicted to be important to survival will be attended to, e.g., pain during flight will tend not to be felt as it affords no benefit to the escape act.

The above, arguably, combines, and extends perspectives put forward by Laird (2007) and Prinz (2004) concerning the functions of emotional feelings. For Laird, neural-dynamic represented bodily states provide a means for comparing predicted and actual (feedback) states as a type of cybernetics control process. For Prinz, stable representations (or stabilized activation states that *come to represent* the body) allow for tracking of core relational themes via filtering noisy sensory input. According to this extrapolation, the sequence of events in an emotion episode can be described as follows (visualized in **Figure 4**). The ECS is perceived (e.g., the snake). Brain areas implicated in evaluating the significance of the stimulus (e.g., amygdala, ventral-medial prefrontal cortex) activate elicitor sites in the brain-stem and thereby simultaneously instigate emotional bodily changes and activate regions of the brain responsible for registering bodily feeling states. The first sites to be activated in the brain-stem register coarse feeling activation patterns and those in turn activate parts of the brain (e.g., somatosensory cortices, anterior cingulate cortex) that register more context-elaborated feeling activation patterns (according to convergent exteroceptive, interoceptive, and proprioceptive inputs). However, by this stage, in accordance with the as-if body loop hypothesis, the more context-based feeling states have already been triggered by the ECS evaluation sites. This means an early, contextual prediction of body state can be constructed while input from coarse patterns in the brain-stem constitutive of early stages of body change registrations provide (1), a coarse prediction–feedback loop, and (2), the initial phases of feedback control to the contextual prediction–feedback loop in the cortex. Bodily feedback can be seen as an ongoing process of comparing neurally registered body states to actual body states. This allows the organism access to an embodied dynamic comprising a representation of a prediction of how its body will be perturbed by the perceived ECS.

This description of an emotion event broadly captures the essence of the ATPFL. However, it may be the case that subthreshold activation exists in body registering brain sites. Such activity comes to represent the body to the extent that it moves to a suprathreshold state, e.g., following input from the body that strengthens the neural activation. The ATPFL explanation suggests, therefore, that the as-if body loop, consistent with Damasio, is functional in situations of relative certainty concerning the relation of the ECS to the agent whereas uncertainty requires bodily feedback (a type of somatic marker).

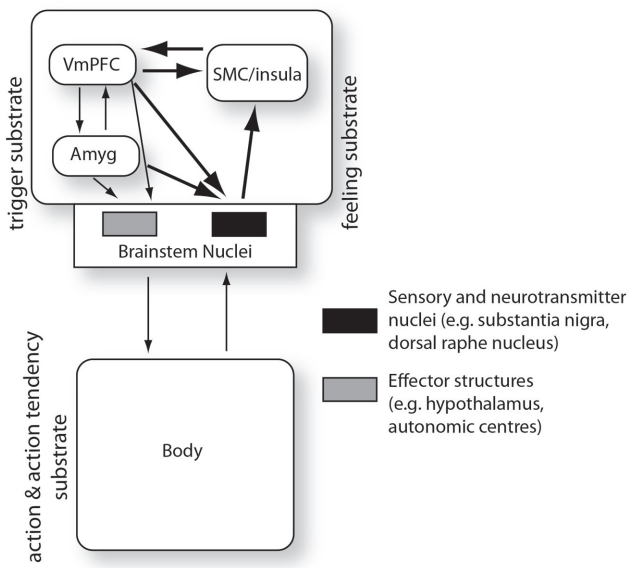


FIGURE 4 | Brain–body prediction–feedback loops: adaptation of Damasio's brain–body loop theory of emotion and emotional feeling. The "as-if body loop" of Damasio is superimposed (thick arrows) on the "body loop." The diagram can be seen to encapsulate at least two nested prediction–feedback regulatory loops implemented by VmPFC and Amyg projections: firstly, the brain-stem nuclei are provided a coarse predictive representation of body state; secondly, the SMC/insula is provided a more contextual predictive representation of body state as it relates to exteroceptive and proprioceptive late stages of processing in relation to the emotionally competent stimulus. Abbrev: VmPFC (ventral-medial prefrontal cortex), Amyg (amygdala), SMC (somatosensory cortices). Adapted from Lowe and Ziemke (2010).

Emotion regulation – the function of ATPFL

Why should the ATPFL be of functional value? The straight answer is it affords efficient *emotion regulation*. The above-mentioned "As-if body loop" interpretation appears consistent with the exposition of Koole (2009) concerning the primary and secondary emotional responses the latter of which permitting up- or down-regulated emotional activity. Koole suggests: "People's primary emotional response presumably reflects their emotional sensitivity, whereas their secondary emotional response presumably reflects emotional regulation" (p. 7). In this case "sensitivity" can be interpreted as "prediction[11]," i.e., predisposition to emotional response based on past experience (or ontogenetically developed tendency) while the secondary regulatory component can be interpreted as "feedback" (see **Figure 5** for visualization of this up/down regulatory feedback perspective). From a computational perspective (neural field theoretic perspective – see last section), following a suprathreshold primary response this "prediction" might amount to an OR gate whereby PEs concerning the somatovisceral/action tendency representation will either amplify or destabilize (down-regulate) neural-dynamic activity. Where activation is subthreshold, the primary response might be better described in terms of an AND gate such that somatovisceral feedback, insofar as it matches primed activation sites on the field, may only induce suprathreshold activation. In the latter case, the uncertain (unlearned) situation requires bodily feedback to disambiguate the emotional context (cf. Damasio, 1994).

[11] According to a DFT neurocomputational account, however, "prediction" might only relate to suprathreshold activation. "Sensitivity" would perhaps better capture the possibility of DFT states being *either* sub- or suprathreshold.

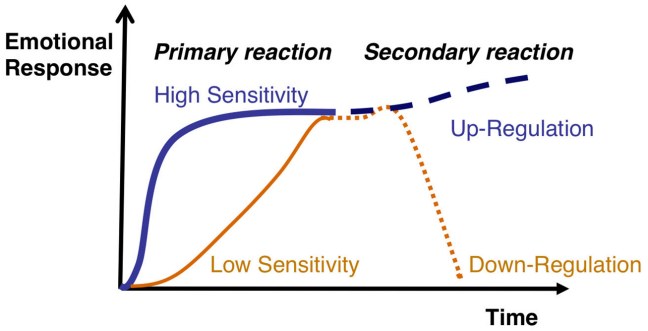


FIGURE 5 | Model of emotional sensitivity versus emotional regulation: taken from Koole (2009). The emotional episode may be viewed as a neural-dynamic representation (our argument) where the primary reaction serves as the prediction of the emotion state and may become stabilized depending on the strength of the prediction. Stabilization would thus occur following some parameterized suprathreshold activation and at such a point activation may impact on behavioral and cognitive systems over an emotion episode. Weaker predictions may not be stabilized. In either case, feedback from the secondary reaction may lead to down- or up-regulation (self-amplification, also see Lewis, 2005). Reprinted with permission.

Frijda (2004) has suggested that the value of emotional feeling is in its (social) behavioral and homeostatic predictive and regulatory effects. From the perspective of (social) behavioral predictive regulation, emotional feeling furnishes the organism with information concerning the social *acceptability* of a particular action. For example, aggression might provide short-term benefits concerning the weakening of a perceived competitor but at the cost of social respect and possible previously existing friendship. Down-regulating (dampening) emotional activity associated with inappropriate action may therefore be a useful option consequent to an ability to represent (feel) the emotion as action tendency. From the perspective of homeostatic regulation, Frijda points to the predictive importance of behavior *availability*. Behaviors that tend to be evoked by particular emotions, e.g., aggressive actions following anger, may just not be feasible to the organism according to physical and energetic resources. The competitor may be perceived as physically too strong, or the perceiving organism may be fatigued. Again, down-regulating the emotion or otherwise modulating the emotion may be appropriate in such circumstances. Frijda suggests: "[i]f there seems to be nothing one can do in a given emotional contingency, emotion tends to change – from fear or anger, to despair, for instance. Not seeing the possibility for meaningful action can deeply affect emotional motivation" (Frijda, 2004, p. 166). The opposite may also be true where emotions are "up-regulated" (augmented) "[b]y contrast, if actions are readily available, motivation may be enhanced, say, from irritation to outright rage."

The same arguments Frijda posits for being able to foresee possible future actions according to acceptability and availability also applies to predicting possible *action tendencies*: in terms of acceptability, action tendencies increase the likelihood of producing inappropriate behavior as well as expressing emotional states that may (socially) inappropriately belie the underlying activity; in terms of availability, readiness to action is metabolically costly, and energetic resources used on actions that may not be acceptable or available are lost that might otherwise be used serving the organism's "total set" of needs and concerns. Moreover, an emotion episode rooted in a tendency to act reduces the capacity and speed with which cognitive–behavioral programs – that concern more deliberative processing, e.g., planning, decision making, declarative knowledge construction – appropriate to the situation may be invoked. Koole (2009), with particular reference to negatively valenced emotions, also alludes to the above-mentioned points: "Negative emotional states are costly, because they mobilize a wide array of mental and physical resources within the individual [. . .] emotion regulation may thus be adaptive, by allowing individuals to conserve these resources by promoting a rapid return to hedonically agreeable states" (p. 15).

Allostatic regulation – ATPFL and resource mobilization

On the basis of the above discussion, regulation of emotion state occurring through ongoing prediction–feedback loops of *motivation-grounded* action tendency across an emotion episode seems apt. We suggest that the key neurophysiological substrate by which this may occur is the "as-if" body loop proposed and evidenced by Damasio and Bechara, utilizing phase-synchronized neural activity (representations) in zones of convergent proprioceptive, exteroceptive, and interoceptive processing routes (cf. Damasio, 2010). We suggested that, emotional feelings rooted in such a neural-dynamic representational substrate, provide the foundation on which ongoing prediction–feedback loops can operate.

Using the ATPFL position we may accommodate and even reconcile perspectives previously referred to in this article. For example, we can identify two PE (prediction error) scenarios requisite to ATPFL – (1) overestimation of action tendency, (2) underestimation of action tendency – that may precipitate "down-regulation" and "up-regulation" or "stabilization/maintenance," respectively (cf. Koole, 2009). In the case of (1) overestimation, following learning, a particular ECS could induce a pattern of activation in areas of the brain that enable representation of action tendencies (i.e., somatovisceral changes that are signaled by neuromodulatory activity in appetitive and defensive networks). The bodily response invoked by the trigger brain areas, however, will not exactly reproduce the predicted action tendency. It may be overestimated by the neural-dynamic representation according to *availability* of energetic or skeletomusculature resources – the organism may be fatigued through lack of sleep, nutrition, water, or intense physical activity. The mismatch (error) may then serve to down-regulate the action tendency. This will occur in the case of lack of resources and provoke the recruitment of other cognitive and behavioral programs following termination, or redirection (e.g., from one emotion state to a less costly emotion state – Frijda, 1986, 2007), of the emotional episode. In the case of (2) underestimation, the relationship between the organism and the ECS may not have been well learned and consequently the bodily response that is activated may be stronger than that which is anticipated in the neural representation. In this case, consistent with Damasio (cf. Bechara and Damasio, 2005), the body guides decision making where outcomes are uncertain or not well learned. Such activity may lead to a relative loss of (volitional) control (cf. Leventhal,

1980) where "the spontaneous motor system overrides the control of the voluntary system" (Leventhal, 1980, p. 169). Such error-triggered activation might be viewed in terms of a secondary emotional response (Koole, 2009) leading to a down-regulation of emotional activity in order to reassert volitional control.

Sterling's (2004) notion of allostasis might also be understood in terms of ATPFL, particularly concerning up-regulation of emotional responsivity. For example, suprathreshold neural representation of action tendency (predictions/feelings) can recruit metabolic resources in the service of emotional behavior. This would precipitate up-regulation at the secondary response constrained, nevertheless, by *availability* and *acceptability* concerns. The perceived availability of the action tendency is dependent on prediction–feedback loops but whilst Sterling's view suggests that basal homeostatic activity may be compromised according to an agent–environmental pressure to recruit metabolic resources to act, a relative lack of available resources (overestimation), or alternatively an unanticipated abundance of metabolic resources (underestimation), may serve to modulate the prediction (modify associations between *trigger* and *feeling* neural-dynamic patterns). This adaptation is schematized in **Figure 6** and is broadly consistent with the predictive coding perspective of Friston (2002; also see Kilner et al., 2007), i.e., that prediction minimization is a self-organizing process following reciprocal interactions between higher and lower processing levels where in the case of emotion the body provides the "lower level" proffering feedback to the "higher level" that is the brain.

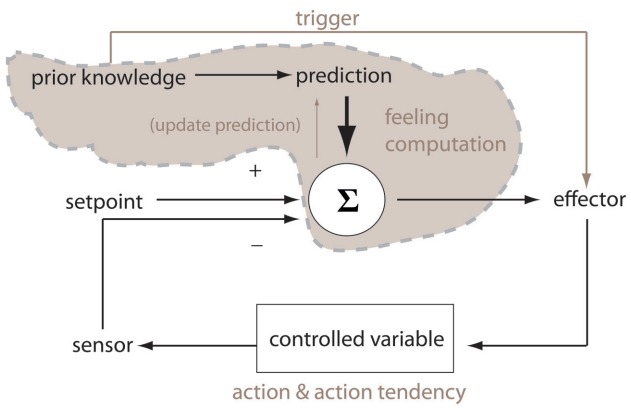


FIGURE 6 | Prediction–feedback control loop: adaptation of Sterling's control theoretic perspective of allostasis. The additional feedback arrow relating to "update prediction" highlights our perspective on the relation between emotional feeling and action tendency. All labels and arrows in gray are additions to the original diagram of Sterling (2004). The primary emotional response concerns a prediction (emotional feeling) that may override homeostatic set points – make less sensitive to negative error – in order that metabolic resources may be recruited, e.g., in an emergency. The "effector" in this case amounts to parts of the brain, e.g., hypothalamus, brain-stem, that instigate somatovisceral changes preparatory to action. The "controlled variable" pertains to the metabolic resources that are aroused/galvanized. The (desensitized) "setpoint" determines whether sufficient resources are available for the particular action tendency (e.g., an active response based on approach or withdrawal). At such a point a secondary emotional response ensues (regulation, cf. Koole, 2009) where up- or down-regulation of the action tendency occurs and prediction may be updated. For example, negative feedback would lead to down-regulation of emotional response and the prediction schema being updated such that set points are not so desensitized in the future. This, however, may also be overridden by higher (contextual) levels of processing. This control loop diagram can be mapped onto the Damasio neural-anatomy diagram in **Figure 4**.

The ATPFL is, therefore, best apprehended as an allostatic regulatory mechanism since neural patterns representative of anticipated body states also exert a top-down modulatory effect as in allostasis. This can also be inferred from Damasio (2010): "[t]he brain states, which correspond to certain mental states, cause particular body states to occur; body states are then mapped in the brain and incorporated into the ongoing mental states. A small alteration on the brain side of the system can have major consequences for the body states . . . likewise, a small change on the body side . . . can have a major effect on the mind once the change is mapped and perceived," (p. 96).

ATPFL IN GOAL-DIRECTED BEHAVIOR: AN APPLICATION

In the previous section, the notion of emotional feeling as a predictive mechanism in a dynamically realized control system, an ATPFL, was proposed. It was suggested that being able to anticipate one's future bodily changes that are preparatory to action might circumvent:

1. *Increased likelihood of triggering inappropriate behaviors and expressions*,
2. *Misuse of resources* – to the detriment of:
   a. Basic homeostatic maintenance, and . . .
   b. Time and efficiency of processing according to invocation of cognitive and behavioral programs.

These two aspects were related to Frijda's notions of *acceptability* and *availability*, respectively, as they concern, according to his perspective, emotional feeling as prediction of *action outcome*. We then suggested that this perspective could be adapted in order to posit a functional role of feeling in the context of predicting *action tendency*.

In order to make clearer still the importance of the two above points to organismic *adaptivity and viability* (functionality), we discuss emotions and emotional feelings in relation to point 2. but with respect to the specific case of emotion regulation of multiple needs or goals and with respect to both biological and artificial (robotic) agents.

ATPFL AS A MECHANISM FOR MEDIATING AMONG MULTIPLE GOALS

Goal-directed behavior is viewed as one of three primary functions that is served by efficient emotion regulation according to Koole (2009) the others being "satisfaction of hedonic need[12]" and maintenance of personality integrity. The pertinence of goal-directed behavior in emotion regulation has been interpreted in terms of feedback, above all, in the service of learning, e.g., Baumeister et al. (2007) – also see Carver and Scheier (1990), and Carver (2003). In the view of Baumeister et al., emotions are motivating – organisms

[12] Emotions will tend to promote future likelihood of experiencing positive affective states and reduce the likelihood of experiencing future negative emotion (also see Baumeister et al., 2007).

seek to act to foster positive emotions and reduce negative emotions – and learning affords tailored predictions of contexts in which positive or negative emotions are likely. Frijda notes that trading off all the concerns (including goals) of the organism (homeostatic balancing) is a critical mediating influence in action selection: "[a]ction is the result of the cost-benefit balance over the consequences of the action for the total set of the individual's concerns" (2004, p. 164). Koole (2009) similarly notes this trade off: "Some of the functions of emotion regulation may extend beyond single goals. In particular, emotion regulation may allow people to balance multiple goals" (p. 16).

The term "goal-directed" has to be used with caution since it is considered often misrepresented. Frijda (1987) in his critique of the work of Oatley and Johnson-Laird (1987), for example, suggested that the authors' use of the term was inaccurate: "it is confusing when the word 'goal' is used for what we commonly designate by 'wish' or 'interest,' or 'concern"' (Frijda, 1987, p. 53/54). Essentially, goal-directed behavior refers to outcome expectation based on plans arrived at through deliberative processing (cf. Daw et al., 2005; Frijda, 2010). This is to be distinguished from *habitual activity* – largely stimulus-driven behavior that, unlike goal-directed behavior, is relatively inflexible following changes in reinforcement outcomes (cf. Daw et al., 2005). Goal-directed behavior is also to be distinguished from *impulsive activity* (Frijda, 2010) which: (1) comprises automatic emotional responses that are unconscious, (2) occurs at an early (and possibly premature) contextual processing stage (e.g., responding aggressively to insult to someone that is bigger than you), (3) entails urges to act on the expectancy of gain following behavior completion. For Frijda (2010), habits and impulses pertain to stimulus-driven "aims" whereas goal-directed behavior starts with the goal in mind and requires deliberation as to how to achieve this (somewhat) irrespective of stimulus presence.

Many researchers consider emotion functionality in terms of states elicited following interrupts on goal-directed behavior (Simon, 1967; Toda, 1982; Oatley and Johnson-Laird, 1987; Rolls, 1999; Kreibig et al., 2010). Such "goals," however, may be interpreted as being alternatively based on deliberated action plans *or* motivated states regarding present needs divorced from detailed action schemata. Rolls (1986, 1999, 2000, 2005) has adopted a reinforcement learning perspective on emotions and their role in goal (or perhaps "need")-directed behavior. He suggests that emotions can be defined as "states elicited by rewards and punishments, including changes in rewards and punishments" (2000, p. 178). For Rolls, different emotions are elicited by different primary and secondary reinforcers. The specific reinforcement *contingency* (RC) also determines the particular triggered emotion. Anger, for example, is a state elicited by the omission of a previously expected positive reinforcer; fear pertains to the presence of a negative reinforcer; happiness relates to the presence of a positive reinforcer (see **Figure 8** – left-side – for visualization of the dimensional structure of Rolls' perspective). The emotion that is triggered by the RC may thereafter "help to produce *persistent and continuing motivation and direction of behavior*, to help achieve a goal or goals," (Rolls, 2000, p. 181, authors' italics). This perspective might also be extended to incorporate more fully the notion of *prediction*. As mentioned in Section "Prediction in the Brain," Schultz (1998, 2007) made pioneering discoveries on the role of dopamine as a prediction and learning signal that enables organisms to *temporally* associate (primary and secondary) reinforcers. Anger, on this basis, may be seen as a state elicited by the omission of a positive reinforcer predicted to occur at a certain point in time, i.e., feedback provides a negative/omission error. Happiness and fear, on the other hand, are states elicited by positive and negative reinforcers, respectively, according to time-sensitive learned expectations.

In "classical" AI (or perhaps "enlightened" AI), the relevance of emotions to goal/need-directed behavior has been noted. Simon (1967) likened central nervous system activity to a serial information processor where emotions have two essential functions: (1) as "goal"-terminating mechanisms, (2) as interruption mechanisms. These mechanisms were considered to allow organisms to achieve a number of goals whilst simultaneously meeting urgent needs in real-time. More recently, Cañamero (2003), taking inspiration above all from Frijda and Damasio, has proposed that emotions offer important means for action selection where homeostatic regulation of fulfillment of multiple needs is considered critical to the long-term viability of the (robotic) agent. This follows the thinking of McFarland and Spier (1997; McFarland and Bösser, 1993; McFarland, 2008) who have advocated the need for robots to react to real-time environmental opportunities (*opportunism*) when considering homeostatic regulation of multiple internal needs and goal/need directives. Avila-Garcìa (2004), Avila-Garcìa and Cañamero (2005) have suggested that emotion-relevant synthetic hormones allow robotic agents to trade off the need for opportunism with the need to *persist* in the pursuit of a particular need-fulfilling goal. More recently, work by Lowe et al. (2010a), Montebelli et al. (2010) has considered the importance of different types of energy constraints to the homeostatic regulation of adaptive behavior of robotic agents with multiple goals. Lowe et al. (2008) allude to the fact that agents' goals may require different metabolic/physiological resources in order to be successfully carried out and such resources must also be distributed appropriately to those effector systems that are prioritized during the particular behavior. This is true of the *elaborated* action tendencies that are seen to underlie emotions. For example, as Ekman (2003) points out, when we are angry we tend to feel a rush of blood in our upper arms and torso, presumably preparing us for an aggressive response (approach tendency) appropriate, for example, to neutralizing the obstacle to our present goal(s). In the case of fear, on the other hand, we feel a sensation of coldness in our upper body as blood drains away from the upper body areas implicated in aggressive responses so as to provide energetic resources to our legs priming a potential flight response.

On the basis of the above, an estimate of energy costs to carrying out particular behaviors, or to galvanize action tendencies, pertains not only to the goal in mind or available need but must account for all goals relevant to agent viability and well-being. If a particular goal is too costly to achieve in respect to the goal set, it may not serve the agent to persist in the goal/need-directed activity. We propose that ATPFL allows for mediation among goal-directed behaviors in the sense that predictions of action tendencies may be down-regulated if goal/aim-directed behaviors are not

considered available regarding energy costs *with respect to* the total goal set.

How do goal/need junctures or reinforcement contingencies, and the emotional states that they provoke, inform agents how to act? Oatley and Johnson-Laird (1987) consider emotional activity as guiding actions permitting transitioning between goal/need-based states. In their "cognitive theory of emotion," Oatley and Johnson-Laird list five of the basic emotions (they do not consider "surprise" as an emotion) for which goal/need-based behavior may be modulated. The emotion states, the conditions of their elicitation, and the goal/aim-(re)directed behavior they promote are listed in **Table 1**.

Similar to Damasio (1994), Oatley and Johnson-Laird (1987) suggest that emotions can limit a search subspace and circumvent problems inherent in traditional Artificial Intelligence (AI) planning programs. Emotional states are important for generating modes whereby a suite of goals, action possibilities, and skills may be invoked. Frijda (1987), in his *emotions as action tendencies* perspective, in a direct response to Oatley and Johnson-Laird's paper, largely concurred with the authors' "goal"-directed perspective on emotion whereby both event-context and resource competition constrain action selection in the service of a particular goal:

> "[e]motions, one can say, clamour for control precedence for the actions that they motivate, or for the abandonment of actions or plans under way . . . [c]ontrol precedence means claiming exclusive access to available resources for the goal under execution, or readiness to interrupt work on the goal under execution on behalf of the precedent one," (p. 52).

However, Frijda indicated that the presumed innate predisposition to perceive goal junctures (GJs; as emotion triggers) and associations between them and transition states (effective detailed action plans) was at odds with existing research that "could find the elementary qualities of pleasure and pain, and no more" (Frijda, 1987, p. 52). Frijda instead posits an appraisal theoretic angle according to two phases of emotion elicitation: "The first phase is assessment of the fact that a goal is achieved or promises to be so, or on the contrary is threatened or has failed; I call this 'relevance appraisal.' The second phase is assessment of the particular type of juncture at which this happens; I call this 'context appraisal'" (Frijda, 1987, p. 53). This description implies a secondary deliberative processing phase and can be likened to the Koole (2009) and Kuhl (2008) notion of emotional responses consisting of primary (sensitive) and secondary (regulatory) stages, as discussed in Section "Emotional Feelings: Action Tendency Prediction–Feedback Loops." Reconciling these two views we can say that the second phase/stage of the emotional response concerns a deliberation of *availability* and *acceptability* (see Emotional Feelings: Action Tendency Prediction-Feedback Loops) which induces up- or down-regulated emotion. This deliberation must necessarily account for the multiple goals that the agent is required to mediate among. A key output of a neural-dynamic representation (prediction) of action tendency has been suggested to be with regard to the biasing of available behaviors in appetitive and defensive networks.

On the basis of the above, a full emotion episode may require the presence of deliberative processing. It is, however, conceivable that a secondary emotional response could be induced simply by delayed bodily feedback and therefore deliberation may concern Frijda's *acceptability*, but not *availability*, estimations. However, the computation concerning *availability* must also take into account whether or not carrying out the particular behavior is energetically tenable not just for the present goal/need-directed behavior but also with respect to the need to utilize resources for other goals/needs, i.e., the total goal/need set. On the other hand, it is not clear that deliberation is always necessary for a *primary* emotion response to occur (e.g., see LeDoux, 1996). Rather, in the spirit of Oatley and Johnson-Laird (1987), this may merely necessitate a particular sensitivity to reinforcement contingencies particularly if learning may be simplified to (conjunctions and negations of) phasic signals emanating from appetitive and defensive networks[13]. Such an emotional system need not induce more or less hard-wired action transitions but rather bias sub-trees of action tendencies within appetitive and defensive networks. Such a perspective amounts to a compromise between the *discrete action program* and *motivation-grounded* perspective therefore. In reference to Section "What is An (Emotional) Action Tendency and What is its Relation to (Overt) Action?" we might envisage the emotion episode as governed by ATPFL according to **Figure 7**. In this case a "RC" or "GJ" triggers the initial emotional response as captured by a low-dimensional feeling neural-dynamic representational substrate – presented here in the form of pleasantness, arousal, dominance (approach–withdrawal) or "PAD" dimensions. The feeling state has a fundamental role in regulating the action tendencies it represents (or comes to represent). Following feedback from somatovisceral and event-context perception/appraisal PAD is updated and then induces a secondary, regulatory, emotional response. As referred to above, the deliberative processing phase ("acceptability" and "availability" feedback) preceding the secondary emotional response also includes assessment of how action tendencies impact on resource availability that

Table 1 | Oatley and Johnson-Laird's, 1987) list of basic emotions and their relevance to goal/need-directed behavior.

Emotion	Juncture of current plan	State to which transition occurs
Euphoric happiness	Sub goals being achieved	Continue with plan, modifying as necessary
Dysphoric sadness	Failure of major plan or loss of active goal	Do nothing/search for new plan
Anxiety	Self-preservation goal threatened	Stop, attend vigilantly to environment, and/or escape
Anger	Active plan frustrated	Try harder, and/or aggress
Disgust	Gustatory goal violated	Reject substance and/or withdraw

[13]Goal junctures as captured by such signals in low-dimensional, e.g., PAD, space also appears tenable when considering the work of Krieglmeyer et al. (2010) who suggest that associative links between valence and *approach–avoidance* (i.e., a not classically core affective dimension) do not require appraisal but may be automatic and therefore appear grounded upon motivational (appetitive–defensive) systems.

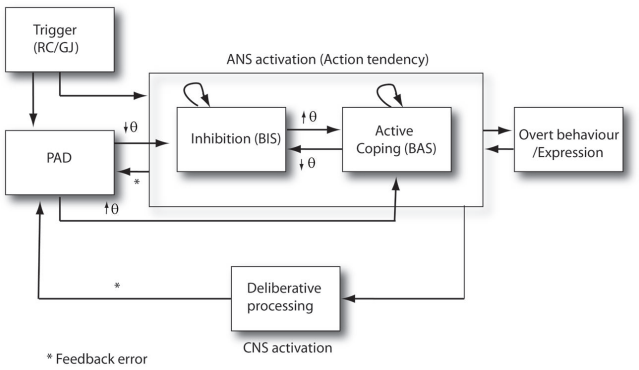


FIGURE 7 | Schema of emotion regulation of goal-directed behavior. Following **Figure 3**, PAD (pleasantness, arousal, dominance) has a key regulatory effect on action tendency and is a fundamental component of the full emotion episode. Feedback, in the case of suprathreshold activity may produce *acceptability/availability* mismatch errors, or in the case of subthreshold activity, galvanize neural activation that comes to represent the action tendency. See main text for details.

affects the goal/need set. Deliberation may be particularly relevant for mediating among goals (in mind) rather than needs (more stimulus-driven) where, in the case of the former, energetically costly behavioral persistence may be required in order to obtain any value from the sequences of steps that lead to the ultimate goal.

The emotion episode in the context of goal-directed behavior, as depicted in **Figure 7**, can be described sequentially as follows:

1. Trigger: an emotional stimulus (ECS) in the context of a RC/GJ is perceived;
2. Primary emotion response (sensitivity/priming): changes in emotional feeling representational space occur simultaneously in neural-dynamic representational regions in the brain (e.g., in somatosensory cortices as captured by PAD dimensional space) – and in the ANS via trigger cites in the brain (e.g., amygdala, hypothalamus, brain-stem);
3. Secondary emotion response (regulation): bodily and cognitive–behavioral feedback which may be more or less automatic or deliberative (evaluation/appraisal of action tendency). This ensues as a function of certainty of outcomes concerning:
   a. Availability: internal resources that impact on not just individual goals but the entire goal/need "set,"
   b. Acceptability: (social) event/GJ, e.g., actual or relative reward or punishment,
4. Strategic/tactical action selection (cf. Cacioppo et al., 2000): goal-persistence or goal-abandonment tendencies and actions may be affectively or emotionally marked – PAD will regulate a persistent bias of appetitive–defensive networks and specific branches of the search space. This occurs:
   a. After 2. when suprathreshold but may be up- or down-regulated
   b. After 3. when subthreshold, i.e., following context (including somatovisceral) disambiguation,
5. Overt action selection: active coping strategy (BAS – see McNaughton and Gray, 2000 and see Section What is An (Emotional) Action Tendency and What is its Relation to (Overt) Action), inhibition/attention–orientation (BIS);
6. Feedback from behavior will modulate all of the above over the emotion episode.

In the next sub-section, we will describe in more detail the means by which inhibition and active coping might be emotionally regulated according to goal contexts.

ATPFL: INHIBITION AND ACTIVE COPING AT GOAL JUNCTURES

The view of emotion regulation of goal-directed behavior that we propose is somewhat complementary to that of Baumeister et al. (2007) that concerns anticipation and feedback. However, Baumeister et al. (2007) suggest that emotions primarily function as *feedback* systems that promote the elicitation of behaviors that *anticipate* future emotional states. For these researchers, "automatic affective responses" guide online behavior, while feedback (supporting an emotionally reinforced learning) allows for the updating of such affective responses according to a priming of, above all, cognitive systems for context evaluation. Here, something like core affect (pleasantness, arousal) is of adaptive guidance to online behavior and directly triggered by relevant stimuli (ECSs) but full emotions are, for the most part, dysfunctional to behavior in Baumeister et al.'s view and instead serve only for learning purposes (feedback). To illustrate the comparison, we refer to Baumeister et al.'s perspective as exemplified in the context of aging:

> "[f]indings of aging also seem to support the feedback theory of emotion rather than direct causation. Carstensen et al. (1999) have proposed that as people get older, they shift from emphasizing acquiring knowledge toward emphasizing regulating emotion. Carstensen et al.'s broader assumption is that the value of acquiring knowledge is inversely proportional to the time one has left in life, and so as the person begins to recognize that time is growing shorter, he or she will downplay that goal," (p. 180).

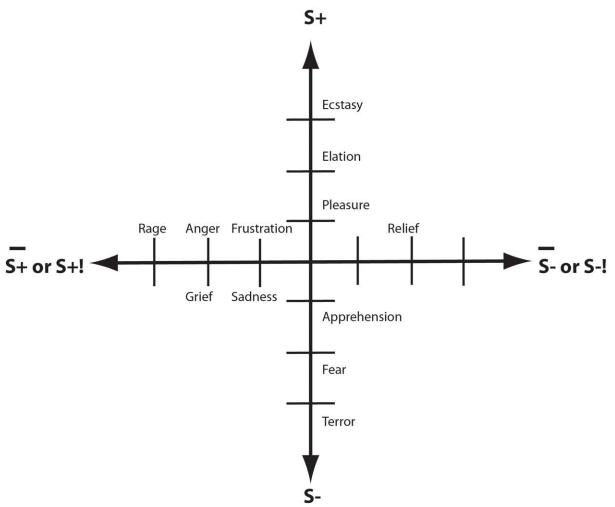
Alternatively, or complementarily, as individuals/organisms age, they tend to have less metabolic/physiological resources to expend on emotions and emotional overt behavior. A tendency to be less emotional (to down-regulate at the secondary stage of the emotional response, cf. Koole, 2009) as one ages, therefore, may be explained according to *overestimation* of action tendency following the initial representation of an emotional contingency in the service of goal/need-directed behavior. On the one hand, therefore, aging organisms have more knowledge to draw upon regarding how to respond behaviorally to junctures in goals, and on the other hand, they have less resources available to produce emotion states (rooted in action tendency changes) that would promote flexible online use of cognitive and behavioral programs as well as learning from feedback of the enduring emotional state. As the organism ages, it is also less likely to *underestimate* its emotional activity rooted in action tendency as availability of metabolic/physiological resources are less likely to overwhelm learned predictive and represented states. Aging, on this basis, can be seen as a process of

moving gradually from up-regulation of emotion states to down-regulation of emotion states with respect to goal-directed behavior. Essentially, an *active coping* response is less required as the individual ages. In general, efficient full emotion guided behavior requires the learning – through ATPFL – of efficient emotion regulation.

Recent research by Boureau and Dayan (2010) provides further context within which we might view emotions according to reinforcement contingencies or GJs. In relation to the phasic neuromodulatory signals of dopamine (DA) and serotonin (5HT) that appear to be central to propelling learning and action, Boureau and Dayan note that contrary to popular understanding DA neurons phasically respond in some aversive/defensive contexts while 5HT neurons may phasically respond in appetitive contexts. The researchers produced a dimensional model of DA and 5HT influence in relation to appetitive and aversive contexts, on the one hand, and to active and inhibitory contexts on the other. They suggested that DA neurons fire in the context of appetitive expectation (a positive valence signal) but also with respect to action tendency (invigoration). In the latter case, the invigorated response can be seen as indicative of an expected relative reward in the sense that through action, punishment is expected to be avoided. 5HT, on the other hand, is observed where punishment is expected but also where inhibition of ongoing behavior occurs. Since the latter case can also occur in an appetitive context (expected omission of reward), to make consistent with the popular understanding of 5HT function the appetitive–inhibitory context, signaling is considered indicative of expectation of relative punishment, i.e., failure to achieve reward. This failure might be interpreted as occurring either as a consequence of continuing a behavior where inhibition is appropriate (Boureau and Dayan, 2010), e.g., in a social context, but it may also conceivably occur in the absence of finding an alternative active coping response.

The junctures presented by Boureau and Dayan (2010) concern: appetitive reward expectation; an aversive punishment expectation; omission of expected reward; omission of expected punishment. As such, these junctures can be compared to Rolls' dimensional approach describing emotion elicitation at reinforcement contingencies (see **Figure 8**). Boureau and Dayan do not refer to emotion elicitation but we suggest that expected relative or actual punishment inducing an inhibition of ongoing behavior might precipitate an active response phase that is galvanized by an emotional action tendency. Consistent with Lang and Bradley (2010) an emotion episode may, in the context of motivated behavior, be characterized by an inhibition stage (step 1 in **Figure 8** – right; activating the BIS system, McNaughton and Gray, 2000) where uncertainty resides concerning the appropriate action tendency or emotional response (a prediction is not fully formed according to a neural-dynamic representation based on ATPFL). Following a stage of more or less deliberative processing (Step 2 in **Figure 8** – right; somatovisceral and environmental contextual disambiguation), where up-regulation occurs (emotion regulation, Koole, 2009), an appropriate action response may be initialized permitting the agent to move into an active coping phase leading to expected actual or relative reward. Down-regulation on the other hand, following deliberation, may lead to continued inhibition or less active coping responses, e.g., calling for help. As a simplification, a magnitude negation of DA and 5HT might inform of the appropriate response orientation in this case and, following learning, be of *statistical predictive value*, i.e., noting that the mapping between approach–avoidance to appetitive–defensive networks (behavioral possibilities) is not entirely unambiguous (again see Frijda, 2010).

This ATPFL interpretation of Boureau and Dayan's (2010) model according to active coping provides an elaboration of the



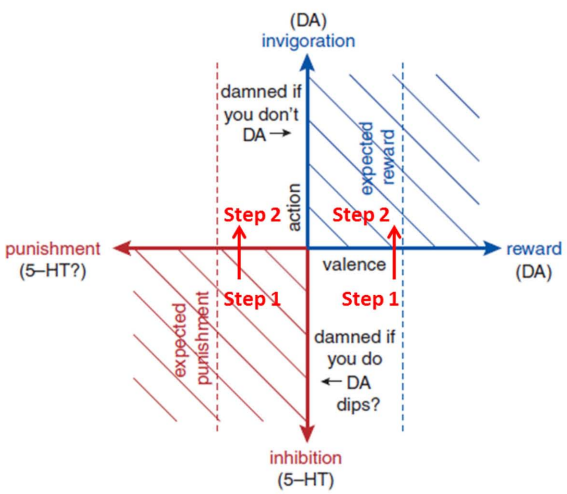


FIGURE 8 | Reinforcement–contingent emotional dimensional models. Left: Rolls' (1999) dimensional model of emotion-based on omission (! | –) or fulfillment (+ | –) of reinforcement. Reprinted with permission. Right: adaptation of Boureau and Dayan (2010) model (step 1 and step 2, and red arrows denoting step transitions have been added). The Rolls model can be mapped onto the Boureau and Dayan model where activation spans outward from the origin (center) and cuts across the diagonals of the four quadrants in the Boureau and Dayan model. In this manner, the upper right quadrant provides the S+ dimension, the lower left quadrant the S− dimension, the upper right quadrant the S− omission/termination dimension, the bottom right quadrant the S+ omission/termination dimension. Reprinted with permission.

Oatley and Johnson-Laird (1987) explanation. Emotion regulation provides insight into the dynamics of behavior that may occur according to GJs as a function of comparison of expected (relative and actual) rewards and punishments. This offers an, above all, *motivation-grounded* example of the facility of the ATPFL whilst simultaneously proffering a tentative bridge to discrete *action program perspectives* of (basic) emotions.

CONCLUSION

In this article we have gone through three main stages of enquiry:

1. what is the considered view of the relationship between emotional feeling and action tendency?
2. can the considered view be understood according to a perspective that likens emotional processing to other types of processing in the brain, i.e., one that involves prediction and feedback enabled by the mechanism of neural-dynamic representation?
3. how can this new perspective be understood with respect to a purported key function of emotion: regulating goal/need-directed behavior?

In relation to 1., we distinguished between *discrete action program* and *motivation-grounded* theorists of emotions who have investigated the relation between emotional feeling and somatovisceral changes constitutive of action program or tendency, respectively. This coarse division was made to render more explicable existing controversies in the field. We outlined in 2. the notion that emotional feeling is functionally critical (insofar as it reliably maps to neural-dynamic stable representations) as it provides an operational foundation for "action tendency prediction-feedback loop(s)" which we abbreviate to ATPFL. In relation to the perspective elucidated in point 2., however, emotional activity as it pertains to action tendencies tracked by interoceptive (and proprioceptive) neural-dynamic representational processes may operate differently to sensorimotor modes of processing. Other senses in being focused on the outside world are less subject to the messy and latent dynamic effects of ongoing bodily changes to emotion evoking stimuli. Nevertheless, we have suggested that, using mechanisms not dissimilar to sensory and motoric perceptual systems in the brain, i.e., comprising prediction and feedback, emotional feelings might best be understood as predictive mechanisms for regulating action tendencies. This perspective has been put forward on the basis of neural–anatomic and computational/functional feasibility and can also be seen as an extension to other perspectives that implicitly or explicitly attribute a predictive or/and regulatory role to emotion (e.g., Leventhal, 1980; Rolls, 1999; Frijda, 2007; Laird, 2007; Koole, 2009; Damasio, 2010). Mechanistically, it is argued that the perspective posited requires a neural-dynamic representational substrate upon which predictions and feedback can be compared over the varying brain and bodily time scales inherent in the emotion episode, as it (the emotion), for example, tracks and organizes, or recalibrates, a goal/need-directed behavior. The top-down/bottom-up predictive–feedback loops thereby permitted offer a means by which energetic resources may be appropriately allocated among the constituents of a goal/need-directed behavior set and also guards against the elicitation of inappropriate behaviors and expressions. In relation to the ATPFL explicated and exemplified in points 2 and 3., respectively, advocates of the (*motivation-grounded*) view of emotion might suggest that positing the existence of more than two dimensions represented in feeling neural-dynamic substrates is not parsimonious given existing evidence – that more unequivocally argues for the dimensions of valence (pleasantness) and arousal. However, the existence of a single dimension additional to the core affect perspective (embraced by, e.g., Russell, 1980, 2003; Frijda, 1986, 2010; Cacioppo et al., 1992, 2000; Baumeister et al., 2007) concerning approach–withdrawal tendency or dominance: (1) has received strong evidential support where existing measures to track it may be unreliable (e.g., Russell and Mehrabian, 1977; Davidson, 1993; Mehrabian, 1996; Mauss and Robinson, 2009), (2) may still be considered to offer a low-dimensional ANS-specification of emotion broadly consistent with the *motivation-grounded* perspective (rooted in core affect; e.g., see Krieglmeyer et al., 2010), and (3) may be conceived according to core learning and behavior guidance signals in appetitive and defensive networks (cf. Boureau and Dayan, 2010). Furthermore, importantly, a *core affect* + perspective affords a bridge between dimension theory and basic emotion theory (as also usefully studied by Christie and Friedman, 2004) which may be necessary to furthering the theoretically dense field of emotion science.

The present article began with an allusion to how the proposed dynamic interdependence of feeling–action tendency reflects a relative inseparability between cognition and emotion – emotion as a form of action tendency regulation involves prediction, memory, representation and, in some cases, planning, i.e., anticipation over longer-time scales or anticipation of other emotion episodes[14]. In this sense, emotional activity bears the hallmarks of classically conceived cognitive processes. It was also alluded to, however, in Section "The Relationship Between Emotional Feeling and Action Tendency: A Review," that any full-fledged theory of emotion should account for the role of *triggers, action and action tendencies,* and *feelings.* In this article, on the other hand, we have focused on the latter two emotional phenomena. In the previous section, however, we made reference to reinforcement and goal-directed behavior contingencies (e.g., Oatley and Johnson-Laird, 1987; Rolls, 1999; Boureau and Dayan, 2010) as effective emotion triggers which need not, in all circumstances, be the result of deliberative or cognitive appraisal processes. We suggested that the use of conjunctions and negations of the neuromodulator learning/action selection signals key to TD computations in appetitive and defensive (i.e., motivational) systems may encode goal/need contextual information critical to triggering appropriate active coping responses. However, the exact means by which emotion elicitation occurs may involve complex ontogenetic development of cognitive and behavioral programs. We intuit that the relation between stimuli and instrumental approach–avoidance activity as mediated by core affective components (Russell, 1980), are indeed central to such development. However, it has not been the aim of this paper to attempt to address the issue of emotional

[14]Though the cognitive–behavioral programs to which we allude and that follow stable feeling states may be considered in terms of more deliberative, e.g., linguistic/declarative, forms of processing that are somewhat underdetermined, though not independent from, the emotional feelings.

development, though clearly an important area. Rather, as a first step to better understanding emotions, their relation to cognition and their involvement in goal-directed behavior, it is suggested that a clearer elucidation of the relationship between *feelings* (both conscious and unconscious) and *actions and action tendencies* in emotion regulation is required. This article has described a perspective that makes tentative steps in this direction. Emotional feeling states and their constituents are notoriously difficult to track using standard neuroscientific and psychological methods. As Scherer remarks regarding feeling states: "[s]o far, we have little hope of getting even close to measuring the processes represented by (non-communicated feelings)" (2004, p. 139). Postulating global principles of brain–body functioning may help researchers in emotion science to see the wood for the trees. By framing the issue of emotional–cognitive activity according to (1) predictive regulation, (2) goal-directed behavior, (3) use of artificial agents, and suitable mechanisms for studying emotional constituents (e.g., machine learning and DFT), it may be possible to arrive at a computationally tractable means of understanding how emotions *are*, or how they *could be*, with respect to organisms that have multiple needs and goals and that are required to exist in the real world.

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The influence of emotion on cognitive control: relevance for development and adolescent psychopathology

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The last decade has witnessed an explosion of research into the neural mechanisms underlying emotion processing on the one hand, and cognitive control and executive function on the other hand. More recently, studies have begun to directly examine how concurrent emotion processing influences cognitive control performance but many questions remain currently unresolved. Interestingly, parallel to investigations in healthy adults, research in developmental cognitive neuroscience and developmental affective disorders has provided some intriguing findings that complement the adult literature. This review provides an overview of current research on cognitive control and emotion interactions. It integrates parallel lines of research in adulthood and development and will draw on several lines of evidence ranging from behavioral, neurophysiological, and neuroimaging work in healthy adults and extend these to work in pediatric development and patients with affective disorders. Particular emphasis is given to studies that provide information on the neurobiological underpinnings of emotional and cognitive control processes using functional magnetic resonance imaging. The findings are then summarized and discussed in relation to neurochemical processes and the dopamine hypothesis of prefrontal cortical function. Finally, open areas of research for future study are identified and discussed within the context of cognitive control emotion interactions.

Keywords: review, emotion cognitive control interaction, development, anxiety, depression

BRIEF INTRODUCTION TO THEORIES OF EMOTION AND COGNITIVE CONTROL

Past research has investigated the influence of emotion on a variety of cognitive processes including basic visual (Pourtois et al., 2005; Brosch et al., 2008) and sensory processing (Moratti et al., 2006), memory (Banich et al., 2009), or attentional biases (Bar-Haim et al., 2007). A longstanding effort has been to understand the basic mechanisms of cognitive control, which indexes our ability to regulate and pursue goal-oriented behavior. A major motivation for this article, and this special issue, has been the assumption that control of such goal-driven behavior is especially required in the presence of emotionally evocative information. Surviving an encounter with a grizzly bear in the woods requires strong inhibitory control to overrule the initial tendency for a flight response, and remain calm and still instead. Thus, how does emotion influence cognitive control? Do all types of emotion, positive, and negative, have the same effect on regulatory processes? Does emotion enhance or impair cognitive control abilities and are these effects short-term or more sustained? The goal of this review is to examine these questions by reviewing studies from the emerging field of cognitive control emotion interactions and the influence of emotion on executive processes specifically. Due to the vastness of the field, this study will not examine the flipside of this approach, i.e., studies that merely concern the regulation of emotion *per se*.

Although much of this research has been conducted in adults, preliminary parallel research in child and adolescent development is emerging and has provided some intriguing findings. Therefore, a second goal of this review is to integrate these parallel lines of research and examine how experimental studies in adults can inform future directions for developmental cognitive neuroscience and *vice versa*.

To set the stage and provide a theoretical context within which cognitive control by emotion interactions can be evaluated, the article will begin with very brief introductions into the current state of cognitive control and emotion research. However, it must be noted that these introductions merely serve to provide brief overviews, and therefore, these sections cannot do justice to provide a detailed and balanced account of all theories. Then, the available evidence on cognitive control and emotion interactions will be surveyed, first in adults and then in development. In particular, this survey will examine to what extent emotional influence is common across diverse cognitive control abilities such as working memory, inhibitory control, or task switching and whether this influence is dependent on the type of emotional presentation such as prior mood induction, emotional distraction, or availability of reward. The review will end

Abbreviations: ACT, attentional control theory; AMY, amygdala; BD, bipolar disorder; dACC, dorsal anterior cingulate; dlPFC, dorsolateral prefrontal cortex; DMN, default mode network; FFA, fusiform face area; FG, fusiform gyrus; IFG, inferior frontal gyrus; IPL, inferior parietal lobule; IPS, inferior parietal sulcus; MFG, middle frontal gyrus; MNI, Montreal Neurological Institute; Nacc, nucleus accumbens; OCD, obsessive–compulsive disorder; OFC, orbito-frontal cortex; PPA, parahippocampal place area; PTSD, post-traumatic stress disorder; rACC, rostral anterior cingulate; SFG, superior frontal gyrus; SPC, superior parietal cortex; SPL, superior parietal lobule; vlPFC, ventrolateral prefrontal cortex.

with a discussion of open questions for future research and a brief summary[1].

THEORIES OF COGNITIVE CONTROL

Much advance in the field of cognitive control has been made over the last half century and several models at both the theoretical or neuroanatomical level have been proposed (Norman and Shallice, 1986; Fuster, 1997; Smith and Jonides, 1999; Braver and Cohen, 2000; Duncan and Owen, 2000; Stuss and Alexander, 2000; Miller and Cohen, 2001; Petrides, 2005; Banich, 2009). Although a definitive set of executive functions has not been agreed upon, many cognitive skills have been attributed to this category including planning (Koechlin et al., 2000), goal maintenance (Koechlin et al., 1999), task switching (Robbins, 1996; Brass and von Cramon, 2002; Dreher et al., 2002), response conflict, error monitoring, and decision uncertainty (Botvinick et al., 1999; Ridderinkhof et al., 2004), inhibitory control (Aron et al., 2007), and working memory (Wager and Smith, 2003). Through latent-variable analyses, it has been suggested that there are (at least) three different core executive processes, which comprise inhibitory control, the ability to shift (task) sets, and maintenance and updating of working memory (Miyake et al., 2000).

In any case, while most authors would now agree that the lateral prefrontal cortex (PFC) plays a critical role in executive function, models with regards to the specificity of this localization of function have not been without contention (Duncan and Owen, 2000; Stuss and Alexander, 2000; Petrides, 2005; Banich, 2009). While some authors ascribe specific executive functions to particular parts of the brain (Petrides et al., 1993; Goldman-Rakic, 1995), others propose a common network of the lateral PFC, which engages across a diverse set of cognitive demands including response conflict, task novelty, working memory delay, and perceptual difficulty (Duncan and Owen, 2000).

Given the variety of theoretical and neuroanatomical models the search for a unifying model of executive function continues. Some models identify particular executive processes with neuroanatomical locations, which are recruited in a sequence ("cascade") of events (Koechlin et al., 2003; Banich, 2009). For example, in the "cascade-of-control model" (Banich et al., 2009) top-down biases toward task-relevant processes are established in the posterior region of the dlPFC and passed on to the mid-dlPFC, which selects the most relevant out of the actively maintained task representations. In the next step of this cascade, the posterior portion of the dorsal ACC selects the appropriate response among the available response options and so on. By comparison, and in-line with other models (Botvinick et al., 1999; Ridderinkhof et al., 2004), the anterior dorsal ACC monitors and evaluates the responses and, in case of an occurring error, dACC signals back to the posterior dlPFC for greater control requiring re-initiation of certain steps of the cascade of events. The central tenet of such a cascade model is that cognitive operations are executed sequentially, and, if not accomplished adequately at a previous step, these operations require to be processed at a subsequent step. As the search for an integration of theory and neuroanatomy in cognitive control continues, the aim to specify the role of emotional processes during cognitive control will either facilitate (at best) or further complicate (at worst) this endeavor. The next section will provide a brief glance at current thoughts in emotion theory.

PROCESSING AND REGULATING EMOTIONS

Human feelings and emotions have long occupied the thoughts of scientists and philosophers alike. Several cognitive and non-cognitive theories of emotions have been proposed (cf. Dalgleish, 2004). Among the cognitive theories, appraisal theories (e.g., Arnold, 1960; Frijda, 1988; Lazarus, 1991; Scherer, 1999; Roseman and Smith, 2001) postulate that emotions are caused (elicited) by appraisals, i.e., subjective evaluations of occurring events. For instance, the final acceptance of an article in a journal could give rise to several emotions in the author including joy, pride, relief, or content. The precise emotion, however, will depend on the specific appraisal by that individual. By virtue of mediation between the event and the emotion, appraisal theories provide a multi-level approach, which allows for subtle variations between and within individuals as to which emotion will be elicited at which specific moment in time and at which specific situation. Within that framework, other authors propose degrees of motivation and emotional behavior that depends on the immediacy and severity of relevant change from the current state of events (e.g., Roseman and Smith, 2001). For example, the distance to the deadline for submission of said article may determine whether the author approaches the manuscript with "cold" but motivated and goal-driven behavior to finish the paper within the next month or may experience "hot" emotion and react with frozen shock and readiness to jump into action by the realization that the deadline is a mere 48 h away.

By contrast, biologically based, non-cognitive theories of emotion propose a direct relationship between the event, the emotion, and the physiological or neural state. Among the most well-known is the James–Lange theory (James, 1884; Lange, 1885), which proposes that emotions are mere experiences of the change of the bodily state. In such a case, the experience of the physical changes involved in fleeing from a threatening stimulus would be equated with the emotion of fear. Both sets of theories have led researchers to hypothesize different parts of the brain to be involved in the processing of emotion. These different neural foci include the amygdala (LeDoux, 2000), the septo-hippocampal system (SHS; Gray and McNaughton, 2000), the orbito-frontal cortex (OFC; Rolls and Grabenhorst, 2008), the ventromedial PFC (Damasio, 1996), or the brainstem circuitry (Panksepp, 1998). For example, in Damasio's (1996) "somatic marker hypothesis," the ventromedial PFC plays a strong role in processing those physiological changes of the body, which have previously been tagged as emotionally significant events. Other researchers have attributed a prominent role to the OFC in emotion processing, in particular as it pertains to motivational aspects of behavior *vis-a-vis* emotional learning of stimulus–reward associations (Rolls and Grabenhorst, 2008). That line of research assesses and classifies motivational valence of a stimulus as being positive and rewarding or negative and punishing. Yet another group of researchers highlight the amygdala as a central hub in the processing of fear (LeDoux, 2000). These investigators suggest two routes of emotional processing; a

[1]Note that despite historical differences in usage between the terms "executive function" and "cognitive control," both will be used interchangeably throughout the article to refer to all brain regions involved in such functioning.

fast, direct route from the thalamus to the amygdala, which can process crude information quickly and without awareness while a second, indirect and slow route uses a thalamo-cortical–amygdala pathway. This pathway allows a more fine-grained and conscious analysis of the stimulus. However, this distinction has recently come under criticism. Although Pessoa and Adolphs (2010) also suggest two central hubs, the amygdala and the pulvinar, they emphasize strong communication of these regions with broad cortical and subcortical regions. In addition, these authors suggest equally fast processing of affective and non-affective visual information.

Theories of emotion regulation have particularly focused on the dlPFC and the cingulate system and their role in modulation and controlling an emotional response (Bush et al., 2000; Ochsner and Gross, 2005). In particular, these theories distinguish between the dorsal ACC, which is involved in cognitive control, and the rostral ACC, which finds its role in regulating emotions (e.g., Bush et al., 2000). Parcellation of the ACC into a rostral and dorsal part, in turn, has come under scrutiny from recent reviews that suggest an integration of reinforcers and goal-directed behaviors within the "anterior mid-cingulate cortex (aMCC)" (Shackman et al., 2011).

Regardless of the type of brain system involved in affective processing, some investigators have pointed to a hemispheric divide when processing emotions. Following earlier authors (Mills, 1912), the work of Davidson (1995) suggests different specialization for specific emotion processing in the two hemispheres. In their theory, negative emotions are predominantly processed by the right hemisphere, while positive emotions are processed by the left hemisphere. However, this hypothesis has also been challenged by a meta analysis of functional imaging studies that points toward a more complex picture including regional specificity (Wager et al., 2003).With these conceptual distinctions in mind, the next section will review the available evidence of emotional influences cognitive control processes.

COGNITION BY EMOTION STUDIES IN ADULT VOLUNTEERS

FINDINGS IN HEALTHY ADULT VOLUNTEERS

Studies in neurologically healthy adults have used several methodologies and experimental paradigms including behavioral investigations, event-related potentials (ERP), repetitive transcranial magnetic stimulation (rTMS), and functional magnetic resonance imaging (fMRI), *n*-back tasks, recency probes task, flanker and stop-signal tasks, to task switching, delayed item working memory, go/no-go tasks, antisaccade, Simon, and Stroop tasks (**Table 1**).

Behavioral and psychophysiological evidence

Behavioral studies have provided the groundwork to establish the paradigms that can be used to demonstrate emotional impact on cognitive control processes. This line of research has shown consistent impairment of cognitive control during concurrent, task-irrelevant emotional processing. High emotional distracting stimuli, for example, impaired performance during task switching, inhibitory control tasks, working memory, or target detection tasks (**Table 1**). Traditional psychophysiological measures have provided valuable information regarding the neurobiological and neurophysiological processes underlying such interference effects. For instance, the startle response, i.e., an involuntary response to an unexpected and sudden stimulus, is closely linked to affective processing (Lang et al., 1990). Using this startle reflex during a standard flanker task, Hajcak and Foti (2008) reported that startle response magnitude was larger after errors than after correct responses. They suggested a close connection between the emotional regulatory system and cognitive control processes such as error monitoring. Other psychophysiological indices of control processes are also sensitive to emotional modulation. A larger fronto-central No-Go P3 amplitude has been reported in a positive relative to a negative context during a go/no-go task with incidental emotional stimuli in the background (Albert et al., 2010). Consistent with this finding, reaction times (RT) to Go trials were faster in the positive relative to the negative or neutral context. These data suggest that psychophysiological responses can peg the influence of defensive reflexes and positive context on cognitive control processes such as error monitoring and inhibition.

Evidence from fMRI

Functional neuroimaging studies have provided important information on the neural underpinnings of these effects, thus complementing psychophysiological findings. For example, Gray et al. (2002) induced a positive, negative, or neutral emotional state with short video clips before participants completed two different 3-back working memory tasks, one with verbal and one with facial stimuli. With verbal stimuli, the dorsolateral PFC [Brodmann area (BA) 9] was activated more for unpleasant emotional state relative to pleasant state, while with face stimuli, BOLD responses were increased for pleasant relative to unpleasant states. In another study negative but not positive or neutral mood resulted in increased error rates during a Simon task (Sommer et al., 2008). Concurrent with this behavioral finding, negative mood was associated with reductions in lateral PFC in incompatible when compared to compatible trials. While these data suggest that lateral PFC activation to working memory or conflict demands is sensitive to negative mood state, other studies have focused on the influence of attentional load on emotion processing (Pessoa et al., 2005; Lim et al., 2008; Bishop, 2009).

In a target detection task, letter arrays consisting of the same distracters (low attentional load) or different distracters (high attentional load) were displayed across emotionally valenced faces (Lim et al., 2008). When threat faces were compared to neutral faces in the low attentional load condition, increases in activation were apparent in several regions including the SPL, MFG, dACC, and FG. By contrast, this effect was absent during the high attentional load conditions. In particular, amygdala activation followed the same pattern as in the other regions suggesting vulnerability of this region to changes in top-down influences of cognitive load. In opposition to these findings is a study in which neutral or negatively valenced IAPS pictures were presented before a simple or a complex arithmetic problem (Van Dillen et al., 2009). Here, right amygdala activation was decreased during negative images during the complex task relative to the simple task. Similarly, the high but not low load negative condition led to increased activations in the same regions as in Lim et al. (2008), namely in the dlPFC and SPC. One possible difference between these two studies that might account for the discrepancy in findings is that emotional stimuli were present prior to the task in Van Dillen et al. (2009) while

Table 1 | Lists relevant studies in the field of cognitive control emotion interactions in healthy adults.

First author	Year	Sample size	Gender	Method	Cognitive control paradigm/task	Emotional/ motivational stimuli	Main point
Buhle and Wager	2010	24	m/f	beh	3 *n*-back letter WM task	Pain stimulation	↓ Pain during WM than during control condition; ↑ in heat reduced WM performance
De Houwer and Tibboel	2010	51	Females	beh	Go–no-go	IAPS neg, pos, neut	Pictures in high emotional valence impaired no-go trials
Dreisbach and Goschke	2004	18 (exp1) 32 (exp2) 17 (exp3)	m/f	beh	Task switching	IAPS positive, neutral, negative (exp3)	Positive affect ↓ perseveration but ↑ distractibility
Erthal et al.	2005	24 (exp1) 36 (exp2) 30 (exp3)	m/f m/f m	beh	Line orientation comparison with central emotional distractors	Emotional distraction (neutral/unpleasant IAPS + internet)	Emotional distraction higher during low than high cognitive load
Kensinger and Corkin	2003	46 (exp1) 41 (exp2) 25 (exp3) 30 (exp4) 36 (exp5)	m/f	beh	Several WM tasks	Eckman and Friesen + words	Significant interaction on *n*-back task for face but not word stimuli (exp5)
Krebs et al.	2010	20	m/f	beh	Stroop	Reward vs. no reward	Enhanced color naming during reward
Lavric et al.	2003	36	m/f	beh	*n*-back WM task (spatial vs. verbal)	Emotion induction via threat of shock	Performance on spatial but not verbal task was impaired during threat relative to safe
Legrain et al.	2011	10	m/f	beh	1 *n*-back or zero back color WM task	Pain stimulation	WM ↓ response prolongation seen in 0-back condition during pain vs. tactile stimulation
Levens and Phelps	2008	44 (exp1) 45 (exp2) 52 (exp3)	m/f	beh	WM (modified recency probes paradigm)	IAPS, ANEW	Trials with emot. stimuli ↓ interference relative to neut trials; (response) facilitation of interference resolution in WM
Pereira et al.	2006	23 (exp1) 30 (exp2) 23 (exp3) 22 (exp4)	m m m/f m/f	beh	Target detection task	IAPS (pleasant, unpleasant, neutral)	Consistent slowing of RT during unpleasant pictures while (blocked) presentation of pleasant stimuli resulted in RT improvement
Savine et al.	2010	26 (exp1) 30 (exp2)	m/f	beh	Task switching (exp1), delayed item recogn. WM (exp2)	Reward vs. no reward	Reward ↑ cognitive control
Zhou et al.	2011	20 (exp1) 24 (exp2) 12 (exp3) 20 (exp4)	m/f	beh	Task switching	NIMSTIM (fearful, neutral)	Fearful cues ↑ switch costs
Albert et al.	2010	30	m/f	ERP	Go–no-go task	IAPS neg, pos, neut distractors	No-go P3 amplitude was ↑ in positive context than negative context

Hajcak and Foti	2008	31	m/f	ERP + startle response	Arrow flanker task	–	Startle magnitude was ↑ after errors than after correct responses
Kanske and Kotz	2010a	25	m/f	ERP	Emotional flanker task	LANG	↑ Conflict-sensitive N200 during positive emotion
Leyman et al.	2009	18 (exp1) 22 (exp2)	Females	rTMS	Negative affective priming	KDEF	rTMS over right but not left dlPFC lead to changes in negative affective priming
Beck et al.	2010	31	m/f	fMRI	Delayed item recognition WM	Reward vs. no reward	Interaction between cognitive control and reward circuitry depending on type of reward (money vs. liquid)
Bishop et al.	2006	13	m/f	fMRI + genes	House-faces task	IAPS	Positive association of Val allele with vlPFC, OFC, PPA during negative face distractors
Blair et al.	2007	22	m/f	fMRI	Modified affective Stroop	Interspersed IAPS neg, neut, pos	Signif. interaction of task by emotion in right middle frontal gyrus: negative emotion (relative to neutral) increased incongruent vs. view trial
Boehler et al.	2011b	15	m/f	fMRI	Visual discrimination task (high vs. low demand)	–	Changes in substantia nigra during high demand trials
Boehler et al.	2011a	12	m/f	fMRI	Stop-signal task	–	Changes in substantia nigra during trial-to-trial adjustments
Compton et al.	2003	12	m/f	fMRI	(Blocked) emotional Stroop	Neg, neutral, color words	↑ dlPFC activation in both negative and incongruent relative to neutral
Dolcos and McCarthy	2006	18	Females	fMRI	Delayed WM (faces)	IAPS plus in house distractors	Emotional vs. neutral performance correlated in right vlPFC
Dolcos et al.	2006	15	Females	fMRI	Delayed WM (faces)	IAPS plus in house distractors	↑ Coupling of amygdala and IFG during processing of emotional distractors; these distractors also impaired WM performance
Dolcos et al.	2008	14	Females	fMRI	Delayed WM (faces)	IAPS plus in house distractors	Complex (confusable face) distractors ↑ dlPFC while simple (non-confusable, non-face) distractors ↓ dlPFC activation
Egner et al.	2008	22	m/f	fMRI	Emotional Stroop/conflict adaptation	Eckman and Friesen	lPFC resolved non-emotional conflict while rACC resolved emotional conflict but decreased AMY to emotional distractors
Etkin et al.	2006	19	m/f	fMRI	Emotional Stroop/conflict adaptation	Eckman and Friesen	Emotional conflict activates amygdala, PFC; emotional conflict resolution activates rACC
Goldstein et al.	2007	14	m/f	fMRI	Go–no-go task	Pos, neg, neutral words	Interaction of emotional valence and go vs. no-go in fronto-limbic (e.g., med OFC) regions
Gray et al. 2002 Gray and Braver 2002	2002	14	m/f	fMRI	3-Back WM task	Prior emotional induction (short videos)	Signif. interaction in dlPFC: ↑ BOLD for pleasant vs. unpleasant emotion during face stimuli but opposite pattern for word stimuli; performance correlates with fMRI signal
Hare et al.	2005	10	m/f	fMRI	Go–no-go task	NIMSTIM	Emotional valence as target or non-target moderate IFG and amygdalar responses
Herrington et al.	2005	20	m/f	fMRI	(Blocked) emotional Stroop	ANEW, neutral pleasant, unpleasant	↑ dlPFC activation to pleasant relative to unpleasant stimuli
Kanske and Kotz	2011a	20	m/f	fMRI	Emotional flanker task	LANG	vACC only active in neg (incongruent vs. congruent) but not in neutral (incongruent vs. congruent)
Kanske and Kotz	2011b	22	m/f	fMRI	Auditory emotional conflict	LANG	vACC activation during emotional conflict processing
Koch et al.	2007	40	m/f	fMRI	*n*-back WM	Neg. olfactory stimulation	Gender differences in cognitive control of emotion

(Continued)

Table 1 | Continued

First author	Year	Sample size	Gender	Method	Cognitive control paradigm/task	Emotional/ motivational stimuli	Main point
Krebs et al.	in press	14	m/f	fMRI	Target discrimination task	Reward vs. no reward	Interaction of reward and task difficulty in thalamus, caudate, substantia nigra, and mid-cingulate
Krebs et al.	2011	19	m/f	fMRI	Stroop	Reward s. no reward	Activations in dlPFC, superior and inferior parietal cortex, and fusiform gyrus reflect interactions between relevant and irrelevant reward during task
Levens and Phelps	2010	27	m/f	fMRI	WM (modified recency probes paradigm)	ANEW	Emotion interference resolution in IFG, OFC, anterior insula
Li et al.	2009	33	m/f	fMRI	Stop-signal task	–	"Risk taking trials" identified by RT distributions; during these trials "risk" was associated with activation in MFG, posterior cingulate, inferior parietal cortex
Lim et al.	2008	29	m/f	fMRI	Faces with superimposed letter arrays/high load and low load, selective conditioning to some faces preceded experiment	Eckman and Friesen + NIMSTIM	Emotion × attentional load interaction only during low but not high cognitive load in SPL, dACC, MFG, FG; even with highly salient faces
Mohanty et al.	2007	14	m/f	fMRI	Emotion word Stroop and color word Stroop	ANEW	dACC and rACC differ according to cognitive or emotion conflict
Ousdal et al.	2008	25	m/f	fMRI	Go–no-go task	–	↑ AMY activation to relevant vs. non-relevant stimuli; for both go and no-go stimuli
Padmala and Pessoa	2010	35	m/f	fMRI	Stop-signal task	Reward vs. no reward	↑ Activations in IFG and precentral gyrus during control relative to reward during successful vs. unsuccessful trials
Padmala and Pessoa	2010	54	m/f	fMRI	Response conflict task	Reward vs. no reward	Fronto-striatal–parietal changes in reward vs. no reward contrast during stimulus incongruency
Pereira et al.	2010	11	Males	fMRI	Target detection task	IAPS	Mid-cingulate cortex activation during presentation of unpleasant context with BOLD mirroring pattern of behavioral interference
Pochon et al.	2002	6	m/f	fMRI	*n*-back task	Reward vs. no reward	Common activations in lateral PFC and deactivation in ventral and medial PFC during reward and cognitive load conditions; no behavioral effect of reward
Polli et al.	2009	21	m/f	fMRI	Antisaccade	–	↑ AMY activation during erroneous vs. correct antisaccades
Savine and Braver	2010	16	m/f	fMRI	Task switching	Reward vs. no reward	Incentives ↑ cognitive control performance and moderate dlPFC, IPL, and dACC activation during task switching
Schulz et al.	2009	24	m/f	fMRI	Emotional go–no-go task	NIMSTIM	Activation to emotional faces during no-go vs. go in IFG, IPS, FFA, insula, AMY, mid-cingulate
Shafritz et al.	2006	13	m/f	fMRI	Emotional go–no-go task vs. letter go–no-go task	Eckman and Friesen	Emotional inhibition activated IFG/insula while inhibition during letter task did not activate such regions

Sommer et al.	2008	12	Males	fMRI	(Blocked) Simon task	Interspersed mood inducing IAPS (neg, pos, neut)	Negative relative to neutral and positive showed ↓ in incompatible vs. compatible in IFG, MFG, cingulate
Taylor et al.	2004	12	m/f	fMRI	WM task	Reward vs. no reward	↑ Impact of high reward on WM load than low reward in right dlPFC
Van Dillen et al.	2009	17	m/f	fMRI	Arithmetic problems	Interspersed IAPS	↓Limbic activation (amygdala/insula) to negative images for complex vs. simple task; reverse effect for dlPFC and parietal regions
Vollm et al.	2006	52	Males	fMRI + pharmac	Go–no-go task	–	↑ Activation in OFC, temporal pole, insula during go vs. no-go during mirtazapine (serotonergic) challenge
Whalen et al.	1998	9	m/f	fMRI	Emotional counting Stroop	Negative, neutral words	Task performance ↓ACC signal for negative and neutral rel. to fixation
Wittfoth et al.	2010	20	m/f	fMRI	Auditory decision task	Pos, neg, neut sentences	Valence-specific effects in left IFG, STG, caudate, thalamus
Yamasaki et al.	2002	10	m/f	fMRI	Shape discrimination task with emotional distractors	IAPS	↑ Bilat IFG activation to emotional vs. non-emotional distractors

Beh, behavior; m, male; f, female; ↑, increase; ↓, decrease; neg, negative; pos, positive; neut, neutral; WM, working memory; CPT, continuous performance task; WCST, Wisconsin Card Sorting Task; LANG, Leipzig Affective Norms for German (Kanske and Kotz, 2010b); KDEF, Karolinska Directed Emotional Faces (Lundqvist et al., 1998; Ekman and Friesen, 1976); IAPS, International Affective Picture System (Lang et al., 2008); NimStim (Tottenham et al., 2009); ANEW, Affective Norms for English Words (Bradley and Lang, 1999); Gur faces (Gur et al., 2002); ">", greater activation.

they occurred simultaneously in Lim's study. While the type of attentional task may also have played a significant role, these findings provide supportive evidence that attentional load moderates emotional processing.

Inhibitory control processes are also vulnerable to modulation of emotional valence. The pars opercularis and pars triangularis of the IFG evidenced reductions on no-go trials in response to happy but not sad or neutral faces (Schulz et al., 2009). An effect for sad (but not happy or neutral) stimuli, on the other hand, was observed in the posterior insula. Previous studies have highlighted different responding of neurocircuitry to different emotions (e.g., Vytal and Hamann, 2010). An interesting question related to this finding and within the context of emotion cognition interactions would be the sensitivity of distinct cognitive control regions to specific emotional valences. In other words, would happy or fearful emotion recruit the IFG but sad or angry emotion other areas?

If emotional valence exerts effects on inhibitory control in cognitive control workhorses such as the IFG, a pivotal question would be whether the reverse can also be observed, i.e., evidence of executive control processes in emotional hubs such as the amygdala. In their psychophysiological study, Hajcak and Foti (2008) had suggested a connection between defensive reflexes and error processing. Given the amygdala's role in the neurobiology of fear (LeDoux, 2000), an ideal proof-of-concept would be to demonstrate presence of such error processing in this region. Indeed, in an antisaccade task with strong non-emotional inhibitory control requirements, amygdala activation was increased during erroneous relative to correct antisaccades (Polli et al., 2009). Further corroborating evidence comes from other cognitive control studies void of an emotional context, which have examined variations in RT distributions. For example, in a stop-signal task, RT to "go" trials on trial (n) can either be faster or slower than RT on the previous trial ($n-1$). Li et al. (2009) argued that this variability in performance may originate from participant's anticipation of a looming "stop" trial. Thus, responding faster in the presence of a potential "stop" trial is "risky" and may lead to an error whilst a slower response may show "risk-aversion." Comparisons between individual fast and slow responses relative to the mean of all "go" trials in Li's study revealed heightened amygdala and vmPFC activations during fast "risky" trials relative to slow "non-risky" trials. Evidence of error processing or adaptive changes in trial-to-trial fluctuations during non-emotional tasks would support the idea of an involvement of regions traditionally associated with emotion in cognitive control. Intracranial recordings in patients undergoing invasive surgery complement these research lines. Two separate intracranial recording studies have documented modulation of subcortical structures such as the Nucleus Accumbens (Münte et al., 2008) or the amygdala (Pourtois et al., 2010) during error monitoring processes whilst performing flanker or go/no-go tasks. These data support the idea of involvement of regions traditionally associated with emotion in cognitive control.

From "hotspots" to "patterns of activation": evidence from functional connectivity

Recent trends in the cognitive neurosciences have witnessed a shift from a localization-oriented analysis approach to examining the pattern of activations between brain regions. Analysis of

such functional connectivity has offered intriguing insight into the influence of emotion on cognitive control from a brain pattern perspective. As is well-known, during the color naming condition of the Stroop task, cognitive interference is created by the actual meaning of the presented word relative to the font it is presented in (e.g., the word red presented in blue font). Using this principle, Banich and colleagues modified the Stroop task to present words that interfered at an emotional level (e.g., war) when participants were required to name the font color (Mohanty et al., 2007). They reported that during conflict trials, reactivity of both dorsal and rostral ACC predicted amygdala activation. By comparison, only dorsal ACC predicted dlPFC activation. A different team of researchers created emotional conflict by superimposing words of emotional states on congruent or incongruent emotional facial expressions (e.g., the word "happy" presented on a fearful face). Functional connectivity analyses during this task revealed a negative coupling between the rACC and the amygdala, which indicated a decrease in amygdala activation with an increase in rACC activity (Egner et al., 2008). In addition, the fact that lateral PFC was positively coupled with the fusiform face area (FFA) during non-emotional conflict resolution but negatively during emotional conflict resolution attributes a critical role of valence to connectivity patterns.

Impact of motivation on cognitive control

As noted in the introduction, some theories discriminate between emotional and motivational processes in emotion (Plutchik, 1962), while others argue that both processes are necessary to fully account for goal-driven behavior (Roseman, 2008). An intriguing question arising from these theories is to what extent influences of these two emotional systems on cognitive control abilities might be similar to each other. Studies in the category of motivational processing have examined the impact of reward (vs. no reward) on a variety of executive processes including the stop-signal task, the task switching paradigm, working memory, antisaccade performance, or the Stroop task (**Table 1**).

In an inhibitory control task, reward, by virtue of monetary incentive, interacted with stop-signal task performance in several brain regions (Padmala and Pessoa, 2010). Particularly when no incentive was provided, the left dlPFC responded more actively to successful vs. unsuccessful trials. This difference was reduced during the incentive condition. In stark contrast to these data are the results of another imaging study, in which monetary incentive increased the BOLD response in the left dlPFC during task switching (Savine and Braver, 2010). This response was also positively correlated with the incentive benefit of improved RTs during this condition. These studies on motivation suggest that positive incentive such as monetary reward can influence responding of the lateral PFC.

However, one aspect that may modulate the strength of this response could be the potency of the reward. Non-human primate studies commonly utilize primary reinforcers such liquids (e.g., juice; Bermudez and Schultz, 2010; Kobayashi et al., 2010). Studies in humans, by comparison, frequently rely on secondary reinforcers such as monetary incentive (Padmala and Pessoa, 2010; Savine and Braver, 2010). The critical distinction between primary and secondary reinforcers is that while the former is immediately rewarding in itself, the latter can be collected and later exchanged for a rewarding stimulus (e.g., ice cream after a long day of revisions). Recently, Beck et al. (2010) contrasted primary (liquid) vs. secondary (money) reinforcers while volunteers performed a working memory task. Although behavioral performance improved with both types of reward, a double dissociation was apparent in the underlying neural circuitry. Consistent with the findings by Padmala and Pessoa (2010) and Savine and Braver (2010), monetary reward increased the hemodynamic response in the dlPFC. In contrast to these prior findings, the primary reinforcer evoked neural activation in striatal regions and the amygdala. Taken together, these data seem to indicate that some cognitive control circuitry such as the dlPFC is commonly moderated by emotional as well as motivational stimuli, while other executive structures such as the basal ganglia (striatum) are preferentially sensitive to motivational aspects of goal-driven behavior.

Let's talk about sex (in cognition emotion interactions)

Regardless of the distinction between emotional vs. motivation behavior, the previous sections have focused on manipulations that changed from trial-to-trial. One critical question in emotion research is to what extent emotional interference may be driven by inherent differences between participants. One such critical factor, especially with relevance to preponderance for different forms of psychopathology, is biological sex. To avoid potential sex-driven confounds, some studies have elected to recruit exclusively female or male groups of participants (cf. **Table 1**). However, directly contrasting men and women may reveal important differences in how emotion may impact executive control between the genders. In one such endeavor, sex differences were explicitly investigated using aversive olfactory stimulation to induce negative mood while participants performed a working memory task (Koch et al., 2007). Sex-specific interactions between working memory and negative emotion revealed stronger activation for females relative to males in emotion networks including the OFC (BA11) and the amygdala. By comparison, males exhibited stronger activations than females in a wide temporo-parietal–occipital network. These authors suggested (Koch et al., 2007) that during concurrent cognitive control demands within a negative emotional context, perceptual–cognitive processing was predominant in men, while the processing of emotions was prioritized in females. To foreshadow findings from the second part of this review, these results in adults are consistent with documented sex differences in developmental groups (Tottenham et al., 2011) and propose sex-specific processing of emotions during cognitive control tasks making it a critical variable in cognition emotion interaction research.

Genetic and neuropharmacological contributions

Mounting interest in genetic neuroimaging has motivated researchers to examine the impact of different genotypes on cognitive control and their relation to emotion processing. For example, while neurotropic (e.g., BDNF) or serotonergic (e.g., 5-HT) genes contribute to mood and anxiety disorders (Martinowich et al., 2007), dopaminergic (e.g., COMT) genes have been implicated in cognitive control (Barnes et al., 2011). In a small sample of volunteers, Bishop et al. (2006) reported an influence of the COMT

genotype on the attentional matching task during emotional distraction. In the task, also known as the house-faces task, participants were required to determine whether two presented images along a horizontal or vertical dimension were identical or not. However, the other two images presented at the unattended location either contained neutral or emotional images. Presence of the high dopamine activity val/val polymorphism correlated significantly with BOLD signal change in the negative relative to neutral emotional contrast in several regions including the vlPFC, the OFC, and the parahippocampal place area (PPA). Following a similar reasoning, Vollm et al. (2006) provided healthy male participants with the serotonergic drug Mirtazapine to investigate the impact of 5-HT on inhibitory control. Here, the drug modulated inhibitory function in the lateral OFC, the temporal pole, and the insula. Although the influence of the drug was also assessed on a separate reward task in the same study, the interaction of reward on inhibitory control was unfortunately not explicitly examined. Whilst tentative, these promising data indicate effects at the neurotransmitter level on OFC function during emotional challenges.

IMPACT OF MOOD AND PERSONALITY ON EMOTION AND COGNITIVE CONTROL

The above studies have revealed that responses in goal-driven behavior during emotional challenges may differ based on gender or genetic make-up. Therefore, it seems likely that not only transient emotional responses (e.g., surprise, fear) could impact cognitive control, but also sustained variability in mood or personality of the individual. This section reviews such variation in individual differences (**Table 2**).

Anxiety and cognitive control

At some point in our lives, individuals may experience shorter or longer periods of anxiety. According to Eysenck et al. (2007), "anxiety is an aversive emotional and motivational state occurring in threatening circumstances" (p. 336). However, in addition to the state anxiety at a particular moment in time, an individual can also be characterized on how anxious they feel in general, or their level of trait anxiety. Their attentional control theory (ACT) was developed to make specific predictions on how levels of anxiety within the healthy population will impact cognitive control (Eysenck et al., 2007). One central axiom of ACT is that processing effectiveness, which describes the quality of task performance (response accuracy), is less affected by anxiety than processing efficiency, i.e., the relationship between processing effectiveness and cognitive effort exerted during the task (RT). In particular, being in a state of anxiety reduces attentional control and thus processing efficiency. Distracting and salient threat-related information draws additional processing resources away from goal-oriented attention further reducing effective processing. In a series of behavioral studies to probe ACT, Derakshan and colleagues examined the impact of subclinical anxiety on cognitive control processes (Ansari et al., 2008; Derakshan et al., 2009a,b). In a traditional task switching design, participants switched between or repeated basic arithmetic operations (Derakshan et al., 2009b). These arithmetic problems were either simple (addition, subtraction) or complex (division, multiplication). During switching, when cognitive control levels were high, performance was slowed in high anxious but not low anxious participants, an effect that was particularly the case for complex but not simple mathematical operations. In another study, she investigated processing of emotional faces during inhibitory control by virtue of the antisaccade task (Derakshan et al., 2009a). Consistent with the first study and ACT, processing efficiency, as seen in prolonged latency, was reduced in high anxious relative to low anxious participants. By comparison, processing effectiveness, i.e., antisaccade error rates, were less affected.

Subjective levels of anxiety have also been shown to influence psychophysiological correlates of cognitive control. The error-related negativity (ERN) component of the ERP commonly shows an increased negativity over central scalp after an error is committed relative to correctly executed trials (Falkenstein et al., 1990; Gehring et al., 1993). Using a two-dimensional, non-emotional (color or orientation judgments) go/no-go task, Aarts and Pourtois (2010) recently documented that the amplitude of the ERN was larger in high anxious vs. low anxious subjects. Similarly, Amodio et al. (2008) examined differences in personality on a go/no-go task. In his study, higher levels on behavioral inhibition (BIS) were associated with larger N2s and ERNs during no-go trials. Higher scores on the behavioral approach system (BAS), on the other hand, were associated with greater left-sided frontal asymmetry. These data are consistent with the findings by Hajcak and Foti (2008), who had found that the magnitude of the ERN predicted the degree of the defensive reflex during erroneous responses. Taken together, these data suggest that high levels of anxiety or BIS moderate error-related processing.

As alluded to earlier, some researchers have attributed different roles of the left and right hemispheres in processing positive and negative emotions (Davidson, 1995). Engels et al. (2007), for instance, asked participants to perform a (blocked) emotional Stroop task with neutral, pleasant, and unpleasant words while they underwent fMRI. A large cluster of the left but not the right lateral frontal cortex (BAs 9, 44, 45, 47) became visible during negative words relative to neutral words. However, this effect was further moderated by anxious state such that participants scoring high on anxious apprehension showed this effect but not participants having high levels of anxious arousal or low anxious subjects. By comparison, anxious arousal subjects showed right lateralized inferior temporal gyrus activations in the negative relative to neutral contrast. Similarly, undergraduates scoring high on depressive symptoms also showed right temporal gyrus activations for processing of unpleasant words in an emotional Stroop task and, conversely, a leftward lateralization for pleasant words (Herrington et al., 2010). While these studies may suggest a hemispheric divide, an earlier-meta analysis of 65 imaging studies suggests a more complicated picture that includes region-specificity and effects in lateralized processing of emotions (Wager et al., 2003).

Rather than examining emotional valence on lateralized processing within the same task, another approach is to test the effects of valence on different tasks that engage either the left or right hemisphere. One example would be the processing of verbal information by the left and spatial information by the right hemisphere (Smith and Jonides, 1999). Unfortunately, findings in this line of research have not been without contradictions either. Gray (2001)

Table 2 | Lists relevant studies in the field of cognitive control emotion interactions in healthy adults using an individual differences approach.

First author	Year	Sample size	Gender	Method	Cognitive control paradigm/task	Emotional/ motivational stimuli	Main point
Ansari and Derakshan	2011	64	m/f	beh	Delayed antisaccade	–	Delayed latency for high vs. low anxious in standard antisaccade task; introduction of delay evened out performance between groups
Ansari et al.	2008	59	m/f	beh	Antisaccade	–	Reduced antisaccade switch costs in high anxious relative to low anxious
Davis and Nolen-Hoeksema	2000	62	m/f	beh	WCST	–	Ruminators make more errors on WCST than non-ruminators
De Lissnyder et al.	2010	96	m/f	beh	Affective shift task	KDEF	↑ Switch cost for high vs. low ruminators
Derakshan et al.	2009b	59	m/f	beh	Task switching	–	↓ Performance on switch trials for high anxious vs. low anxious in high complexity vs. low complexity trials
Derakshan et al.	2009b	18 + 19	m/f	beh	Antisaccade	Parke-Waters three-dimensional face model	High anxious slower latency during angry faces than low anxious in antisaccade but not prosaccade
Gray	2001	152	m/f	beh	2-Back WM task (verbal vs. spatial)	Prior mood induction via video clips	Spatial performance enhanced by withdrawal (fearful) and impaired by approach (happy); the reverse for verbal stimuli
Holmes and Pizzagalli	2007	57	m/f	beh	Simon + Stroop tasks	Rigged feedback	Subclinical depression moderates impaired posterior and post conflict performance adjustment; particularly during negative but not positive feedback
Koven et al.	2003	138	m/f	beh	Emotional Stroop	ANEW	Anxiety sensitivity moderates responding to threat relative to neutral words
Shackman et al.	2006	31 (exp1) 24 (exp2) 42 (exp3)	m/f	beh	3-Back WM task (verbal vs. spatial)	Emotion induction via threat of shock	Threat of shock reduced spatial but not verbal *n*-back performance
Whitmer and Banich	2007	43	m/f	beh	Task switching	–	Depressive rumination affected set inhibition; anger and intellectual rumination affect switch costs
Aarts and Pourtois	2010	32	m/f	ERP	Two-dimensional go–no-go (color vs. orientation)	–	ERN/Ne to errors ↑ for high vs. low anxious
Amodio et al.	2008	48	m/f	ERP	Letter go–no-go task	–	BIS scores correlated with no-go N2/ERN; BAS scores correlated with frontal asymmetry
Ansari and Derakshan	2011	34	m/f	ERP	Antisaccade	–	↓ERP waveforms in high anxious subjects during antisaccade trial preparation
Luu et al.	2000	42	m/f	ERP	Target detection task	–	↑ ERN for participants with high negative affect and negative emotionality
Bishop et al.	2004	27	m/f	fMRI	House-faces task	Eckman and Friesen (distractors)	Relationship between ACC and PFC activations and individual levels of anxiety during task

Denkova et al.	2010	18	Females	fMRI	Delayed WM	Emotional distraction (NIMSTIM)	Perceptual and executive areas correlate with anxiety and WM performance
Engels et al.	2007	42	m/f	fMRI	(Blocked) emotional Stroop	ANEW, neutral, pleasant, unpleasant	Anxious type interacts with hemisphere in left IFG in negative vs. neutral words
Fales et al.	2008	96	m/f	fMRI	3 *n*-Back task (neutral words)	Mood induction through video clips (neutral and fearful)	Trial type × anxiety interaction in right vlPFC
Fales et al.	2010	29	m/f	fMRI	Emotional 2 *n*-back task	Gur (happy, fearful, neutral)	Trait anxiety modulates PFC activation
Gray et al.	2005	60	m/f	fMRI	*n*-Back WM task	–	↑ BAS scores correlated negatively with activation in lateral PFC, ACC, IPL
Gray et al.	2002	14	m/f	fMRI	*n*-Back WM task	–	↑ BAS scores correlated negatively with activation in caudal ACC
Vanderhasselt et al.	2011	34	m/f	fMRI	Emotional go–no-go task	KDEF	Positive correlation between dlPFC BOLD response and brooding scores during inhibiting negative vs. inhibiting positive information

Abbreviations as in ***Table 1***.

on the one hand, and Shackman et al. (2006), on the other hand, used similar variations of the *n*-back working memory task with a perceptual/spatial variant and a verbal variant. Gray induced mood in participants through affective video clips before the two different 2-back working memory tasks. On the perceptual version, spatial performance was improved by previous encounter with fearful stimuli and impaired during happy context blocks, while the reverse, impairment during fearful and improvement during happy conditions, was true for the verbal task. Importantly, these findings were strongest for subjects with low cognitive control (high error group). By stark contrast, using a similar design and methodology, Shackman et al. (2006) found that threat of shock (i.e., fearful condition) reduced performance on the spatial task but not the verbal task. Moreover, this was particularly the case for participants with high levels of cognitive control (low error group). These findings were replicated in a second experiment of the same study in high anxious subjects (high BIS scores) but not in low anxious subjects. A third study using the threat of shock procedure also reported impaired performance on the spatial but not verbal *n*-back task and a positive association between anxiety levels and performance impairment (Lavric et al., 2003). While these findings support the idea of a differential emotional impact on tasks tapping into different cognitive domains, future research will need to clarify to what extent these contradictory findings may have been driven by individual differences in cognitive control abilities (high vs. low performers) or methodology (introspectively induced emotion vs. continuous threat of physical shock).

Leaving hemispheric contributions on their respective sides, so-called resting-state fMRI studies have documented that a state of anxiety modulates brain rhythms during periods of rest (Zhao et al., 2007; Liao et al., 2010). Neural circuitry usually activated during such periods of rest has also been described as the default mode network (DMN). Recent enquiry has begun to address the impact of anxiety on the engagement of the cognitive control network for a particular task when changing from the "idle" state of the DMN. In a large fMRI study ($n = 96$), Fales et al. (2008) induced mood in high anxious and low anxious participants prior to an emotionally neutral 3-back working memory task. High anxious participants showed a lower level of sustained activation in the DMN than low anxious participants and this group also showed increased activation of the right vlPFC (BA47) during trial-related engagement. Such preliminary evidence might suggest that the neural activation during rest associated with a particular mood state may be predictive of subsequent engagement of cognitive control. Future study will have to challenge such a conjecture.

Executive deficits in depression

Similar to theories in anxiety, models in mood disorders have postulated a critical involvement of executive function processes in the development and maintenance of depression (Joormann, 2010). Joormann (2010) proposed that, in depressed persons, a deficit in being able to regulate mood during the presentation of negatively valenced material within working memory leads to impairments in cognitive control. According to the model, a negative event might induce negative mood, which in turn activates negative cognitions. Patients vulnerable to depression may linger ("ruminate")

on these negative cognitions, thus preventing that this material can be expelled from working memory to free processing resources.

Some studies have examined dimensional measures of depression-linked traits on cognitive control (Holmes and Pizzagalli, 2007; De Lissnyder et al., 2010). Holmes and Pizzagalli (2007) investigated the impact of subclinical depressive symptoms on non-emotional Simon and Stroop task performance. However, performance feedback was rigged such that subjects were presented with positive or negative feedback regardless of their actual performance. Participants with increased depressive traits evidenced impaired adjustment in RT on trials following an error (Simon task) or conflict (Stroop task), especially during negative but not positive feedback. However, it should be noted that high and low depressive symptom groups did not differ on the main variables of congruency or conflict suggesting no global impact on performance. Rather, these data show that subclinical depressive symptomatology may moderate subtle trial-to-trial behavioral adjustments.

Studies in patients with mood disorders including major depressive disorder (MDD; Harvey et al., 2005; Joormann and Gotlib, 2008; Vasic et al., 2009; Levens and Gotlib, 2010), Mania (Elliott et al., 2004), or bipolar disorder (BD; Wessa et al., 2007) are consistent such proposals. These studies have found perturbed activations in lateral frontal circuitry such as the IFG, MFG, or SFG during emotional go/no-go tasks (Elliott et al., 2004) or emotionally neutral *n*-back tasks (Harvey et al., 2005). In addition, a connectivity study in MDD patients during a working memory task revealed perturbations in functional connectivity in the ACC (BA 24/32) and left dlPFC (BA9/46) and vlPFC (BA44; Vasic et al., 2009). Taken together, these data suggest vulnerability of the lateral PFC to negative mood that spans across several executive functions including working memory and response inhibition.

However, an often-noted constraint of fMRI methodology more generally is that it is limited in its capacity to allow inferences on causal relationships between behavior and patterns of activation in the brain. Yet, critical questions regarding the causality and directionality of mood and cognitive control remain untouched. Provocatively asked, is reduced cognitive control a risk-factor for a mood or anxiety disorder or does having an affective disorder cause reductions in cognitive control? One possible way to address these issues is by use of invasive technology such as brain stimulation. rTMS has been used successfully to disturb cognitive performance by discharging neurons in the cortex and is thus ideally suited to examine lateral PFC function (Walsh and Pascual-Leone, 2003). In a series of studies, Vanderhasselt et al. (2009a,b), administered rTMS over the left dlPFC in patients with MDD while these performed a switching task between different modalities (auditory, visual). These studies revealed two findings. First, a single session of rTMS improved attentional control. Second, behavioral control was also improved in treatment responders but not non-responders after 2 weeks of rTMS therapy. These data point toward TMS as a possible tool to further investigate the contribution of perturbations of cognitive control to mood and anxiety disorders.

The previous sections provided an updated overview of the current state of research in emotion and cognitive control interactions. This overview can now be used to assess similar progress on this topic in developmental cognitive neuroscience.

DEVELOPMENTAL (COGNITIVE) NEUROSCIENCE

Over the past 10 years, developmental cognitive neuroscience has blossomed to a major field of enquiry. Within the present context, this line of research can provide valuable knowledge of the developmental contribution to the interplay between cognitive control and emotion in the immature organism. Anatomically, longitudinal studies have delineated distinct developmental trajectories for individual brain regions such as a slow rate of PFC maturation (Giedd et al., 1999; Paus et al., 1999; Gogtay et al., 2004). If the PFC and other regions such as those of the limbic system evidence different developmental trajectories, then one would expect task performance and behavior to reflect these differences. Indeed, it has been proposed that the increased risk taking of teenagers relative to children and adults originates from discrepancies in PFC and striatal development (Dahl, 2004; Ernst et al., 2006; Somerville and Casey, 2010). These authors (Ernst et al., 2006; Somerville and Casey, 2010) argue that the relatively faster development of reward-related striatal neurocircuitry and slower development of regulatory PFC systems result in increased sensitivity to reward seeking and risky behavior. As such, children and adolescents would be expected to show greater difficulty in coping with distracting emotional information during goal-directed behavior than adults. Mounting research has examined the development of cognitive control (Casey et al., 1995; Bunge et al., 2002; Crone et al., 2006; Rubia et al., 2007; Ernst and Mueller, 2008) and emotion regulation (Herba et al., 2006). The next section will review two sets of studies: (1) studies of cognition emotion interactions in healthy development and (2) cognitive control (and their interaction with emotion) in pediatric mood and anxiety disorders (**Table 3**).

COGNITION EMOTION INTERACTIONS IN HEALTHY DEVELOPMENT

A recent, large behavioral study examined the influence of emotional valence on cognitive control in children ages 4–11 and adults, who completed two simple variants of a conflict task (Lagattuta et al., 2011). In the day–night variant, participants responded with the opposite term of an image they were presented with (e.g., respond with "day" to an image showing the night sky). In the happy–sad variant, again, participants were required to respond with the opposite expression. As expected, performance improved linearly with age but performance in the happy–sad task was worse across all groups than the day–night task. In another developmental study, Tottenham et al. (2011) compared performance on an emotional go/no-go task among children (5–12 years), adolescents (13–18 years), and adults (19–28 years). Using a block design, inhibitory control performance was assessed when emotional faces served as the prepotent "go" or the non-prepotent "no-go" stimuli. In-line with prior research, cognitive control, and emotional regulation improved with age. Importantly, across age, false alarms were higher during emotional no-go stimuli relative to neutral no-go stimuli suggesting reduced inhibitory control in the presence of salient emotional information. Additional effects of sex indicated better discrimination of emotion in girls relative to boys. An fMRI variant of the same task that

Table 3 | Lists relevant studies in the field of cognitive control emotion interactions in healthy children and adolescents and pediatric mood and anxiety disorders.

First author	Year	Sample size	Gender	Method	Cognitive control paradigm/task	Emotional/ motivational stimuli	Main point
STUDIES IN HEALTHY DEVELOPMENT AND INDIVIDUAL DIFFERENCES							
Jazbec et al.	2006	53	m/f	beh	Antisaccade	Reward vs. no reward	Reward ↑ performance in both groups; incentives modulate incorrect antisaccades in adolescents
Kohls et al.	2009	65	Boys	beh	Go–no-go task	Reward vs. no reward	Reward (social and non-social) ↑ inhibitory control
Tottenham et al.	2011	100:53 kids 24 adol. 23 adults	m/f	beh	Emotional go–no-go task	Eckman and Friesen faces	Improvement in cognitive control and emotion regulation across age groups
Hare et al.	2008	12 + 24 + 24	m/f	fMRI	Emotional go–no-go	NIMSTIM	Age moderates emotional processing during no-go task
Ladouceur et al.	2009	31 (HA) + 26 (LA)	m/f	beh	Emotional face *n*-back task	NIMSTIM	High anxious participants had slower RT on fearful *n*-back task; this effect was increased in younger relative to older participants
Visu-Petra et al.	2010	60	m/f	beh	Emotional odd-one-out task	NIMSTIM	High anxious (rel. to low anxious)showed slower RT and ↑ errors to happy faces but improved performance to angry faces
STUDIES IN PEDIATRIC MOOD AND ANXIETY DISORDERS							
Dickstein et al.	2007	26 + 33 + 33	m/f	beh	Set-shifting task	–	Bipolar relative to controls impaired on some set-shifting abilities
Hardin et al.	2009	25 + 25	m/f	beh	Antisaccade	NIMSTIM emotion + monetary reward	ANX improved antisaccade latency to fear faces; controls improved latency to happy faces
Jazbec et al.	2005	28 + 11 + 12	m/f	beh	Antisaccade	Reward vs. no reward	Altered latency patters of incorrect antisaccades across groups
Ladouceur et al.	2005	17 + 16 + 24 + 18	m/f	beh	Emotional *n*-back task	Child friendly IAPS face or scene?	MDD + co-morbid AXN/MDD longer RT during negative vs. neutral background; controls longer RT during positive background
Ladouceur et al.	2006	23 + 19 + 26	m/f	beh	Emotional go–no-go task	Eckman and Friesen	Changed go RT during emotion trials in ANX/DEPR groups
McClure et al.	2005	34 + 18	m/f	beh	Flanker, stop, stop–change, CPT, face labeling	–	Deficits in cognitive control and emotion processing (same study but different expts.)
Mueller et al.	2010b	20 + 23	m/f	beh	Antisaccade	Reward vs. no reward	Reward improved performance in controls but not bipolar group
Rich et al.	2007	35 + 21 + 26	m/f	ERP	Affective Posner task	Rigged feedback	Reduced P3 amplitude in bipolar relative to control group during negative feedback
Britton et al.	2010	15 + 20	m/f	fMRI	Set-shifting task	–	Controls showed higher activation than OCD in left IFG in mixed vs. single blocks
Carrion et al.	2008	16 + 14	m/f	fMRI	Blocked go–no-go task	–	Control: ↑ activation in left MFG; early stress: ↑ activation PTSD in cuneus, IOG, ACC
Huyser et al.	2010	25 + 25	m/f	fMRI	Tower of London task	–	Control > OCD during planning relative to control in IFG and parietal lobe
Leibenluft et al.	2007	26 + 17	m/f	fMRI	Stop-signal task	–	Control > bipolar on correct vs. incorrect stop trials in vlPFC
Mueller et al.	2010a	12 + 21	m/f	fMRI	Stop–change task	–	Early stress ↑ activations in fronto-striatal circuitry
Nelson et al.	2007	25 + 17	m/f	fMRI	Stop–change task	–	Bipolar > control in dlPFC in change relative to go trials
Singh et al.	2010	26 + 22	m/f	fMRI	Go–no-go task	–	Bipolar > control in dlPFC in no-go – no

Abbreviations as in ***Table 1***.

aimed to capture the neural correlates of these effects, increased RT to fearful faces was coupled with higher amygdala activation, while an inverse relationship existed in the ventral PFC (Hare et al., 2008). Moreover, amygdala activation was increased in adolescents relative to children and adults. Taken together, these data imply that basic emotional interference as compared to non-emotional conflict is evident as early as 4 years of age and that emotional neurocircuitry during such conflict tasks is sensitive to development.

Similarly, mirroring parallel research in adults (e.g., Beck et al., 2010; Padmala and Pessoa, 2010; Savine and Braver, 2010), studies of adolescent development have also investigated the impact (Jazbec et al., 2006) and type (Kohls et al., 2009) of motivation on performance. Jazbec et al. (2006) paired antisaccade eye movements with positive (win $1 for a correctly executed antisaccade) or negative (lose $0.5 for an erroneous antisaccade) reward/punishment contingencies. Although monetary incentive improved inhibitory control during antisaccades in both adolescents and adults, this effect was stronger for the adolescent group. Kohls et al. (2009) used both monetary incentive and social (i.e., positive faces) feedback and documented a stronger improvement of inhibitory control during a go/no-go task in the monetary reward condition in 10-year-old children. These data would advocate that despite the importance of positive caregiver feedback during development, children already show preponderance to secondary reinforcers such as monetary reward.

COGNITION EMOTION INTERACTIONS IN CHILDHOOD MOOD AND ANXIETY DISORDERS

The previous set of studies informed on the influence of emotion on cognitive control in typically developing individuals. However, neuroanatomical work has implicated disturbances in developmental trajectories of brain circuitry in childhood psychiatric disorders (Shaw et al., 2010). Studies in youths suffering from psychopathology provide a window into the (long-term) developmental changes that mood perturbations may exert on cognitive control. Research in this particular area has utilized several tasks including the (emotional) go/no-go task, the Tower of London task, the flanker task, set-shifting tasks, *n*-back tasks, and the antisaccade task (**Table 3**).

Antisaccade research in pediatric anxiety has revealed emotion-dependent responding whilst executing strong inhibitory control (Hardin et al., 2009). In that study, adolescents with and without an anxiety disorder had to fixate on a centrally presented image of an emotional facial expression (happy, fearful, or neutral). After a short delay, a peripheral asterisk appeared and participants were required to perform an antisaccade to the opposite direction to the asterisk. During instruction prior to the task, participants were told that the face and its emotion were irrelevant and should be ignored. In anxious adolescents, antisaccade latency improved when subjects had to saccade away, i.e., disengage from fearful faces (relative to neutral expressions). By contrast, for controls, antisaccade latency was speeded during the presentation of happy faces relative to neutral faces. Although the principle behind such a study design is reminiscent of tasks investigating attentional biases (e.g., Bar-Haim et al., 2007), the antisaccade task necessitates the execution of strong inhibitory control, which is absent from traditional attentional bias designs. Secondly, although Hardin et al. (2009) presented face stimuli centrally, it is also possible to present these images peripherally allowing a comparison between engagement and disengagement of emotional stimuli during inhibitory control. Using a similar principle in a study with manual responses, Ladouceur et al. (2005) presented emotional stimuli as background images during performance of a non-emotional *n*-back task in anxious and depressed youths. Interestingly, her findings were the opposite to those of Hardin et al. (2009). Here, Ladouceur et al. (2005) documented prolonged RTs for the depressed group (with and without co-morbid anxiety) in the presence of a negative background (relative to neutral), while controls responded slower during the presentation of a positive background. Due to the differences in methodologies (eye tracking vs. behavioral), tasks (antisaccade vs. *n*-back), groups (depressed vs. anxious), and stimuli (whole scenes vs. faces) it is difficult to isolate the factors that might have contributed to the shift in response patterns. However, regardless of the directionality of the effects, these data indicate that cognitive control, specifically during incidental emotional processing, is altered in pediatric anxiety and depression mirroring patterns in adults.

Adolescents suffering from BD also evidence deficits in cognitive control abilities (McClure et al., 2005; Pavuluri et al., 2006). For instance, on the same group of subjects, McClure et al. (2005) examined both cognitive control abilities and emotional processing in bipolar youth using a variety of executive function measures and face labeling tasks. Although she noted impairments in BD youths in both domains, an interesting avenue would have been to employ tasks testing the potential interaction of these deficits. Preliminary evidence from the antisaccade task suggests that BD youths are insensitive to monetary reward, as antisaccade performance improved with incentive in controls but not patients in another study (Mueller et al., 2010b). Although emotional instability is a hallmark of BD, electrophysiological evidence suggests changes in executive attentional processing in BD youths during controlled, elicited frustration (Rich et al., 2007) consistent with increased BOLD activity in dlPFPC during inhibitory control tasks (Nelson et al., 2005; Singh et al., 2010). Targeted studies are needed to directly address the influence of emotion on cognitive control processes in pediatric BD.

Studies in pediatric anxiety come to similar conclusions. While much work has focused on establishing the neural correlates of aberrant emotional processing in pediatric anxiety (McClure et al., 2007; Monk et al., 2008), some functional imaging work has investigated cognitive control in types of anxiety disorder such as Obsessive–Compulsive Disorder (Britton et al., 2010; Huyser et al., 2010) or Post-Traumatic Stress Disorder (Carrion et al., 2008). Britton et al. (2010) asked participants with pediatric OCD to complete a simple set-shifting task while undergoing fMRI. Activations for control subjects were increased relative to patients in the left IFG during shift blocks relative to single blocks. Moreover, correlations of behavioral performance with striatal activation were opposite for both groups indicating a drastic change in cognitive control function in OCD. Consistent with Britton et al.'s (2010) findings, Huyser et al. (2010) tested planning abilities in pediatric OCD by virtue of the Tower of London task. Here, activations for controls were also larger relative to patients in left IFG. Of note, while group activations for patients tended to be reduced in the

OCD studies, they were increased in the bipolar studies. Such data suggests psychopathologic specificity on neural circuitry subserving cognitive control and warrant further enquiry with regards to the emotional phenotype.

Studies in populations at-risk for developing psychopathology provide an intermediate step between healthy development and pediatric mental illness. In two independent but complementary fMRI studies modulation of dACC and PFC activation have been linked to adverse life experiences in cognitive control tasks in these cohorts. Carrion et al. (2008) examined the neural correlates of inhibitory control in youths with post-traumatic stress symptoms due to prior exposure to violence, physical, or sexual abuse. Mueller et al. (2010a) examined cognitive flexibility in international adoptees who had been removed from their biological parents due to emotional neglect. Both studies reported increased activations for the trauma groups relative to age-matched controls in lateral PFC and dACC (BA 32/24). These data suggest that early traumatic experience moderates brain regions involved in cognition emotion interactions. Epidemiological studies suggest that individuals with experience of early trauma are at high risk for developing psychopathology and affective disorders (Green et al., 2010). One question is whether these neurobiological changes in the frontal network may contribute to this increased risk.

SUMMARY AND NEUROCHEMICAL IMPLICATIONS

Several intriguing findings emerged from this review. Among the most consistent results was that at a behavioral level, negative emotion/stimuli impaired executive control processes while (monetary) reward improved cognitive control function in most cases. The impact of positive emotion on cognitive control was less clear: while some studies found beneficial effects on executive function, this appeared to depend on the exact cognitive process concerned (e.g., perseveration vs. distractibility). At the electrophysiological level, ERP studies had been conducted using within-subject or between-subject designs testing individual differences. These studies have shown that emotional stimuli or anxious state moderated ERP components indicative of executive processes such as the N2, P3, or ERN during error monitoring, conflict resolution, or inhibitory control. In the neurocognitive data, consistent findings emerged with regards to the neural circuitry involved in such integration processes. For example, the dorsolateral PFC, particularly the IFG was generally reactive to emotional (negative) stimuli in the context of a cognitive control process. The middle and frontal gyri of the PFC were also frequently activated during emotional distraction. Given the involvement of the ACC in both executive control and emotion, this structure also turned out to be (unsurprisingly) a major player in interactions between the emotional and executive system. However, consistent findings were not limited to lateral cortical structures but also involved deeper subcortical structures like the amygdala during emotional conflict or even pure non-emotional error processing regardless of response modality (e.g., manual or saccadic; **Figure 1**).

Although much of the reviewed literature focused on identifying the loci of emotion cognitive control interactions, some of the presented evidence utilized connectivity analyses to point toward a sophisticated and complex network of interactions between individual brain regions. Most consistently reported was a coupling between the ventral/rostral ACC and the amygdala during various forms of conflict involving emotion (e.g., Egner et al., 2008; Kanske and Kotz, 2011a). These authors (Egner et al., 2008) proposed that the rACC exerts a top-down influence on the amygdala response and thereby inhibiting emotional distracter processing during conflict resolution. In other studies, ACC activity was related to dlPFC activations (Mohanty et al., 2007) attributing a mediator role between executive and limbic processes to the ACC (Shackman et al., 2011).

Despite these intriguing findings, one critical question is how these neurobiological data in gray matter relate to mechanisms at the neurochemical level. Neurotransmitters such as dopamine have been postulated to carry important functionality in cognitive control (Braver and Cohen, 2000), on the one hand, and positive emotion (Ashby et al., 1999) and reward and motivational behavior (Schulz, 2002), on the other hand. Given widespread distribution of dopamine throughout the PFC, and a hypothesized role in all three processes, it would seem likely that dopamine might critically contribute to, or mediate, emotional influences on cognitive control. In their model of dopaminergic influences on positive emotion, Ashby et al. (1999) hypothesized that dopamine release from the ventral tegmental area to a wide cortical and subcortical network including the PFC, ACC, Nacc, striatum, hippocampus, or the amygdala, modulates dopaminergic baseline levels in these structures thus up (or down) regulating levels of

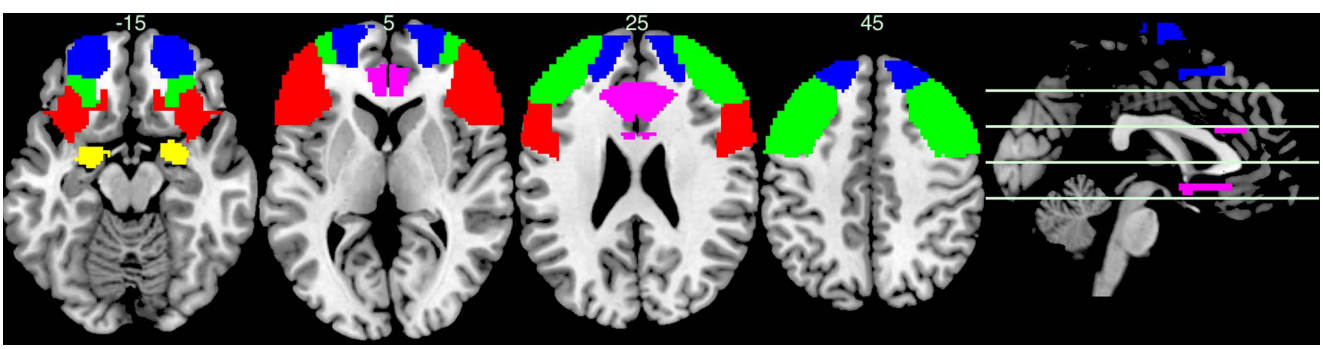

FIGURE 1 | Axial slices of the brain regions consistently reported in studies of cognitive control emotion interactions. Sagittal image on the right shows corresponding height of axial slices. Color schema: Yellow = amygdala, red = IFG, green = MFG, blue = SFG, pink = ACC.

activity and thus influencing affect. According to evidence in favor of this model, a particular crucial role may be played by the Nacc, which enjoys direct connections to the amygdala and PFC and may contribute to evaluating (and updating) goal-directed behavior during error monitoring (Münte et al., 2008). However, as explicitly stated the dopamine theory of positive emotion does not make any predictions about negative emotions or, by extension, valence-specificity of involved brain regions. It could be imagined that the strength of a given region in the evaluation of affective significance relative to the other structures is dependent on the valence of the concerned affective state. Although these details are still under investigation, it appears likely that mesocorticolimbic dopamine pathway is involved in the mediation between emotional and executive processes via subcortical structures. However, it must be pointed out that other neurotransmitters (e.g., serotonin), which also play a pivotal role in emotional behavior, and mood and anxiety disorders in particular (Martinowich et al., 2007), will likely contribute to these neurochemical mechanisms. Therefore, a full account of the neurochemical contribution to emotion cognition interactions should additionally consider other neurotransmitters.

OPEN QUESTIONS FOR FUTURE RESEARCH

As noted in the previous section, corroborative evidence has emerged on the influence of emotion on cognitive control. Yet, this review has also uncovered some inconsistent findings giving rise to new questions that demand future attention. This section aims to highlight some of these open questions. Since the goal of this review was to reconcile on-going work in adults with emerging research in developmental groups, these questions aim to further research in streams.

A finding that provides a bridge to illustrate the necessity to merge fields is provided by an fMRI study in healthy adults (Schulz et al., 2009) and a DTI study in healthy children (Madsen et al., 2010). In adults, Schulz et al. (2009) reported significant activations during response inhibition to emotional faces in the *pars opercularis* of the IFG. In the children, Madsen et al. (2010) found that white matter diffusivity (fractional anisotropy, FA) in the *pars opercularis* predicted behavioral stop-signal reaction time (SSRT). Two conclusions can be drawn. First, these data would suggest that connectivity findings in adolescents may provide clues to explaining some of the variance contributing to performance in functional imaging studies in adults. Second, white matter analyses may contribute to increase our understanding of the neurobiological mechanisms underlying emotion cognition interactions and may complement studies of findings in gray matter.

As noted previously, some theoretical models of emotion propose that both emotion and motivation are necessary to fully account for goal-driven behavior (Roseman, 2008). The reviewed evidence would implicate some shared neurocircuitry such as the PFC (e.g., Van Dillen et al., 2009; Savine and Braver, 2010), while other areas such as the striatum are process-sensitive. While many studies have either investigated reward or emotion separately, one question might concern the level of similarity (or dissimilarity) of effects on cognitive control (see also Chiew and Braver, in this special issue for an outline of such an agenda). Preliminary encouragement for such an endeavor comes from a behavioral study in anxious adolescents that has examined the impact of concurrent reward and incidental emotion processing on antisaccade performance (Hardin et al., 2009). Furthermore, developmental studies have shown that even during young age, secondary reinforcers such as monetary incentive may exert stronger influences than positive (emotional) feedback on executive function (Kohls et al., 2009). Such promising data further strengthen a joint research agenda of adult and developmental work and point toward possible paradigms to examine these issues.

A guaranteed issue for future debate is the extent different emotions (fear, anger, sadness, happiness, disgust) may be processed in diverse neural systems (Habel et al., 2005; Vytal and Hamann, 2010). In other words, to what extent are affective influences on cognitive control emotion-specific? For instance, does one but not another emotion elicit a selective improvement in performance (e.g., Gray, 2001; Lavric et al., 2003)? Does this hold true for the basic emotions such as fear and happiness or does this extend to other emotions such as sadness and disgust?

Although the above questions are of interest to better understand how emotions influence cognitive control, one issue that complicates any positive findings is the generalizability across gender. While some studies have tried to avoid this issue by relying on gender-specific recruitment (Habel et al., 2005; Pereira et al., 2010), initial findings suggest sex-specific neural differences (Koch et al., 2007) supported by developmental work (Tottenham et al., 2011). Such data might suggest that future studies will need to consider any impact of biological sex on their findings regardless of age.

Finally, with regards to clinical relevance and application, the current review has highlighted some exciting developments in adult and pediatric psychopathology in relation to cognitive control dysfunction in mood and anxiety disorders. Following psychological theories that propose executive deficits in depression (Joormann, 2010) or anxiety (Eysenck et al., 2007), a future line of research could examine to what extent therapeutic training on cognitive control measures might improve symptomatology and alter interactions with executive control.

CONCLUSION

Within the last decade much progress has been made to understand the neurobiological mechanisms underlying emotional influences on executive processes. While clear candidate regions, particularly the PFC, have been identified, open questions concern the relationship between individual regions. An important task for future work will be to integrate theoretical models of emotion and cognitive control and define the conditions under which these systems may recruit additional or distinct neurocircuitry from their respective networks. Despite moderate progress in adults, developmental research in the field is still in its infancy. Delineating the dynamic changes of these interactive processes across time may provide critical information on the psychological and neurobiological mechanisms by which affective processes perturb cognitive control in the developing brain. In turn, such discovery might aid in understanding the ontogenesis of pediatric mood and anxiety disorders during a period of constant change.

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Uncertainty and cognitive control

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A growing trend of neuroimaging, behavioral, and computational research has investigated the topic of outcome uncertainty in decision-making. Although evidence to date indicates that humans are very effective in learning to adapt to uncertain situations, the nature of the specific cognitive processes involved in the adaptation to uncertainty are still a matter of debate. In this article, we reviewed evidence suggesting that cognitive control processes are at the heart of uncertainty in decision-making contexts. Available evidence suggests that: (1) There is a strong conceptual overlap between the constructs of uncertainty and cognitive control; (2) There is a remarkable overlap between the neural networks associated with uncertainty and the brain networks subserving cognitive control; (3) The perception and estimation of uncertainty might play a key role in monitoring processes and the evaluation of the "need for control"; (4) Potential interactions between uncertainty and cognitive control might play a significant role in several affective disorders.

Keywords: uncertainty, cognitive control, executive function, emotion, volatility, ambiguity, monitoring, motivation

Decision-making during uncertainty is classically defined as a situation that has limited or incalculable information about the predicted outcomes of behavior (Huettel et al., 2005). Uncertainty is considered a key dimension of everyday behavior that has a significant influence on decision-making (Yoshida and Ishii, 2006), and important links with emotion and psychopathological disorders (Holaway et al., 2006; Boelen and Reijntjes, 2009). Although uncertainty has long been a topic of scientific interest (see Bertelson and Boons, 1960), it has become an object of intense investigation in more recent years, inspiring a growing number of behavioral, neuroimaging, and computational studies.

Most of the available experimental studies referring to the concept of uncertainty have taken place within the realm of research on decision-making processes. More specifically, these studies usually employ tasks in which participants have to learn to predict future outcomes on the basis of past outcomes that often take the form of positive and/or negative reinforcements. Uncertainty in these paradigms is usually manipulated by the variation of the predictive power of past outcomes[1]. Much of this research has focused on statistical models of learning such as Bayesian or Reinforcement learning models (Daw et al., 2005; Kording and Wolpert, 2006; Yoshida and Ishii, 2006; Krynski and Tenenbaum, 2007). Most evidence to date suggests that humans are able to effectively handle uncertainties in the environment to predict future events and make appropriate decisions (Volz et al., 2003; Hsu et al., 2005; Yu and Dayan, 2005; Kording and Wolpert, 2006; Yoshida and Ishii, 2006; Behrens et al., 2007).

However, although these approaches have been generally successful in providing formal algorithms fitting human behavior in uncertain conditions (Steyvers et al., 2003; Chater et al., 2006; Yoshida and Ishii, 2006; Nassar et al., 2010), these models tend to be largely agnostic regarding the specific cognitive mechanisms recruited for this successful adaptation. In particular, with a few exceptions (e.g., Daw et al., 2005), it is unclear if implicit learning processes are sufficient to adapt to uncertainty or if high level cognitive control processes are also involved. Yet, the possibility that cognitive control processes (or executive function) are involved in overcoming uncertainty has often been proposed (e.g., Huettel et al., 2005). In addition, uncertainty may also be to some extent tacitly present in many cognitive control models and tasks (see Botvinick et al., 2001; Miller and Cohen, 2001). However, the field of research on uncertainty and the field of research on cognitive control processes have evolved mostly independently and attempts to integrate them are rare. The goal of this article is to explicitly address the potential relationships between the constructs of uncertainty and cognitive control. We will first outline the conceptual overlaps between uncertainty and cognitive control. Next, we will review a large body of neuroimaging evidence suggesting an overlap between brain areas involved in processing uncertainty and the neural network subserving cognitive control. Then, we will review evidence suggesting that uncertainty has a significant influence on a key component of cognitive control – monitoring processes. Finally, we will review a body of evidence suggesting that the construct of "Intolerance of Uncertainty" (IOU) is linked to affective disorders widely known to be associated with deficits in cognitive control.

THEORETICAL OVERLAPS BETWEEN UNCERTAINTY AND COGNITIVE CONTROL

UNCERTAINTY

Uncertainty is generally seen as a realization that our beliefs and representations of the world are unable to accurately predict future events in our environment. In behavioral and cognitive sciences, uncertainty has mainly been defined within the scope

[1]This form of uncertainty can be referred to as "outcome uncertainty," which has to be differentiated from the concept of "task uncertainty," which refers to uncertainty about the nature of the task to be performed in the next trial in multi-trial task-switching paradigms (e.g., Rubin and Meiran, 2005). The present review focuses essentially on outcome uncertainty.

of decision-making and therefore refers to a difficulty to predict events that are the consequences of our actions (Volz et al., 2003; Hsu et al., 2005; Huettel et al., 2005; Yu and Dayan, 2005). Uncertainty can present itself under many forms. For instance, an influential theory (Yu and Dayan, 2005) proposed that there are two fundamental types of uncertainty: expected and unexpected uncertainty. The former relates to environments in which available information is a weak predictor of future events. However, this unreliability is stable and known by agents in the environment. For instance, driving in a city where most drivers consistently display a poor compliance of traffic rules is a typical example of expected uncertainty: the rules are weak predictors of how drivers behave (and of how they will react to our own behavior), and this unreliability is known and relatively stable. In contrast, unexpected uncertainty refers to a situation in which fundamental changes in the environment invalidate past predictions. For instance, pilots of aeroplanes in cruise at high altitude experience this type of uncertainty when they encounter turbulence that had not been forecast.

Empirical literature on uncertainty has used experimental paradigms that can be subsumed under these two categories. A large number of studies induced uncertainty by lowering the predictive power of task cues (e.g., Bertelson and Boons, 1960; Scheffers and Coles, 2000; Pailing and Segalowitz, 2004; Huettel et al., 2005). Typically in these studies, participants are shown cues which are probabilistic predictors of a given outcome (e.g., a square that predicts the occurrence of a reward for 80% of test trials). Uncertainty in these paradigms is attained by lowering the accuracy of the prediction (e.g., a square that predicts rewards only for 60 or 50% of test trials). Importantly, in many of these paradigms the unreliability of cues remains stable during long blocks of trials (e.g., Huettel et al., 2005), which approximates the concept of expected uncertainty. In contrast, other studies manipulated the *volatility* of task rules (e.g., Behrens et al., 2007), in which uncertainty is achieved by dynamically changing stimulus–response rules across trials. For instance, if rule A involves reward outcomes for 80% of squares and rewards for 30% of circles, volatility will be achieved if this rule frequently alternates with a rule B that would involve the inverse reward probabilities of rule A. A stable (and thus "certain") condition would involve a long sequence of trials in which only one of the rules is true. Manipulating uncertainty through volatility of rules approximates Yu and Dayan's (2005) concept of unexpected uncertainty, as it introduces fundamental changes in the environment that invalidate past predictions.

The common feature of these different categories of experimental manipulations is that they create states that signal the need to actively regulate our representations of the environment, in order to obtain better predictions and therefore achieve better adaptation. This regulation can include a suppression and replacement of current representations, or adjustments of current representations that reflect the level of predictive unreliability of the environment (Daw et al., 2005; Yu and Dayan, 2005).

COGNITIVE CONTROL

Cognitive control, also often referred to as executive function, usually denotes a category of processes that are implemented when automatic schemata are not sufficient for successful adaptation to the environment. These processes tend to be conscious[2], attention-demanding, and they involve a flexible coordination of several cognitive processes in order to attain a specific goal (Atkinson and Shiffrin, 1968; Baddeley, 1986, 2003; Norman and Shallice, 1986; Baddeley and Della Sala, 1996; Miller and Cohen, 2001; Braver et al., 2007).

For instance, experienced car drivers utilize a number of overtrained automatic schemata on a daily basis (e.g., how to negotiate roundabouts and crossroads, how to operate the gearbox, etc.). However, if US or continental European drivers have to drive in a right-hand drive country (e.g., UK or Australia), then these automatic schemata are no longer adapted and a series of conscious, attention-demanding processes have to be implemented on a sustained basis. The driver will have to actively inhibit previously learned automatic behaviors and might have to consistently maintain the new traffic rules that have to be followed in working memory (WM). This example illustrates a typical implementation of at least two canonical cognitive control processes: inhibition of dominant responses and active maintenance of relevant information in WM stores (Miyake, 2001; Braver and Ruge, 2006). It is easy to imagine other cognitive control tasks that these drivers might have to carry out: dual tasks, controlled encoding, and retrieval from episodic memory of new driving rules, updating of rules in WM, etc.

Research on cognitive control has flourished in the last 25 years, and enormous progress has been attained in characterizing different subprocesses or dimensions of cognitive control (Miyake, 2001; Braver et al., 2007), as well as their neural substrates (e.g., Botvinick et al., 2001; Miller and Cohen, 2001; Braver and Ruge, 2006; Sakai, 2008). The question of what "triggers" cognitive control implementation, i.e., what determines that a system has to switch from an automatic toward a controlled processing mode, has remained for a long time unexplored and relegated to a "black box" (Baddeley, 2003). However, a relatively recent trend of research has explored the hypothesis that specific systems are devoted to the detection of the "need for control." Specifically, a number of models have suggested the existence of brain systems devoted to monitoring the need for control and responsible for sending relevant "trigger" signals to other systems responsible for implementing control processes (Botvinick et al., 2001; Davis and Whalen, 2001; Sander et al., 2003; Kerns et al., 2004; Schaefer et al., 2006; Yeung and Cohen, 2006; Schaefer and Gray, 2007). A number of neuroimaging studies have provided clues on the neural network involved in these functions [see below the discussion about the anterior cingulate cortex (ACC), orbital frontal cortex (OFC), and amygdala].

Several hypotheses have been provided regarding the exact contextual conditions that would signal the need for control: detection of errors (Yeung and Cohen, 2006; Braver et al., 2007), changes in the motivational value (or goal-relevance) of the environment (Sander et al., 2003; Schaefer et al., 2006; Baddeley, 2007), conflict between competing responses (Botvinick et al., 2001), and changes in the prediction of error likelihood (Brown and Braver, 2005).

[2]However, there is an ongoing debate on whether cognitive control is necessarily conscious (e.g., Sumner and Husain, 2008; Van Gaal et al., 2010, 2011).

Importantly, the common feature of these hypotheses is a conflict between a current representation used to guide behavior (and often accessed in an automatic mode) and environmental conditions in which expected outcomes are unlikely to be optimally achieved.

POINTS OF INTERSECTION BETWEEN UNCERTAINTY AND COGNITIVE CONTROL

From the considerations above, it becomes clear that the constructs of uncertainty and cognitive control have at least two fundamental points of intersection:

(1) The definition of uncertainty has a remarkable similarity with the conditions usually thought to signal the need for control. In both cases, there is a *mismatch* between available schemata and the actual environment that leads to suboptimal outcomes (errors, conflict between responses, cognitive cost etc.).
(2) This mismatch is a signal that demands the implementation of processes that will actively adjust the representations guiding behavior so that a more efficient adaptation can be achieved.

Despite these similarities, it may be the case that humans do not need cognitive control to resolve uncertainty. It is entirely conceivable that implicit statistical learning could be sufficient to achieve a successful adaptation to many forms of uncertainty. We will hereafter review available research that can potentially provide clues regarding the possibility that cognitive control is recruited during uncertain decision-making.

FUNCTIONAL NEUROIMAGING OF UNCERTAINTY: EVIDENCE OF OVERLAPPING PATTERNS OF BRAIN ACTIVITY BETWEEN DECISION-MAKING UNDER UNCERTAINTY AND COGNITIVE CONTROL TASKS

Neuroimaging studies using mainly functional magnetic resonance imaging (fMRI) have provided one of the most prolific bodies of scientific research on uncertainty in recent years. Available evidence suggests that a distributed network of brain areas is systematically involved in tasks manipulating uncertainty. Next, we examine some of the key components of this network and its overlap with the network of brain areas known to be associated to cognitive control tasks.

DORSOLATERAL PREFRONTAL CORTEX

Traditionally, the dorsolateral prefrontal cortex (DLPFC) is thought to play a crucial role in WM and in the active maintenance of current task goals (Goldman-Rakic, 1995). Extensive neuroimaging and neuropsychological evidence shows that the DLPFC is strongly implicated in cognitive control tasks. For instance, it is involved in the retention of information during WM tasks and may store active representations that are necessary for adaptive decision-making performance (see Curtis and D'Esposito, 2003).The DLPFC has also been implicated in response selection in the presence of interfering or conflicting information (Rowe et al., 2000; Botvinick et al., 2001; Hadland et al., 2001; Schumacher and D'Esposito, 2002; Schumacher et al., 2003), control adjustments (Durston et al., 2003), the formulation and execution of plans of action (Fuster et al., 2000), top-down attentional control (Milham et al., 2003), and in maintaining attentional demands of the task (MacDonald et al., 2000). In addition, the DLPFC is also involved in task switching (Hyafil et al., 2009; Savine and Braver, 2010). Overall, several studies and models suggest that the DLPFC is involved in the implementation of control processes in order to resolve conflict, in contrast with other areas thought to be involved in the detection of conflict (Carter and Van Veen, 2007). This interpretation would be consistent with the fact that the DLPFC has preferential connections with motor system structures which may be central to how the PFC exerts control over behavior (Miller and Cohen, 2001). A wealth of additional studies have explored the links between the DLPFC and cognitive control, and reviewing them would be beyond the scope of this article. We refer the readers to authoritative reviews on this topic for more details (Miller and Cohen, 2001; Braver and Ruge, 2006; Carter and Van Veen, 2007; Mansouri et al., 2009).

Recent research suggests that the DLPFC may also play an important role in decision-making under uncertainty. Particularly, Huettel et al. (2005) found increased hemodynamic activity in the DLPFC during decision-making under uncertainty. The authors presented participants with eight different types of stimuli (varying amounts of triangles and circles). Participants were asked to make high or low confidence judgments about the likelihood of the feedback stimulus being the same as their selection. If the stimuli included all triangles or all circles, the chance of feedback stimuli having the same shape would be 80%, thus the uncertainty would be low at 20%. These authors varied the proportion of shapes presented as stimuli so that selections could be made in 20, 27.5, 35, 42.5, or 50% uncertainty. As uncertainty increased, so did activation in the DLPFC; which they suggest reflects the DLPFC's role in uncertainty resolution in the form of short-term modification of stimulus–response contingencies, a process that might rely on high level cognitive control processes (Huettel et al., 2005). These representations of stimulus–response contingencies are thought to guide our everyday decisions through underlying rules in which we learn that a specific association between a stimulus (S) and a response (R) is linked with a positive or negative outcome (O) (De Wit and Dickinson, 2009). Interestingly, evidence from probabilistic learning tasks such as the weather prediction task (Gluck and Bower, 1988; Gluck et al., 2002) suggests that the involvement of the DLPFC in probabilistic learning might reflect implicit, non-declarative processes rather than conscious control processes (Poldrack et al., 1999; Weickert et al., 2002). However, recent evidence indicates that the DLPFC is not necessary to non-declarative learning in these tasks (Rushby et al., 2011).

Overall, these findings suggest a role for the DLPFC in modifying S–R–O contingencies to enable adaptive decision-making in the face of uncertainty and that activation in the DLPFC is dependent on predictability of S–R–O contingencies (Paulus et al., 2002).Therefore, predictability and thus certainty, may be represented in the DLPFC through maintaining representations of the reinforcement history (previously encountered S–R–O rules) to select an "optimal strategy" (Paulus et al., 2002). It could therefore be speculated that the DLPFC is involved in the active maintenance and manipulation in WM of a history of past contingencies in order to facilitate the prediction of future outcomes. Nevertheless, there is some inconsistency across studies with evidence

indicating that the DLPFC activation increases as decision stimuli become more unexpected (Huettel et al., 2002) and others that suggest that the DLPFC is more active during low error rates, i.e., greater predictability (Paulus et al., 2002).

POSTERIOR PARIETAL CORTEX

Parietal areas and the posterior parietal cortex (PPC) in particular have often been associated to cognitive control tasks (Braver and Ruge, 2006). The PPC has been implicated in an array of cognitive control processes including task switching (Sohn et al., 2000) and movement planning (Snyder et al., 1997). The PPC is also thought to be important for sensory–motor integration that might encode movement intentions (Andersen and Buneo, 2002). Indeed, neurons within the PPC of non-human primates provide codes for decision variables such as probability and expected value when making a decision between potential movement intentions (Platt and Glimcher, 1999).

Along with the DLPFC, Huettel et al. (2005) also found that the resolution of uncertainty was linked with the PPC. These authors suggest that the PPC may also be involved in short-term response selection processes such as modification of S–R–O contingencies during uncertainty. More long term resolution of uncertainty may rely on S–R–O contingency development, i.e., learning adaptive S–R–O rules which might be mediated by areas of the medial frontal cortex (Huettel et al., 2005). Indeed, Volz et al. (2003, 2004) also demonstrated medial frontal cortex and PPC activation increases with increasing uncertainty. In their study, Volz et al. (2003) showed participants pairs of pictures showing comic figures which were systematically associated with a particular reward probability (either 0.6, 0.7, 0.8, 0.9, or 1.0), and these associations were consistent throughout the experiment. As uncertainty increased, posterior parietal areas known to underlie WM functions also increased. It has been suggested that this type of result could reflect the role of the PPC in actively maintaining in WM all S–R–O rules that are valid in an experiment (Bunge et al., 2002).

ANTERIOR CINGULATE CORTEX

The Anterior Cingulate Cortex (ACC) is one of the most prominent neural substrates identified in cognitive control processing (see Botvinick et al., 2001; Braver and Ruge, 2006). For instance, the ACC has been found to be involved in many classical cognitive control tasks such as the Stroop task (Pardo et al., 1990), the Flanker task (Botvinick et al., 1999), N-back task (Harvey et al., 2005), and stop-signal tasks (Brown and Braver, 2005), amongst others. Evidence also suggests that the ACC is involved in monitoring cognitive conflicts (MacDonald et al., 2000; Botvinick et al., 2001) and error detection (Ullsperger and Von Cramon, 2003). Certain electrophysiological components known to be linked to cognitive control (and to conflict monitoring in particular) are thought to be generated by ACC sources. For instance, the N2 and the Error-related negativity (ERN/Ne) event-related brain potentials (ERP) components are thought to originate in the ACC (Dehaene et al., 1994; Dikman and Allen, 2000; Gehring and Knight, 2000; Debener et al., 2005) and are likely to reflect the cognitive demands of situations involving a high level of conflict between competing responses (Yeung and Nieuwenhuis, 2009). Importantly, the ACC is widely thought to be part of a network of brain structures specialized in the detection of environmental conditions signaling the need for the implementation of cognitive control, and responsible for sending "triggers" to other systems specialized in the actual implementation of control (Botvinick et al., 2001; Kerns et al., 2004; Brown and Braver, 2005).

Activity in the ACC has also been demonstrated to correlate with levels of uncertainty (Stern et al., 2010). In their task, Stern et al. (2010) showed participants four sequential draws of cards taken from two decks. Deck A which contained 80% red cards and 20% blue cards and Deck B which contained 20% red cards and 80% blue cards. Participants had to identify the deck from which the cards had been drawn. Uncertainty was manipulated by the frequency of cards presented with the same color in the sequence of four cards presented to the participants. For instance, four blue cards was the highest certainty condition, whereas a combination of two red and two blue cards was associated to maximum uncertainty. The authors found that increased uncertainty was associated with increases in ACC activity. According to the authors, the ACC was specifically involved in "evidence accumulation" whereas the OFC was involved in the execution of the decision following the four sequential draws of cards (Stern et al., 2010). Increased activity in the ACC has also been found to be related to outcome uncertainty and uncertainty-related physiological arousal (Critchley et al., 2001). In their study, Critchley et al. (2001) gave participants a two choice decision-making task, in which a cue (a card) was predictive of the value of a "feedback card." Participants had to decide from the initial card if the feedback card would be higher or lower than the initial card. Uncertainty was manipulated by the degree to which the initial card was a reliable predictor of the feedback card. The participants then experienced a delay period before the outcome (gain/loss) was presented. Critchley et al. (2001) found that the ACC was modulated by both outcome uncertainty and anticipatory delay. In addition, the authors found a distinct region of the ACC that was commonly modulated by both uncertainty and physiological arousal.

The ACC has also been associated with hypothesis testing in uncertain contexts (Elliott and Dolan, 1998). Specifically, Elliott and Dolan (1998) presented participants with a series of pairs of checkerboards with different spatial configurations of black and white squares in which participants were asked to try to work out a rule governing which of each pair was correct (feedback was set at an uncertain 50% correct level). The task requirement was to generate a hypothesis (i.e., top left-hand corner filled) and then test and update it based on information about which checkerboard was correct. Hypothesis testing and making a choice was associated with different foci within the anterior cingulate. Whilst the dorsal ACC was involved in complex hypothesis testing, the ventral ACC was associated with implementing a choice. This suggests different roles of the ACC in purely cognitive aspects of processing (hypothesis testing) and evaluative processes related to the emotional consequences of a choice (Elliott and Dolan, 1998.) Indeed, the ventral region of the ACC is interconnected with limbic regions such as the amygdala which is widely thought to underlie emotional processes (Bracht et al., 2009).

Finally, the ACC has also been linked to volatility, i.e., uncertainty created by frequent changes in S–R–O contingencies (Behrens et al., 2007). In Behrens et al.'s (2007) study, subjects

carried out a one-armed bandit task in which they had to choose between blue and green stimuli. Subjects underwent trials where the probability of a blue outcome was 75% (a certain/stable environment) and trials where reward probabilities switched between 80% blue and 80% green every 30 or 40 trials (an uncertain/volatile environment). Behrens et al.'s (2007) results suggest that ACC activity might reflect a Bayesian estimate of the environment's volatility during a monitoring stage, i.e., when outcomes are being evaluated in order to regulate current beliefs about the underlying stimulus–response contingencies of the environment. The model of Behrens et al. (2007) also suggests that the ACC might encode how much influence feedback should give to subsequent decisions, with more recent outcomes being more salient in uncertain contexts (Rushworth and Behrens, 2008).

The available findings about the involvement of the ACC in decision-making under uncertainty and in cognitive control tasks provide an interesting parallel. On the one hand, studies and models from the realm of cognitive control research suggest that the ACC might be involved in the detection of the contextual conditions signaling the need for control. On the other hand, data from the field of decision-making processes suggest that the ACC might be involved in the computation of the level of uncertainty of current SRO representations (Behrens et al., 2007). An integration of these findings could tentatively suggest that both traditions of research are tapping overlapping processes, in the sense that uncertainty could be easily seen as a condition signaling the need for control. However, results from the field of decision-making under uncertainty (and in particular the study from Behrens et al., 2007) might be providing a more formal description of the computational nature of one of the potential mechanisms that can trigger the implementation of cognitive control. Nevertheless, at this stage, these considerations remain hypothetical, as there is to our knowledge no evidence that clearly demonstrates that the estimation of uncertainty, as described by Behrens et al. (2007), necessarily lead to the implementation of cognitive control, or that the adaptation to uncertainty in these studies relied on processes other than implicit learning.

ORBITO-FRONTAL CORTEX

The Orbito-Frontal Cortex (OFC) has classically been linked to the perception of reward and punishment feedback, including anticipation and receipt of feedback, and is usually thought to be involved in the representation of stimulus–reward relationships (Rolls, 1996; O'Doherty et al., 2003). The OFC has also become a brain area of interest for cognitive control when it was discovered that this region not only detects valenced outcomes but it also probably uses this information to bias future behavioral choices (O'Doherty et al., 2001, 2003). Therefore, it has been suggested that the OFC might also be involved in processing environmental conditions signaling the need for behavioral control (O'Doherty et al., 2003; Braver and Ruge, 2006).

More recently, the OFC has also been shown to differentiate between different levels of uncertainty (Hsu et al., 2005). In this study, Hsu et al. (2005) manipulated uncertainty by creating situations in which participants had to make choices under ambiguity. For instance, one of the conditions included placing a bet on the color of a card (e.g., blue or red) to be drawn from a deck without knowing the actual proportions of blue and red cards of the deck. In control conditions, relevant information was available prior to the choice (i.e., participants were told the exact proportions of red and blue cards in the deck). They found that the level of uncertainty correlated with activation of the OFC and the amygdala. These authors suggest that a network involving the OFC and the amygdala would reflect the operations of a "vigilance"/evaluation system dedicated to evaluate uncertainty and bias behavioral adaptation as a function of uncertainty levels. They further support this idea through a behavioral study on patients with OFC lesions, finding that OFC lesions impair the ability to distinguish between degrees of uncertainty (Hsu et al., 2005).

Related results suggest that the OFC enables acquisition of S–R–O contingencies, whereby patients with OFC lesions show deficits in shifting behavior when the underlying S–R–O rule is volatile (Rolls, 2000). This is consistent with data showing that OFC activation is associated with hypothesis generation of S–R–O rules (Goel and Dolan, 2000) and the detection of change in reward contingencies (O'Doherty et al., 2003). In addition, a number of other studies have also shown that brain activity in the OFC increases with increasing outcome uncertainty (Critchley et al., 2001) and reward uncertainty (Tobler et al., 2006). Finally, activity in the OFC also increases as the probabilistic stimulus–response contingencies become more complex, suggesting that the OFC may reflect additional requirements of dealing with uncertainty (Elliott et al., 1999). However, Stern et al. (2010) suggest that activation of the OFC by uncertainty is absent when there is no risk, suggesting that the OFC does not detect cognitive uncertainty *per se*, but instead responds to uncertainty only when there are incentive consequences.

In summary, available research clearly shows that the OFC is sensitive to several types of manipulation of uncertainty. It is unclear at this stage as to what specific role the OFC plays in the processing of uncertainty. However, research in the field of cognitive control would indicate that the OFC might be involved in tracking valenced information signaling the need for behavioral control. It could therefore be speculated that the OFC detects several forms of outcome uncertainty because it equates them to motivationally relevant contextual cues reflecting the need for control.

AMYGDALA

The amygdala is widely known to be associated to a series of emotional processes, and it has been famously associated to fear and anxiety (LeDoux, 1996), although it is now known that the amygdala is also involved in a wider array of affective processes including positive affect (Zald, 2003). A recent research trend has also found evidence that the amygdala plays an important role in cognitive control. For instance, Schaefer et al. (2006) have shown that increases in amygdala activity significantly predicted an improvement of performance in a canonical executive task, the N-back WM task, across two different experiments. More recent studies have found similar evidence of a relationship between the amygdala and cognitive control processes (Nishijo et al., 2008; Ousdal et al., 2008; Yun et al., 2010; Chiew and Braver, 2011), which adds to earlier evidence in both human and animal research (for a review, see Schaefer and Gray, 2007). A common theory to account for

the role of the amygdala in cognitive control posits that the amygdala is responsible for evaluating changes in the motivational value (or goal-relevance) of the environment, and when these changes exceed a certain threshold, biasing signals are sent to cognitive and motor systems in order to facilitate adaptation (Davis and Whalen, 2001; Sander et al., 2003; Schaefer et al., 2006; Schaefer and Gray, 2007). It is thought that this mechanism can be involved in the implementation of cognitive control, but also in the generation of emotional responses (Schaefer et al., 2006).

As indicated by the findings of Hsu et al. (2005) described above, the amygdala also appears to be linked to the evaluation of contextual uncertainty. Similar conclusions have been reached in animal studies (for a review, see Rosen and Donley, 2006). In particular, Rosen and Donley (2006) suggest that the amygdala may be particularly linked to uncertain situations characterized by unexpected changes in the environment, which would relate to the concept of unexpected uncertainty (Yu and Dayan, 2005), and to the construct of rule volatility (Behrens et al., 2007). The amygdala is also activated under internally driven uncertainty, i.e., self reported levels of uncertainty (Zaretsky et al., 2010), which suggests that the amygdala is activated when sufficient data regarding a potential threat in the surroundings is lacking (Whalen et al., 1998; Zaretsky et al., 2010). For instance, Zaretsky et al. (2010) asked participants to report their perceived certainty regarding the level of danger associated to ambiguous face stimuli. They found that amygdala activity increases were associated with an increase in perceived uncertainty. Consistent with this idea, Davis and Whalen (2001) have suggested a model in which the amygdala is responsible for increasing levels of vigilance in response to uncertain stimuli or situations. This increase in vigilance levels would bias a series of control systems which would in turn facilitate general adaptation to the initial uncertain context.

The similarities between models of the role of the amygdala in uncertainty and models of the role of the amygdala in cognitive control are obvious. In both cases, fundamental changes in the goal-relevance of a situation are thought to trigger a response that signals the need to implement a controlled adaptive response. This mechanism shares similarities with the role often attributed to the ACC in cognitive control and reinforces the thesis that uncertainty plays a significant potential role in "triggering" the implementation of cognitive control.

UNCERTAINTY AND COGNITIVE CONTROL

Whilst many of the aforementioned studies have shown overlaps between regions implicated in uncertainty processing and cognitive control, a recent study by Bhanji et al. (2010) attempted to investigate the dissociations between these two processes. In this study, the researchers manipulated task difficulty, assumed to index the recruitment of cognitive control resources and choice certainty. For each trial, participants were presented with a cue (containing numbers) and had to predict whether a feedback screen would include a number higher or lower than the cue. Outcomes ranged from small ($0.2) to large ($1) gains or losses for correct and incorrect answers respectively. In "Gambling" blocks, participants had to "guess" the value of the feedback. Uncertainty was manipulated through biasing the probabilities of positive/negative outcomes linked to specific number cues. Low choice certainty included cues of 2, 3, 8, and 9 whilst high certainty included cues 4, 5, 6, and 7. In "Rule" blocks, participants had to estimate the value of the feedback following previously learned rules that could be simple or complex. The authors found that the amygdala and the ventromedial prefrontal cortex were less active in choice uncertainty compared to choice certainty, and the insula was more involved in uncertainty compared to certainty. Next, the authors also found that the lateral PFC and ACC areas were related to rule complexity. While this study is a worthwhile attempt to differentiate uncertainty from processes related to cognitive control, some of its results can be explained by an alternative account. For instance, in this study, choice certainty was intrinsically linked with a higher frequency of trial-to-trial positive outcomes (monetary gains). Therefore, the involvement of the amygdala and VMPFC in choice certainty could well be the result of an anticipation of reward, as acknowledged by the authors.

In summary, available evidence from functional neuroimaging shows a strong overlap between the neural networks of uncertainty processing and cognitive control. Specifically, uncertain contexts appear to activate a typical "cognitive control network" including lateral PFC areas, parietal areas and the ACC. In addition, uncertainty is also linked to brain systems thought to be responsible for monitoring the need to implement top-down control (ACC and amygdala). A tentative explanation could posit that different forms of uncertainty share common features that monitoring systems (probably linked to the ACC and the amygdala) are programmed to detect and interpret as signals that top-down control needs to be implemented. Next, biasing signals could be sent to other systems more directly linked to the actual implementation of cognitive control processes (e.g., WM, inhibition, etc.). This explanation would be compatible with existing models of the dynamics of cognitive control and monitoring processes (Botvinick et al., 2001; Yeung and Cohen, 2006), as well as with current models of amygdala function (Davis and Whalen, 2001; Sander et al., 2003; Schaefer et al., 2006; Ousdal et al., 2008). In the next section, we discuss in more detail the potential links between uncertainty and monitoring processes.

UNCERTAINTY AND MONITORING

As suggested in the previous sections, uncertainty might play a role in a key component of cognitive control – monitoring processes. Monitoring usually refers to a set of processes that evaluate the need to implement or adjust top-down control, and this process is often defined within the scope of the specific monitoring of the outcomes of ongoing behavior (Botvinick et al., 2001; Kerns et al., 2004; Yeung and Cohen, 2006; Brown, 2009). In this section we review evidence supporting the idea of a relationship between decision-making under uncertainty and monitoring processes.

First, relevant evidence can be found in the field of complex dynamic control (CDC) tasks. CDC tasks are complex tasks often enacting realistic scenarios with frequent applications in ergonomics and human factors (e.g., flight and driving simulators, virtual markets, virtual problem solving such as forest fire-fight scenarios, etc.). Evidence from CDC tasks suggests that uncertainty is associated to an increase in the frequency of monitoring behaviors and in the amount of attention allocated to the monitoring of decision outcomes (Osman, 2010). For instance, Mosier et al.

(2007) investigated the performance of airline pilots in a controlled flight simulator context. Several scenarios were used which required monitoring (visual search of decision feedback information in control panels) and control and execution (diagnosis and decision) processes. When participants were presented with incongruent or conflicting information, presented in an unpredictable manner (e.g., system failures), diagnosis took significantly longer, and pilots spent more time in monitoring behaviors than when situations and information were stable and information congruent. Similarly, Metzger and Parasuraman (2005) examined the effects of an automated system on performance of air traffic controllers in monitoring free-maneuvering aircraft across two contexts. In one context, participants were provided with a 100% reliable automated system to aid them in a conflict detection task and in the second they were provided an imperfect monitoring aid in which the reliability of the automated system was uncertain. Metzger and Parasuraman (2005) observed that under the uncertain system, traffic controllers had a greater likelihood of detecting conflicts, which can indicate that greater attentional resources were allocated to conflict monitoring when the context was uncertain. Consistent with this interpretation, Diez et al. (2001) measured eye tracking data from pilots during their interaction with a Boeing 747 flight simulator, and in particular during monitoring of decision outcomes (through events in the control panel). A distinction was made between information related to stable vs. uncertain aspects of the flight (for instance, altitude, position and speed are variable and uncertain). Results showed that eye fixations were longer for indicators of uncertain flight-related information compared to stable information. In addition, subsequent recall was higher for uncertain in comparison to stable information.

However, there might be an upper limit to the amount of uncertainty that can be handled by outcome monitoring (Xie and Guo, 2000). Sarter et al. (2007) measured behavioral and eye tracking data from pilots on a 1-h flight simulation under extremely challenging events, where participants experienced expected and unexpected changes in automation modes. In these highly uncertain environments, participant's fixation rates on unexpected changes were low and completely failed to monitor these changes 43% of the time in comparison to a failure rate of 29% in expected automation changes. This uncertainty monitoring bias often comes with a cost, as increasing amounts of uncertainty make it difficult to effectively process relevant feedback (Atkins et al., 2002; Patrick and James, 2004; Bredereke and Lankenau, 2005; Gao and Lee, 2006; Mosier et al., 2007).

Second, evidence of a relationship between uncertainty and monitoring processes is also provided by research using Event-related potentials (ERPs). Outcome monitoring processes are investigated in this field mainly through two brain potentials: the ERN and the feedback-related negativity (FRN; Falkenstein et al., 2000; Pailing and Segalowitz, 2004; Hajcak et al., 2006; Sailer et al., 2010).

The ERN is an early frontocentral negativity time-locked to an incorrect response (Gehring et al., 1990; Falkenstein et al., 1991) and is believed to be generated in the ACC (Dehaene et al., 1994; Dikman and Allen, 2000; Gehring and Knight, 2000). Competing theories postulate that the ERN reflects the activity of a generic error detection (Dehaene et al., 1994) or a response conflict system (Carter et al., 1998). Whilst errors are thought to be processed differently to correct responses (Gehring and Knight, 2000), it has been suggested that uncertainty may cause errors to go undetected because of misrepresentation of the correct response (Pailing and Segalowitz, 2004) and thus may reduce the amplitude of the ERN (Falkenstein et al., 2000; Coles et al., 2001). In a study by Scheffers and Coles (2000), participants completed a two choice reaction time task under degraded stimulus conditions, designed to induce stimulus uncertainty. After response to each trial, participants were asked to rate their perceived accuracy on the trial. Results showed that errors due to premature responses (where errors were accurately identified as errors immediately after the response) had large ERN amplitudes; however, errors caused by stimulus uncertainty (errors where there was uncertainty about the error) had a reduction in ERN amplitude. Similar conclusions were reached by Pailing and Segalowitz (2004), who used letter and tone discrimination tasks in which uncertainty was manipulated by varying the amount of information provided by pre-stimulus cues. Results showed a significant decrease in the ERN amplitude for uncertain conditions. Importantly, this effect was enhanced in conditions in which attention was depleted by a concurrent task. These results suggest that under conditions of increased uncertainty about performance, errors, and correct trials are more likely to be judged or perceived in a similar manner.

The FRN is an early negativity time-locked to the delivery of feedback in decision-making paradigms. The FRN is typically larger following feedback signaling negative outcomes (e.g., "losses") compared to positive outcomes ("wins"; Miltner et al., 1997; Gehring and Willoughby, 2002; Holroyd and Coles, 2002; Holroyd et al., 2003; Yeung and Sanfey, 2004; Nieuwenhuis et al., 2005; Hajcak et al., 2006). However, more recent studies have shown that feedback of positive valence can also elicit FRN (Yeung and Sanfey, 2004; Hajcak et al., 2005; Müller et al., 2005; Yeung et al., 2005). It has been suggested that the FRN reflects a general performance monitoring system that is activated by violations in expectancy (Nieuwenhuis et al., 2004; Oliveira et al., 2007).

The FRN has also been implicated in the processing of outcome certainty (Hajcak et al., 2005; Cohen et al., 2007). For instance, Cohen et al. (2007) used a probabilistic reinforcement task to investigate the effect of outcome probabilities on the FRN. In this study, uncertainty was operationalized through the manipulation of reward probabilities across blocks in a two choice decision task. High uncertainty in win trials (where reward probability was 25%) resulted in larger and more sustained FRN amplitude, in comparison to more certain (50 and 75% reward probability) positive outcomes. Similar results on FRN amplitude come from a recent study by Moser and Simons (2009). Participants performed a two choice gambling task in which they were asked to predict the outcome of their choice prior to selection and after selection. Here, FRN amplitude was highest when participants were uncertain of the outcome and changed their mind from an initial prediction of "lose" to post choice prediction of "win" (see also Sailer et al., 2010 for similar conclusions). In addition, the FRN may also be related to control processes, with research suggesting that this component reflects the activity of a reinforcement learning system which is used to adjust subsequent behavior (Hajcak et al., 2006; Cohen et al., 2007) and is also linked with attentional mechanisms (Moser and Simons, 2009) and WM demands (Suchan et al., 2005).

In summary, reviewed evidence from CDC tasks and electrophysiological research suggests strong links between uncertainty and monitoring processes: uncertainty increases the frequency of monitoring behaviors and the amount of attention allocated to outcome monitoring. Next, uncertainty also modulates brain potentials reflecting the processing of decision outcomes: uncertainty is associated to larger FRNs and reduced ERNs.

INTOLERANCE OF UNCERTAINTY AND PSYCHOPATHOLOGY

INTOLERANCE OF UNCERTAINTY

It is widely acknowledged that psychiatric populations exhibit dysfunctional decision-making (Ladouceur et al., 2000). Interestingly, it is now becoming apparent that psychiatric disorders can also be linked with deficits in coping with uncertainty. It has been suggested that an inability to cope with uncertainty may even act as a driving force behind a number of behaviors and cognitions (e.g., worry, obsessions, compulsions, hypervigilance) associated with various anxiety disorders (Holaway et al., 2006). IOU is a construct defined as "*a tendency to react negatively on an emotional, cognitive, and behavioral level to uncertain situations and events*" (Heimberg et al., 2004, p. 143). IOU has been demonstrated in general anxiety disorder (GAD; Garber et al., 1980; Andrews and Borkovec, 1988; Dugas et al., 1998; Covin et al., 2008; Simmons et al., 2008; Boelen and Reijntjes, 2009), obsessive–compulsive disorder (OCD; Steketee et al., 1998), Schizophrenia (Broome et al., 2007; Dudley et al., 2011), and Eating Disorders (Konstantellou and Reynolds, 2010; Sternheim et al., 2011). Here, we will review the evidence that demonstrates an important role for IOU in a number of disorders known to be associated with deficits in cognitive control tasks.

GENERAL ANXIETY DISORDER

General anxiety disorder is a disorder characterized by excessive and persistent worrying (Dugas et al., 1998) and has been used to highlight the importance of uncertainty in psychiatric disorders (Paulus, 2007). IOU has been postulated to play a central role in anxiety (Krohne, 1989). For instance, measures of IOU can differentiate between clinical and healthy populations in GAD (Buhr and Dugas, 2002) and can predict changes in anxious symptoms (Dugas et al., 2009). Importantly, high worriers show a clear deficit in decision-making under uncertain situations with longer reaction times (see Ladouceur et al., 2000) and more information seeking behavior (Gibbs-Gallagher et al., 2003). Further, IOU has been associated with greater recall and threatening interpretations of ambiguous information (Dugas et al., 2005) and greater attempts to reduce uncertainty (Ladouceur et al., 2000). Worriers take longer to make category judgments than non-worriers, particularly when confronted with uncertain (ambiguous) stimuli (Metzger et al., 1990; Tallis et al., 1991). Raghunathan and Pham (1999) found that anxious decision makers preferred to choose high probability rewards linked to low monetary gains instead of more infrequent but much larger gains. This finding illustrates the tendency of anxious individuals to opt for safer and less uncertain choices, even at a significant cost.

SCHIZOPHRENIA

Patients with psychosis, or at risk of psychosis, display a difficulty in tolerating uncertainty (e.g., Broome et al., 2007). Strauss et al. (2011) claim that reduced reward-seeking behavior in Schizophrenia is critically related to the extent to which patients make exploratory choices when they are uncertain. "Jumping to conclusions" (JTC), a frequent symptom in schizophrenia, has also been found to be associated with the construct of IOU (Freeman et al., 2008). Moritz et al. (2011) have suggested the idea that JTC in schizophrenic patients may contribute to well-being since quick decision-making decreases doubt and uncertainty. Indeed, JTC may be more evident in individuals who find it difficult to tolerate ambiguity (Colbert and Peters, 2002). This suggests a need to reduce uncertainty in schizophrenia, similar to that seen in other psychopathologies. Interestingly, a similar phenomenon is associated to IOU in GAD individuals. High IOU was indeed associated with faster decision-making in anxious individuals when subjects were faced with an outcome delay and thus waiting in a state of uncertainty (Luhmann et al., 2011). Further, an important theory of uncertainty processing (Yu and Dayan, 2005), has been linked to delusions and hallucinations associated with schizophrenia. Specifically, Patel et al. (2010) have suggested that hallucinations may be linked to neurotransmitters involved in the interaction between top-down and bottom-up processing where hallucinations could be experienced due to over-processing of top-down signals resulting in uncertainty in information processing.

OBSESSIVE–COMPULSIVE DISORDER

One of the characteristics of OCD is pathological doubt, which is most clearly evident among patients with checking rituals (Rachman and Hodgson, 1980; Rasmussen and Eisen, 1992). Interestingly, uncertainty extends to long term memory of past events, as OCD individuals tend to be uncertain about their memory for checked events (Van Den Hout and Kindt, 2004). More directly, Tolin et al. (2003) found that repeating rituals were associated with IOU. IOU in OCD patients may reflect the belief on the part of OCD patients that they lack sufficient coping or problem solving skills to effectively manage threatening situations that have the potential to evoke discomfort and negative emotionality (Holaway et al., 2006). Many of the traits associated with OCD can be viewed as the products of control exerted within the emotional, interpersonal, cognitive, and behavioral domains (Gibbs-Gallagher et al., 2003). Difficulty in decision-making in OCD is characterized by a cautious approach to information categorization, frequent requests for information repetition, and doubts about the correctness of decisions (Obsessive Compulsive Cognitions Working Group, 1997).

COGNITIVE CONTROL AND PSYCHOPATHOLOGY

Importantly, patients with these psychopathological disorders not only show an IOU but also exhibit deficits in classical cognitive control tasks. For instance, OCD patients usually show an impaired performance on tests of executive function. Indeed, OCD patients show performance deficits on measures of response inhibition (Aycicegi et al., 2003). Particularly, OCD subjects exhibit higher interference costs in the Stroop task (Hartston and Swerdlow, 1999), making significantly more errors and slower reaction times on the interference trials of the Stroop test (Martinot et al., 1990; Bannon et al., 2002). It has been suggested that OCD subjects

exhibit deficits in behavioral and cognitive inhibition (Enright and Beech, 1990; Rosenberg et al., 1997), which might underlie the repetitive symptomatic behaviors of the disorder, such as compulsions and obsessions (Bannon et al., 2002). Deficits among patients with OCD may also be associated with co-morbidity with depressive symptoms and schizotypal personality features (Aycicegi et al., 2003). Schizophrenia is also usually characterized by deficits in executive function (Liddle and Morris, 1991). For instance, schizophrenic patients perform worse in NoGo conditions in a Go/NoGo tasks suggesting a failure of response inhibition in schizophrenic patients (Weisbrod et al., 2000). Deficits in cognitive control have also been observed in patients with high anxiety (Wood et al., 2001). For instance, these deficits include dysfunctional response inhibition (Gernsbacher et al., 1990) with more attention paid to threatening distracters in the Stroop task (Mathews et al., 1990). High levels of anxiety have also been shown to limit WM capacity (Eysenck and Calvo, 1992).

NEURAL PROCESSING OF UNCERTAINTY IN PATIENTS WITH AFFECTIVE DISORDERS

Together, these results imply that disorders such as GAD, OCD, and Schizophrenia show deficits in both uncertainty processing and cognitive control. In addition, these abnormalities are coupled with dysfunctional neural processing. Indeed, hyperactivity of frontal–amygdala limbic regions suggests that greater IOU is associated with an elevated affective response to uncertainty (Krain et al., 2008). Here, we will review neurological evidence for deficits in neural substrates that are known to underlie cognitive control and uncertainty processing.

Schizophrenic patients have been shown to activate frontal areas insufficiently (Berman et al., 1992) with patients showing deficits in response inhibition that are coupled with disruption of the frontal P300 ERP amplitude, which may also indicate dysfunction of frontal neuronal circuits (Weisbrod et al., 2000). Interestingly, a fronto-parietal dysregulation has been observed in schizophrenic patients where the assessment of uncertainty is linked to decreased activation of the medial prefrontal cortex and an increased activation in the parietal cortex (Paulus et al., 2002). Further, schizophrenic patients show a deficit in reward-related probabilistic trial-and-error learning (Koch et al., 2010). This impairment was associated with the inability to reduce processing resources in association with increasing predictability, suggesting that patients exhibit a deficit in neural processing in response to uncertainty. Indeed, patients showed altered activation patterns in mainly frontal, cingulate, and striatal brain areas; regions known to be involved in uncertainty processing in healthy volunteers during feedback- or reward-based probabilistic learning (Fiorillo et al., 2003; Koch et al., 2008; Schlosser et al., 2009). These regions are also strongly implicated in decision-making, performance monitoring, and cognitive control (Volz et al., 2003; Zysset et al., 2006; Koch and Preuschoff, 2007; Koch et al., 2008).

General anxiety disorder subjects show increased activation in the ACC in response to infrequent errors, suggesting the propensity to be more engaged in error-related processing, which might then increase anticipation of adverse outcomes and contribute to fearfulness and avoidance of future conflict processing (Paulus et al., 2004). Indeed, hyperactivity in the ACC, which is chiefly involved in both uncertainty and cognitive control (see previous sections), may be a key feature of anxiety disorders (Paulus, 2007). Further, dysfunctional ACC activity has been observed during performance of an emotion-word Stroop task in anxious individuals (Engels et al., 2007) and with post-traumatic stress disorder (PTSD; Bremner et al., 2004; Etkin and Wager, 2007). Indeed, the degree of activity in the dorsal ("cognitive") region of the ACC has been said to be a predictor of the level of self reported negative affect across individuals during reappraisal (Ochsner et al., 2002). These findings suggest that cognitive control regions play an important role in reappraisal and controlling emotional responses (Banich et al., 2009).

In addition, GAD patients high in IOU have shown less activation in fronto-median areas (Schienle et al., 2010), a region thought to be involved in uncertainty processing (Volz et al., 2003). Schienle et al. (2010) have suggested that this finding might reflect a potential deficit of systems devoted to cognitive coping and preparatory actions in individuals who suffer from IOU (Schienle et al., 2010).

Finally, OCD symptoms are said to be caused by abnormalities in fronto-striatal circuitry (Insel, 1992). Particularly, OCD is thought to be linked to a dysfunction in the circuits connecting the basal ganglia to the OFC, which would produce a number of cognitive and motor abnormalities (Wise et al., 1989; Alexander et al., 1990). The OFC has directly been implicated in patients with OCD (Rauch et al., 1994, 2002). As discussed previously, the OFC is known to be involved in certain aspects of cognitive control and it also appears to be involved in uncertainty processing (Hsu et al., 2005). It has been proposed that the deficit of OCD patients in the OFC might reflect an altered processing of reward history and valuation of options due to a relative disconnection between the dorsolateral, orbitofrontal, and anterior cingulate cortices with limbic regions (especially the amygdala) and with the basal ganglia (Holaway et al., 2006).

To date, relatively few studies have directly addressed the link between IOU and cognitive control in patient populations. However, a study by Broome et al. (2007) attempted to link IOU with deficits in cognitive control in individual at high risk of developing psychosis. In this study, patients were shown two jars of colored beads, informed of the relative proportions of beads in each and then told that they would be shown a series of beads drawn from one of the jars. They are then asked, on the basis of the observed sequence, to judge which jar is the source of the beads, and to be "as certain as possible." The ability to hold information about bead color online was assessed using an adaptation of the digit span task that used a string of different colored beads. The authors found that IOU was negatively correlated with WM performance (assessed by the colored beads task) and positively correlated with JTC.

In summary, reviewed evidence indicates that individual differences in how we deal with uncertainty are linked to a series of psychopathological disorders known to be associated with deficits in cognitive control. Next, IOU seems to be linked to altered patterns of brain activity in systems involved in both uncertainty and cognitive control (Schienle et al., 2010). Finally, there is evidence that IOU might also be linked to an impairment in WM, a typical sub-process of cognitive control (Broome et al., 2007).

SYNTHESIS AND CONCLUSION

The evidence reviewed in this article shows that available research provides a wealth of both indirect and direct evidence of potential and even intrinsic relationships between uncertainty and cognitive control. First, functional neuroimaging studies investigating uncertainty have uncovered a neural network that has a remarkable overlap with brain networks usually associated with cognitive control tasks. In particular, a network involving lateral PFC areas, parietal cortex, and the ACC seems to be constantly activated for decision-making tasks in which outcome uncertainty is manipulated and also in a wide range of classical cognitive control tasks. Importantly, neuroimaging studies of uncertainty have also unveiled behavioral patterns that are consistent with the recruitment of controlled processes, such as an increase of response time in uncertain conditions (an effect that had already been shown in a vast body of behavioral research, see for instance Bertelson and Boons, 1960).

Second, a number of behavioral and electrophysiological studies indicates a strong relationship between uncertainty and a key component of cognitive control – outcome monitoring. In particular, it appears that highly uncertain environments tend to increase the recruitment of monitoring processes. In addition, studies on the ERN and FRN brain potentials suggests that systems devoted to processing the valence of decision outcomes are hyper-activated during uncertainty, and that correct and incorrect decisions are more difficult to discriminate in such contexts. This pattern of effects could indicate that uncertainty is a state that triggers an enhanced activity of systems dedicated to the monitoring of the outcomes of our actions. A tentative explanation for these effects could be that when there is a realization that the outcomes of our decisions are difficult to predict, an enhanced processing of outcomes might be an adaptive solution. This strategy can potentially facilitate the accurate detection of changes in the underlying rules in the environment and lead to a more successful adaptation. Interestingly, monitoring processes are often seen as a trigger for the implementation of cognitive control processes, by providing a mechanism that evaluates the "need for control" of a given situation. In this sense, the construct of uncertainty could be seen as a summary of the contextual conditions that are the causal antecedents of the implementation of cognitive control, rather than a set of processes that would be independent from cognitive control. This explanation would be consistent with the fact that many of the overlapping activations in neuroimaging studies from the field of cognitive control and the field of decision-making under uncertainty, point toward a network of brain areas thought to be involved in the detection of contextual cues signaling the need for control (ACC, OFC, and amygdala), and in the utilization of this information to bias behavior (see the section on functional neuroimaging). Future studies will be needed to explicitly examine whether and how manipulating levels of outcome uncertainty can trigger the implementation of cognitive control.

Third, although there is evidence that uncertainty is strongly linked to the monitoring stage of cognitive control, only indirect published evidence exists of the potential role of cognitive control in the resolution of uncertainty in post-monitoring stages. The involvement of lateral PFC areas in conditions of increasing outcome uncertainty may suggest that systems devoted to the implementation of cognitive control are used to resolve uncertainty, as reviewed in the neuroimaging section. This idea is also supported by behavioral patterns observed during uncertainty that are consistent with the recruitment of cognitive control (such as increased RT during high uncertainty). Overall, the data and models reviewed in this article suggest a two-way transaction between outcome uncertainty and cognitive control: outcome uncertainty can be seen as a state that triggers the implementation of cognitive control and in turn one of the main functions of cognitive control processes would be to facilitate learning about uncertain S–R–O mappings. It is important to stress that this possibility does not rule out that implicit learning processes also play a role in the adaptation to uncertain environments. Further research will be needed to investigate the extent to which cognitive control contributes to the resolution of uncertainty above and beyond implicit learning processes. For instance, future research should focus on paradigms in which uncertain decision-making is combined with concurrent tasks tapping cognitive control (e.g., Bhanji et al., 2010, provide an example of such paradigms).

Fourth, an emerging literature in psychopathology indicates that individual differences in IOU are linked to a series of affective disorders known to be associated with deficits in cognitive control, such as OCD and GAD. In addition, some evidence (Broome et al., 2007) indicates that IOU is also correlated with lower performance in cognitive control tasks. This body of evidence tentatively suggests that a number of affective disorders may be characterized by an inability to adapt to uncertainty, and that this deficit might be linked to lower efficiency in cognitive control function. However, further research will be needed to characterize more precisely the potential causal links between uncertainty, cognitive control and affective disorders. In addition, an interesting potential area of development in the field of IOU could be the development of cognitive training techniques targeted at decision-making under uncertainty. Cognitive and attentional training techniques seem to be providing promising results in clinical populations (See et al., 2009). Testing training schedules for clinical populations aimed at using cognitive control in order to cope more effectively with uncertainty might inform us about the potential causal links between IOU, uncertainty and cognitive control.

Finally, it has to be noted that the fundamental distinction between expected and unexpected uncertainty does not appear to have led to systematic differences in brain activity. Paradigms tapping these two concepts (e.g., volatility studies for unexpected uncertainty and reward probability manipulations/ambiguity studies for expected uncertainty) have been associated to approximately similar brain networks in functional imaging studies. Future research will be necessary to refine the techniques used to investigate the neural correlates of these two types of uncertainty, and also to specify further the taxonomy of different forms of uncertainty.

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A test for the implementation–maintenance model of reappraisal

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Reappraisal has been defined as a conscious, deliberate change in the way an emotional stimulus is interpreted, initiated in order to change its emotion-eliciting character (Gross, 2002). Reappraisal can be used to down-regulate negative emotions, including anxiety (reviewed in Kalisch, 2009). There is currently a strong interest in identifying the cognitive processes and neural substrates that mediate reappraisal. We have recently proposed a model (termed implementation–maintenance model or IMMO) that conceptualizes reappraisal as a temporally extended, dynamic, and multi-componential process (Kalisch, 2009). A key tenet of IMMO is that reappraisal episodes are marked by an early phase of implementation that may comprise strategy selection and retrieval of reappraisal material into working memory, and a later phase of maintenance that may comprise working memory and performance monitoring processes. These should be supported by dissociable neural networks. We here show, using a detachment-from-threat paradigm and concurrent functional magnetic resonance imaging, that reappraisal-related brain activity shifts from left posterior to right anterior parts of the lateral frontal cortex during the course of a reappraisal episode. Our data provide first empirical evidence for the existence of two separable reappraisal stages. Implications for further model development are discussed.

Keywords: emotion regulation, reappraisal, detachment, distancing, fear, anxiety, prefrontal cortex

INTRODUCTION

Reappraisal modulates the evaluation of a stimulus in terms of its emotional–motivational meaning for the organism, a set of processes that is collectively termed appraisal. According to appraisal theorists, appraisal precedes the emotional response and is causal in its generation (is a "response-antecedent"; Roseman and Smith, 2001; Scherer, 2001). If one makes a simplifying assumption that the processing of an emotional stimulus consists in only one sequence of stimulus detection – appraisal – response, this leads to the prediction that reappraisal must work at the appraisal stage and thus occur before the behavioral response is elicited (Gross, 1998). Reappraisal would thus be a quick and largely effortless process that lastingly changes the trajectory of the emotional response without the need for extended monitoring of reappraisal success (see Gross, 2002, and Richards, 2004, for reviews). At the neural level, this line of thinking has led to a model whereby reappraisal-related brain activity should be observable exclusively during early time points of an emotional episode (Goldin et al., 2008).

However, theorists have also argued for a more dynamic view of reappraisal that incorporates the idea of multiple, sequential "detection – appraisal – response" cycles (Gross and Thompson, 2007; Kalisch, 2009). This refinement is in congruence with modern appraisal theory which views appraisal as a recurrent process (Roseman and Smith, 2001; Scherer, 2001): Emotional stimulation is often temporally extended and varies over time; further, our own emotional reactions may change the situation and may also function as emotional stimuli in their own right. Hence, the appraisal process must continuously incorporate new information and integrate it in order to enable continuous response adjustments. For reappraisal, this highly dynamic nature of the appraisal process means that very often, if one desires to change one's emotional state into a specific (e.g., more positive) direction, one may also have to continuously adjust one's reappraisals (Kalisch, 2009). In addition, there may be situations where the emotional stimulation is rather monotonous and its essential meaning quickly appraised, but because the stimulus is so significant and strong we may have to make a continuous effort to overwrite our spontaneous and natural appraisals with the ever same reappraisal thoughts. This may be the case, for instance, when we experience extended pain or anticipate potential harm (anxiety).

To take these considerations into account, we have proposed a model (implementation–maintenance model or IMMO) which conjectures that the requirement for flexibility may result in a switching between operations that promote the implementation of a reappraisal strategy (that is, choosing between, and retrieving, potential reappraisals from memory) and those that promote the maintenance of a chosen strategy (that is, working memory). Maintenance processes must involve a component that monitors success in emotion regulation and can initiate new implementation activity or enhance maintenance efforts. Under normal circumstances, that is, with at least moderate reappraisal success, one can make a simplifying prediction that implementation processes should be predominant early during a reappraisal

episode, while maintenance processes should prevail during later periods. Both sets of processes should be associated with distinct neural activation patterns (Kalisch, 2009).

Supporting this simple biphasic model, a meta-analysis of existing neuroimaging studies of reappraisal had indicated that early and late phases of reappraisal episodes are indeed supported by different neural substrates. Studies with comparatively short reappraisal episodes, which should be marked mainly by implementation efforts, preferentially activated left posterior lateral frontal cortex (LFC). By contrast, studies with comparatively long reappraisal episodes, where maintenance processes should increasingly come into play, preferentially activated right anterior LFC (Kalisch, 2009).

In the present experiment, we asked whether this pattern can be reproduced at the level of a group study. If so, this would be empirical evidence for the existence of an early and a late stage of reappraisal, at least within the context of the paradigm employed in this study. Specifically, we tested whether the major focus of reappraisal-related activation would shift across a reappraisal episode from left posterior to right anterior lateral frontal sites.

MATERIALS AND METHODS

OVERVIEW OVER DESIGN

Anxiety was induced using a classical instructed fear paradigm (also known as "anticipatory anxiety") which consisted in forewarning subjects that they might receive a painful electric stimulus at any time during a 17.7-s trial (Threat condition, T). During a control condition (No-threat, NT), subjects were told they would not be stimulated during the trial. In a fully balanced, two by two factorial design, subjects either employed reappraisal (Reappraisal condition, R), or not (No-reappraisal comparison condition, NR). For details, see **Figure 1** and the section below. For the Reappraisal condition, subjects were given a short self-statement and an associated visual imagery that both expressed a distanced, detached observer position. Subjects were supposed to bring up this reappraisal material at the beginning of a trial when cued, and then to mentally rehearse it throughout. Prior to the experiment, subjects had received some moderate training in retrieving and rehearsing the strategy. In the No-reappraisal condition, subjects were supposed to attend to the situation and to their emotional reactions but not to try to change them. In both conditions, subjects were eyes closed.

Four general considerations motivated our choice of design. First, a continuous threat of shock is a type of emotional stimulation that is both temporally extended and rather monotonous, the latter because its meaning to the organism is easily and quickly appraised and does not change over the course of a trial. It should thus leave enough time for observing potential spatio-temporal activation shifts while at the same time eliminating to some extent inter-trial and inter-individual variance in appraisal and reappraisal processes that might otherwise arise from a complicated or varying external situation (such as when stimulated by affective pictures or film clips). Both should facilitate the detection of common spatio-temporal activity patterns across subjects. Second, threat of pain is naturally and spontaneously appraised as relevant to the organism. Stimulus relevance is considered a major appraisal

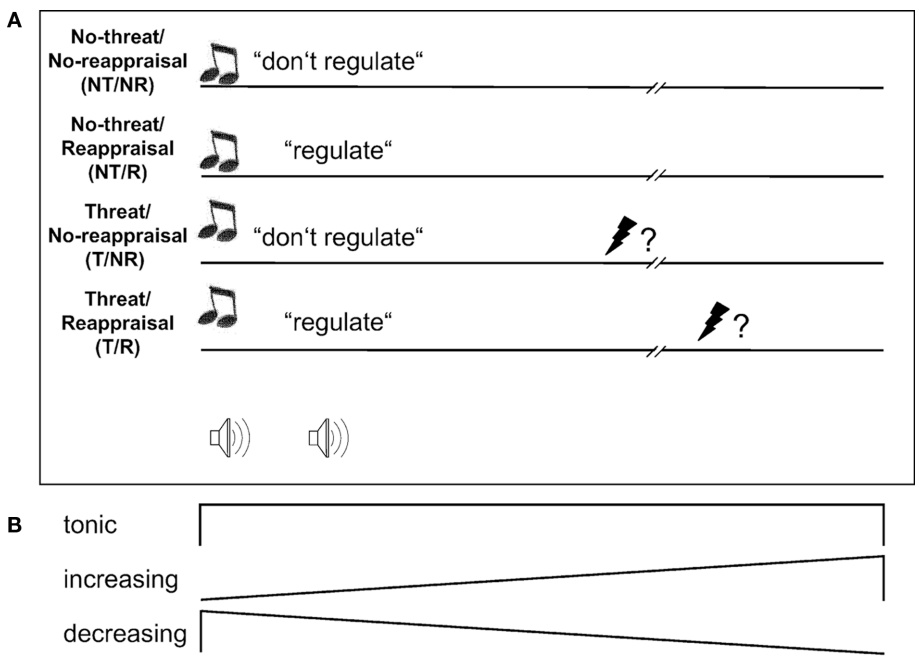

FIGURE 1 | Design. (A) At the onset of threat (T) trials, a high-pitch double-beep signaled subjects they might receive a painful electric stimulus to the hand at a probability of 25% at any time during the trial, which lasted 17.7 s. At the onset of no-threat (NT) trials, a low-pitch double-beep signaled safety. Reappraisal (R) trials were then signaled by the word "regulieren" ("regulate"), no-reappraisal (NR) trials by the word "belassen" ("leave" or "do not regulate"). Subjects remained eyes closed throughout trials. **(B)** To capture the predicted dynamic network behavior during reappraisal, neural activation during trials was modeled as tonic, linearly increasing and linearly decreasing responses (see Materials and Methods).

criterion and an important factor in determining whether a stimulus induces a lasting emotional reaction (Phillips et al., 2003; Sander et al., 2005) which is a logical pre-requisite for investigating late reappraisal activity. Third, we gave subjects detailed reappraisal instructions rather than leaving it to them how to exactly reinterpret the situation, because we reasoned that this would further reduce variance. Another motivation for this measure was to enhance chances that subjects do use reappraisal and not some other regulation technique, a problem that is pertinent in reappraisal studies where full control over the experimental manipulation is always limited by reappraisal being a purely mental process. Fourth, a final deviation from most reappraisal studies, which usually only compare a reappraisal to a no-reappraisal condition during emotional stimulation, is the use of a two by two factorial design. In such designs, one cannot only test for main effects of emotional stimulation (here, threat):

T–NT = −NT/NR−NT/R + T/NR + T/R or [−1 −1 1 1]

or reappraisal:

R–NR = −NT/NR + NT/R − T/NR + T/R or [−1 1 −1 1]

but also for two complementary types of interaction. The interaction contrast:

$(\text{T–NT})_{\text{NR}} - (\text{T–NT})_{\text{R}} = -\text{NT/NR} + \text{NT/R} + \text{T/NR} - \text{T/R}$ or [−1 1 1 −1]

tests for threat-related effects (T–NT) in anxiety measures or brain activity that are attenuated by reappraisal (in other words, anxiolysis). We use this contrast as a quantitative reappraisal success index (RSI) in the analysis of our behavioral data.

The inverse interaction contrast:

$(\text{R–NR})_{\text{T}} - (\text{R–NR})_{\text{NT}} = +\text{NT/NR} - \text{NT/R} - \text{T/NR} + \text{T/R}$ or [1 −1 −1 1]

tests for reappraisal-related effects (R–NR), e.g., in brain activity, that are larger under threat, that is, when one has to stay or become detached in spite of a concurrent emotional challenge. Hence, both interaction contrasts can provide additional interesting information. Crossing of the reappraisal factor with the threat factor was possible here because one can detach from any kind of situation, including a less emotional one (condition NT/R).

SUBJECTS

Twenty-one right-handed healthy male subjects with an average age of 28 ± 4 (mean ± SD) years (range 21–38 years) participated in the experiment. Subjects reported no current or past neurological or psychiatric illness, including anxiety disorders. All were of Caucasian origin; 15 were University students. Their average trait anxiety (Spielberger, 1985) was 32.1 ± 4.8 (range 25–45) and thus in line with norm population values (compare Laux et al., 1981). All subjects gave informed consent. The study was approved by the ethics committee of the Hamburg Medical Board and conformed to all relevant regulatory standards. Remuneration for participation was 40 Euros.

REAPPRAISAL STRATEGY AND PROCEDURE

We chose to use a distancing reappraisal strategy by which subjects, rather than immersing themselves in the situation and experiencing potential threat and the ensuing anxiety as directly affecting them, took a detached observer position and told themselves that all on-going external and internal events were not self-relevant. Such reappraisal has previously been shown to successfully attenuate anticipatory anxiety (Houston and Holmes, 1974; Kalisch et al., 2005). Other variants of distancing reappraisal have been used to alleviate depressed mood (Kross and Ayduk, 2008) or affective responding to negative picture material (e.g., Ochsner et al., 2004; Dillon et al., 2007; Erk et al., 2010). For the purpose of distancing, subjects were told to build a mental image of a cloud in the sky that would symbolize the current external situation such as being in the scanner, being safe from shock (NT) or being threatened (T), as well as accompanying internal sensations, feelings, and thoughts such as relaxation or relief (NT) or tension and anxiety (T). Hence, the image of the cloud was applicable to both the No-threat and the Threat condition but might symbolize different situations/feelings/thoughts depending on the condition. They were then asked to imagine themselves far away from this cloud, for example standing on a hill and observing the cloud from a distance (but not to look away). In addition to this mental image, they were given a self-statement that expressed the detached perspective: "Die Wolke ist weit weg am Horizont. Ich betrachte sie aus der Ferne." ("The cloud is far out on the horizon. I observe it from a distance.")

On the day of the experiment, subjects were explained the strategy and then trained in using it by first having them read aloud the statement 10 times, then having them freely recall each statement 5 times and finally provide verbal ratings of the effort necessary to recall the statement (0: not effortful at all – 10: extremely effortful) and of its emotional valence (0: very pleasant – 10: very unpleasant). All subjects were able to perfectly recall the statement, and final effort ratings were 1.2 ± 1.2. Final valence ratings were in the neutral range (3.7 ± 1.4). Subjects then had to spend 1 min eyes closed, performing visual imagery, followed by a free description of the imagined scene in their own words and ratings of effort, intensity or vividness of the images (0: not vivid at all – 10: very vivid) and valence (0: very negative – 10: very positive). In every subject, the imagined scene was in agreement with the distancing strategy and could be easily produced (final effort ratings: 2.5 ± 1.6). Imagery was sufficiently vivid (intensity ratings: 6.3 ± 1.4) and neutral in valence (valence ratings: 4.6 ± 0.5).

Training was followed by pain stimulus calibration. Stimuli were applied to the back of the right hand using a Digitimer DS7A electrical stimulator (Digitimer Ltd., Welwyn Garden City, UK) delivering 2 ms square-wave pulses of 0.01–100 mA through a surface electrode with platinum pin (Clyde's Polo Kit Supplies, Bexley, UK). Current levels were chosen which induced intermediate subjective anxiety. To achieve this, subjects rated their anxiety during a 15-to-0 countdown on a 100 point-scale. Subjects were told they might receive a triple-stimulus of a previously experienced level at any time during the countdown at a probability of 25%. This procedure was repeated with different current levels, starting at low levels, until an anxiety level between 50 and 80 was reached.

In a brief pre-experimental session, subjects were then familiarized with the experiment inside the scanner, but without receiving any pain stimulus. The actual experiment (see below) was split into three functional runs of 13 min duration each. Before each run, the pain stimulus was tested (rated) and recalibrated if necessary. This was done to preclude reinforcer devaluation effects that might

otherwise explain the anxiolytic effects of reappraisal. In addition, after each run subjects provided the same ratings on statement and imagery as after training (see above). Subjects' efforts to subvocally rehearse the statement and to mentally imagine the scene were slightly higher during scanning than at training but stable across runs (statement: 2.9 ± 1.5, 2.9 ± 1.4, and 3.1 ± 1.3; scene: 3.9 ± 2, 3.9 ± 1.6, and 4.2 ± 2). The vividness of imagery was comparatively reduced but also stable (5.9 ± 1.8, 5.7 ± 1.9, and 5.9 ± 2.2). The emotional valence of the statement and the imagined scene was stably judged as neutral to mildly pleasant (statement: 3.3 ± 1.5, 3.1 ± 1.5, and 3.5 ± 1.5; scene: 3.9 ± 1.3, 3.8 ± 1.3, and 4.6 ± 1.9). The latter is important to exclude that emotion regulation in this task consisted in simply replacing negative affect by some strong, opposing positive emotion (that is, self-distraction). After the experiment, subjects were interviewed about the strategies they used and about their experiences during the scans. No subject had to be excluded due to apparent unsatisfactory commitment.

TASK

There were altogether 84 randomized 17.7 s trials (28 per run), 18 in each of the conditions NT/NR and NT/R and 24 in each of the conditions T/NR and T/R (see **Figure 1** for trial structure and cues). During six of the T/NR and T/R trials subjects received a triple pain stimulus (pulse intervals: 80 ms) which occurred randomly within a time window of 3–12 s after offset of the auditory instruction (that is, from approx. 2.7 s into the trial). A trial ended with the instruction "Augen auf, Rating!" ("Eyes open, rating.") followed by a 5-s presentation of a rating screen with the question "Wie groß war Ihre Angst/Anspannung?" ("How strong was your anxiety/tension?") and a visual analog scale below. On the scale, subjects could move a red star using their keypad between poles "no anxiety" (0) and "very strong anxiety" (100). The position of the star at the onset of each rating was randomized. The subsequent 5 s break was cued by the words "Augen zu, Pause!" ("Eyes closed, break.").

DATA ACQUISITION

Skin conductance (SC) was measured at a sampling rate of 1000 Hz from electrodes on the palm and the thenar of the left hand using a CED2502-SA SC unit (Cambridge Electronic Design, Cambridge, UK) and recorded with Spike 2 software (CED). Offline, SC data were down-sampled to 100 Hz and filtered (Gaussian kernel, FWHM 0.1 s). SC level (SCL) was defined as the average SC across a trial minus SC at the first time point in that trial. Values were z-transformed (Buchel et al., 1998). In one subject, SC data could not be used due to technical problems, reducing sample size for SC analysis to $n = 20$.

Functional imaging was performed on a 3-Tesla MR scanner (Siemens Trio, Erlangen, Germany) equipped with a 12-channel head coil, using a gradient echo T2* weighted echo-planar imaging (EPI) sequence with blood oxygenation level-dependent (BOLD) contrast (TE = 30 ms, TR = 2.47 s, flip angle = 80°). TE was minimized using a parallel acquisition technique (generalized autocalibrating partially parallel acquisitions, GRAPPA) with an acceleration factor of 2 and 24 reference lines. Each volume comprised 38 axial slices (AC–PC orientation) of 2 mm thickness and 2 mm × 2 mm in-plane resolution with a slice gap of 1 mm. Participants were placed in a light head restraint within the scanner to limit head movement during acquisition. A T1-weighted structural image was also acquired.

DATA ANALYSIS

Trials during which subjects received pain stimuli were excluded from all analyses of experimental effects. Statistical analysis of behavioral data was performed within SPSS 17.0 (SPSS, Chicago, IL, USA).

Functional magnetic resonance imaging (fMRI) data were preprocessed using SPM8 (www.fil.ion.ucl.ac.uk/spm; Friston et al., 2007). The five initial EPI images were discarded to account for T1 equilibration. To correct for head movement and movement-by-distortion interactions, they were then realigned to the sixth volume and unwarped. The structural T1 images were coregistered to the EPI images and then segmented and spatially normalized to a standard T1 template using the "New Segment"-routine as implemented in SPM8. The normalization parameters from this procedure were then applied to the EPI images. The normalized EPI images were spatially smoothed (Gaussian kernel, FWHM 6 mm), temporally high-pass filtered (cut-off 128 s) and corrected for temporal autocorrelations using first-order autoregressive modeling.

Statistical analysis was performed using a standard approach for fMRI, involving a general linear convolution model at the single-subject level and a random-effects analysis at the group level within the SPM software (see Friston et al., 2007, for details). The three runs were concatenated into a single time series and, for each subject, regressors were defined that modeled the predicted time courses of experimentally induced brain activation changes. Each of the four experimental conditions (NT/NR, NT/R, T/NR, T/R) was modeled using two different temporal response profiles during the 17.7-s trials: a tonic response lasting the whole duration of a trial and a response that increased linearly across a trial (see **Figure 1**). Receipt of pain was modeled as distinct "events" (delta functions with 0 duration). Blocks during which subjects actually received pain stimuli and ratings were modeled as "boxcar" (on-off) regressors. Onset of pauses after the ratings were modeled as events. Each regressor was convolved with the canonical hemodynamic response function. Using these regressors in a general linear model (multiple regression) of brain activation at each voxel yields parameter estimates of the contribution of each regressor to the fMRI signal measured in each voxel. The subject- and regressor-specific parameter estimate images were spatially smoothed (FWHM 10 mm) and, for the standard analysis reported in Results, entered into a random-effects group analysis using SPM's "flexible factorial" model which permits correction for possible non-sphericity of the error term (here, dependence of conditions). Group-level design matrices included 25 regressors (4 regressors of interest corresponding to the 4 experimental conditions NT/NR, NT/R, T/NR, and T/R, plus 21 subject constants). Linear combinations ("contrasts") of the regressors of interest were used to test for main effects and interactions as defined in the Overview section above. Here, multiplication of the parameter estimate images for the linearly increasing regressors by −1 allowed for also assessing linearly decreasing responses

as depicted in **Figure 1**. Significance of effects was tested using voxel-wise one-tailed *t*-tests.

For the standard analysis, correction for multiple comparisons following Gaussian random field theory (family wise error, FWE method) at a threshold of $p < 0.05$ ("small volume correction") was limited to regions of interest (ROIs) for which we had *a priori* hypotheses and which we identified from the literature. Specifically, for main effects and interaction contrasts related to threat responding we used the two activation peaks which had shown maximally consistent activation across studies in a meta-analysis of instructed fear experiments (Mechias et al., 2010), one located in the dorsomedial prefrontal/anterior cingulate cortex (dmPF/ACC, MNI coordinates *x,y,z* = 0,16,36) and one in the right anterior insula (36,20,0). Like in previous work (Raczka et al., 2010), the dmPFC/ACC ROI was a box of dimensions *x,y,z* = 20,16,16 mm, covering the mPFC bilaterally. The insula ROI was a sphere with a 12-mm radius. Note that the instructed fear paradigm does not reliably and consistently activate the amygdala (see Mechias et al., 2010, for meta-analysis) and does not do so in our hands, even when taking into account possible habituation (Kalisch et al., 2005). Recent developments have highlighted a role for the dmPFC/ACC as another important mediator of fear expression in both animals and humans (reviewed in Etkin et al., 2011). For main effects and interaction contrasts related to reappraisal we used the two activation peaks which had shown maximally consistent activation across studies in a meta-analysis of reappraisal experiments (Kalisch, 2009), one in the dmPFC (−4,20,52) and one in the left LFC (−40,10,48). The corresponding box in the dmPFC was midline-centered ($x = 0$) to cover the dmPFC bilaterally. The LFC ROI was again a sphere with a 12-mm radius. In addition, we used the peak effect from our previous detachment-from-threat study (Kalisch et al., 2005) where we had seen increasing reappraisal activity over episodes in a right anterior LFC focus (42,48,18; 12-mm sphere).

In the results tables, anatomical localization of activations was carried out with reference to the atlas of Duvernoy (1999). Unambiguous white matter or liquor clusters are not reported. Cluster submaxima are reported when more than 8 mm apart.

For the test of IMMO by comparison of the spatial distributions of linearly increasing and decreasing reappraisal main effects (see Results), we created an anatomical mask of the bilateral LFC that included all parts of the superior, middle, and inferior frontal gyri, based on a standard anatomical atlas (Tzourio-Mazoyer et al., 2002).

RESULTS

BEHAVIORAL RESULTS: ANXIETY RATINGS AND SKIN CONDUCTANCE

Anxiety ratings showed a significant main effect of Threat [$F(1,20) = 367.37$, $p < 0.001$], a trend-like main effect of Reappraisal [$F(1,20) = 4.11$, $p = 0.056$], and a significant Threat by Reappraisal interaction [$F(1,20) = 10.17$, $p = 0.005$]. The interaction was apparently driven by a reduction of anxiety in the T/R compared to the T/NR condition (**Figure 2A**), an anxiolytic effect corresponding to the interaction term [−1 1 1 −1] or RSI as defined above in "Materials and Methods," Overview section. The anxiety-rating-RSI was significantly >0 [9.1 ± 13, $t(20) = 3.19$, $p = 0.003$ one-tailed, planned *post hoc* *t*-test]. A caveat is that, rather than a true reduction of anxiety by reappraisal, these ratings may also reflect the demand characteristics of the task. SC is an index of the sympathetic arousal that usually accompanies anxiety and thus a more objective metric for anxiety. SC was significantly elevated by threat [main effect of threat: $F(1,19) = 97.4$, $p < 0.001$], not affected by reappraisal as such [main effect of reappraisal: $F(1,19) = 0.26$, $p = 0.619$], and showed the critical interaction of Threat and Reappraisal [$F(1,19) = 5.37$, $p = 0.032$;

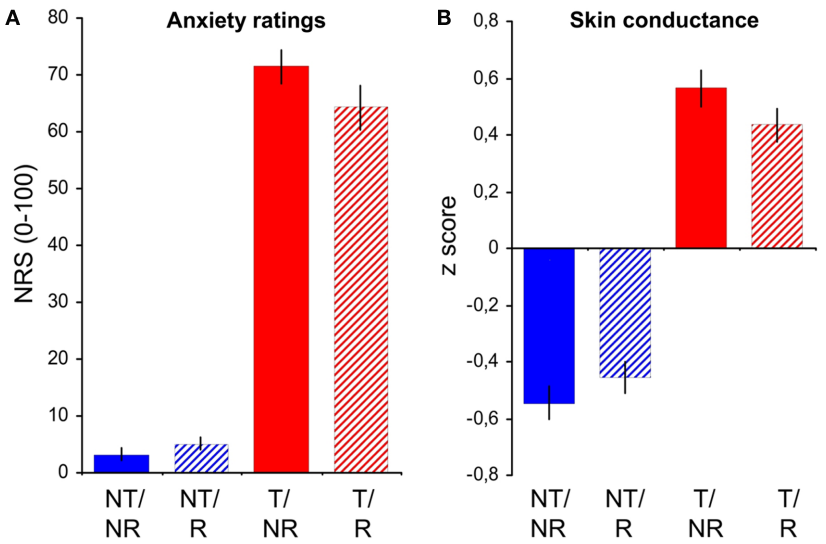

FIGURE 2 | Behavioral results. Average trial-by-trial anxiety ratings **(A)** and skin conductance levels **(B)** in the conditions no-threat/no-reappraisal (NT/NR), no-threat/reappraisal (NT/R), threat/no-reappraisal (T/NR), and threat/reappraisal (T/R). Error bars: SEM. NRS, numerical rating scale.

Figure 2B]. The SC–RSI was significant [0.22 ± 0.42, $t(19) = 2.32$, $p = 0.016$ one-tailed], further confirming the anxiolytic effect of reappraisal.

IMAGING RESULTS: STANDARD ANALYSIS

For an overview, main effects and interactions in tonic and linearly increasing and decreasing responses are reported in **Tables 1–3**. Globally, threat of shock induced the typical widespread activations in dmPFC/dACC, anterior insula, basal ganglia, thalamus, brainstem, cerebellum, and other areas (compare Mechias et al., 2010, for meta-analysis). In our predefined dmPFC/dACC and right anterior insula ROIs (see Materials and Methods), there were significant tonic (dACC/dmPFC: 2,20,34, z score $= 3.63$, $p = 0.004$ corrected for multiple comparisons; right anterior insula: 34,26,4, $z = 5.13$, $p < 0.001$ corr., and others) and decreasing (dmPFC/dACC: −2,10,36, $z = 2.92$, $p = 0.028$ corr.; right anterior insula: 38,26,4, $z = 3.23$, $p = 0.013$ corr.) main effects of threat.

Main effects of reappraisal were observed in the previously described network comprising medial and lateral frontal areas, parietal, and temporal cortex, cerebellum, basal ganglia, and others (compare Kalisch, 2009, for meta-analysis). In our predefined dmPFC, left LFC and right anterior LFC ROIs (see Materials and Methods), there were significant tonic (dmPFC: −8,12,48, $z = 5.87$, $p < 0.001$ corr.; left LFC: −46,0,46, $z = 7.45$, $p < 0.001$ corr.; **Figures 3A,B**), increasing (dmPFC: −2,28,48, $z = 2.91$, $p = 0.029$ corr.; left LFC: −40,20,42, $z = 3.11$, $p = 0.019$ corr.; right anterior LFC: 42,44,8, $z = 3.25$, $p = 0.012$ corr.; **Figures 3C–E**), and decreasing (left LFC: −44,0,44, $z = 3.45$, $p = 0.007$ corr.; **Figure 3F**) responses. The parameter estimates in the figure suggest that some of these responses tended to be higher when subjects performed reappraisal under threat (condition T/R) compared to when they were safe (condition NT/R). This apparent threat by reappraisal interaction however rarely reached significance (see below), presumably because it was masked by the massive reappraisal main effect.

Threat by reappraisal interactions of the form [−1 1 1 −1], corresponding to threat-related activity that is reduced by reappraisal (see Materials and Methods, Overview section), were sparse (see **Tables 1–3**). The only significant effect was found in our right anterior insula ROI for linearly decreasing responses (36,28,4, $z = 2.97$, $p = 0.027$ corr., and others; **Figure 4A**), in an area close to the peak showing a decreasing main effect of threat (see above and **Table 3**). The pattern of parameter estimates in **Figure 4A** shows that reappraisal (condition T/R) abolished the signal decrease otherwise observed as a response to threat (condition T/NR), in line with an anxiolytic effect of reappraisal.

Inverse threat by reappraisal interactions of the form [1 −1 −1 1], corresponding to reappraisal-related activity that is larger under threat (see Materials and Methods, Overview section), were mainly observed outside the frontal cortex (**Tables 1–3**). A trend-level effect was found in our dmPFC ROI for linearly increasing responses (6,26,60, $z = 2.44$, $p = 0.086$ corr.; **Figure 4B**).

Globally speaking, these results are in agreement with previous imaging studies and further confirm the behavioral findings of successful anxiety induction by threat of shock and successful down-regulation of anxiety (anxiolysis) by detachment-reappraisal.

IMAGING RESULTS: TESTING IMMO

If early and late stages of reappraisal rely on different neural substrates, then reappraisal-related activations with linearly decreasing and increasing response profiles should show dissociable anatomical distributions. More specifically, if the observations in Kalisch (2009) hold, then linearly decreasing effects should be mainly observed in the left posterior LFC while increasing effects should be mainly located in the right anterior LFC. The SPM glass brains for both types of reappraisal main effect contrasts in **Figures 5A,B** seem to confirm this prediction, with exclusive left posterior activation in the decreasing contrast and additional and more prominent recruitment of right-sided areas in the increasing contrast.

To formally test this, we used methodology analogous to our previous meta-analysis (Kalisch, 2009). We averaged within each of the 21 subjects in the study and for each type of contrast (decreasing, increasing reappraisal main effect) the coordinates of all (left- and right-sided) activated voxels contained in the *a priori* bilateral LFC mask defined in Section "Materials and Methods," at an uncorrected threshold of $p < 0.01$. This threshold turned out to be most appropriate as it yielded supra-threshold voxels in nearly all subjects (20 for increasing responses, 19 for decreasing responses), thus assuring a sufficient number of data points and comparability across response types. Averaging of coordinates resulted in one single coordinate for each subject and contrast (**Figure 6**) that expressed the "center of gravity" of lateral frontal reappraisal activation in that subject and contrast. That is, if in a given subject and contrast the majority of activated voxels was located in, for instance, the left LFC, this would result in an average coordinate with a negative (left-sided) x value. Analogously, if in a given subject and contrast the majority of LFC voxels was located in, for instance, posterior LFC, this would "push" the average coordinate toward smaller (more posterior) y values. Note this "center of gravity" is a virtual coordinate which may not correspond to any actual locus of activation. For the decreasing response, the group average of these coordinates was $x,y,z = -38,13,33$ and thus located significantly more to the left (smaller x value) and more posteriorly (smaller y value) than the group-averaged coordinate for the increasing response [$x,y,z = 12,36,19$; x value: $t(17) = 4.76$, $p < 0.001$; y value: $t(17) = 2.91$, $p = 0.005$; one-tailed paired two-sample t-tests]. This result was robust to removal of outliers (± 2 SD, all $p < 0.003$).

In addition, we observed an unpredicted effect in the z values [$t(17) = 2.39$, $p = 0.029$ two-tailed; after removal of outlier: $p = 0.019$; not shown], with decreasing responses being located slightly more dorsally (higher z value). A further unpredicted finding was that the tonic reappraisal main effect showed an average center of gravity ($x,y,z = -12,7,17$) that had a similar left posterior location as the decreasing response (**Figures 5** and **6**).

DISCUSSION

Our data provide first evidence for the existence of at least two separable stages of mental activity occurring during a sufficiently long reappraisal episode, one early stage apparently marked by left

Table 1 | Imaging results: standard analysis. Activations with tonic response profile across trials.

Region	Cluster maximum (MNI)			*z* Score	Cluster size (# voxels)	*p* Corr. <0.05? (whole brain)
	x	*y*	*z*			
MAIN EFFECT OF THREAT (T–NT): [−1 −1 1 1]						
Bilat basal ganglia, extending to:	−24	6	−2	6.06	7321	Yes
Thalamus, bilat ant insula, midbrain, brainstem	22	4	−2	5.23		Yes
R ant insular S	34	26	4	5.13		Yes
L cerebellum	−18	−76	−34	5.31	3581	Yes
	−36	−60	−38	4.66		Yes
	−50	−76	−32	4.24		
R inf pariet G	66	−44	34	4.99	907	Yes
Splenium/isthmus	2	−30	20	4.4	339	Yes
R cerebellum	34	−54	−36	4.10	303	
L middle front G, ant	−40	52	24	3.72	129	
L inf pariet G	−64	−42	34	3.72	131	
Cerebellum	2	−54	−26	3.65	121	
dACC	2	20	34	3.63	248	
R middle front G, ant	32	50	18	3.58	190	
R inf front S, post	42	2	44	3.35	32	
R post dACC/(pre-) SMA	12	8	58	3.33	9	
Med sup front G	4	24	52	3.19	10	
MAIN EFFECT OF REAPPRAISAL (R–NR): [−1 1 −1 1]						
L middle front G, extending to:	−46	−4	44	7.67	6179	Yes
L pre-SMA/dACC	−8	12	48	5.87		Yes
L inf front S, extending to:	−46	24	30	4.22		
Lat fissure/ant insula						
L intraparietal S	−28	−56	46	5.97	3154	Yes
	−32	−74	28	4.50		Yes
	−12	−68	44	4.43		Yes
R cerebellum	30	−62	−30	5.28	2347	Yes
	8	−72	−28	4.94		Yes
L sup temp G	−62	−36	−2	4.60	828	Yes
R middle front G, post, extending to:	54	2	46	4.35	647	Yes
R sup front S, post	34	−2	48	4.22		
R intraparietal S	28	−66	40	3.97	223	
L dors striatum	−20	−2	8	3.85	217	
Dors midbrain, extending to:	4	−34	−28	3.78	590	
L ventr cerebellum	−16	−26	−28	3.75		
	−10	−44	−26	3.45		
L lat fissure, post	−50	−40	22	3.14	1	
R dors striatum	22	0	16	3.14	3154	
INTERACTION DOWN-REGULATION OF ANXIETY $(T–NT)_{NR}$–$(T–NT)_{R}$: [−1 1 1 −1]						
R sup temp S, post	40	−62	28	3.86	532	
	48	−50	18	3.37		
R sup temp S	58	−26	0	3.44	75	
L mid temp S	−56	−40	−12	3,3	14	
L mid temp G, extending to:	−38	16	−44	3,27	77	
L mid temp S	−30	16	−38	3,19		
L sup temp G	−70	−20	−2	3.25	48	
INTERACTION THREAT-SPECIFIC REAPPRAISAL (R–NR) – $(R–NR)_{NT}$: [1 −1 −1 1]						
No voxels surviving threshold						

Statistical threshold: $p < 0.001$ uncorrected. Definition of contrast as in Section "Materials and Methods," Overview section. G, gyrus; L, left; R, right; S, sulcus. MNI, Montreal Neurological Institute.

Table 2 | Imaging results: standard analysis. Activations with linearly increasing response profile across trials.

Region	Cluster maximum (MNI)			*z* Score	Cluster size (# voxels)	*p* Corr. <0.05? (whole brain)
	x	*y*	*z*			
MAIN EFFECT OF THREAT (T–NT): [−1 −1 1 1]						
L ant dors striatum	−18	14	8	3.17	7	
L post caudatum/white matter	−24	−26	22	3.15	5	
MAIN EFFECT OF REAPPRAISAL (R–NR): [−1 1 −1 1]						
R temp-parietal–occipital transition	58	−38	36	4.69	2372	Yes
zone, incl. intraparietal S	54	−46	34	4.58		Yes
R middle temp G	72	−16	−20	4.15	228	
R sup front S	38	14	42	3.89	865	
L inf pariet G	−56	−52	38	3.89	644	
R insular G, post	36	−2	2	3.86	129	
L lat OFC	−46	50	−10	3.85	251	
L inf temp G	−52	−2	−36	3.66	167	
R post orbit G	36	40	−14	3.62	60	
R sup front S	20	40	32	3.6	163	
L middle temp G	−64	−30	−18	3.57	311	
R insular S, vent	30	18	−20	3.38	73	
R inf front S, ant	40	44	4	3.36	130	
L cerebellum	−30	−70	−38	3.28	20	
L ant dACC/pgACC	−6	46	16	3.28	146	
L middle front S	−42	22	42	3.13	5	
INTERACTION DOWN-REGULATION OF ANXIETY $(T–NT)_{NR}–(T–NT)_R$: [−1 1 1 −1]						
L supramarginal G	−38	−50	32	3.65	106	
L cerebellum	−56	−62	−38	3.38	54	
L cerebellum	−2	−92	−30	3.34	96	
	−8	−82	−28	3.18		
L cerebellum	−50	−78	−28	3.32	30	
L cerebellum	−40	−60	−28	3.17	9	
INTERACTION THREAT-SPECIFIC REAPPRAISAL $(R–NR)_T–(R–NR)_{NT}$: [1 −1 −1 1]						
No voxels surviving threshold						

Statistical threshold: $p < 0.001$ uncorrected. Definition of contrast as in Section "Materials and Methods," Overview section. G, gyrus; L, left; OFC, orbitofrontal cortex; pgACC, perigenual ACC; R, right; S, sulcus; SMA, supplementary motor area. MNI, Montreal Neurological Institute.

Table 3 | Imaging results: standard analysis. Activations with linearly decreasing response profile across trials.

Region	Cluster maximum (MNI)			*z* Score	Cluster size (# voxels)	*p* Corr. <0.05? (whole brain)
	x	*y*	*z*			
MAIN EFFECT OF THREAT (T–NT): [−1 −1 1 1]						
L sup pariet G, med	−4	−62	72	4.08	247	
R cuneus	8	−104	8	3.29	35	
R insular S, ant	38	26	4	3.24	19	
MAIN EFFECT OF REAPPRAISAL (R–NR): [−1 1 −1 1]						
L sup front S	−44	−2	44	3.7	103	
L sup front G, med/(pre-) SMA	−6	2	66	3.58	68	
INTERACTION DOWN-REGULATION OF ANXIETY $(T–NT)_{NR}–(T–NT)_R$: [−1 1 1 −1]						
See interaction threat-specific reappraisal $(R–NR)_T–(R–NR)_{NT}$: [1 −1 −1 1] in **Table 2**						
INTERACTION THREAT-SPECIFIC REAPPRAISAL $(R–NR)_T–(R–NR)_{NT}$: [1 −1 −1 1]						
see Interaction Down-regulation of Anxiety $(T–NT)_{NR}–(T–NT)_R$: [−1 1 1 −1] in **Table 2**						

Statistical threshold: $p < 0.001$ uncorrected. Definition of contrast as in Section "Materials and Methods," Overview section. G, gyrus; L, left; R, right; S, sulcus; SMA, supplementary motor area. MNI, Montreal Neurological Institute.

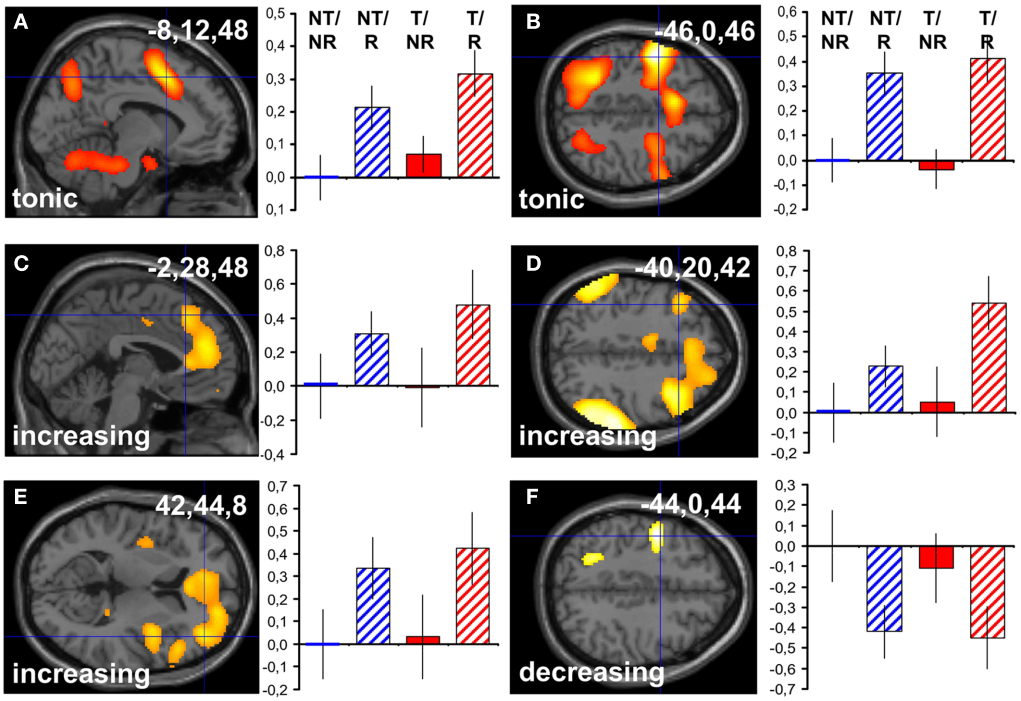

FIGURE 3 | Standard analysis: reappraisal main effects. Reappraisal main effects (contrast R–NR or [−1 1 −1 1], see Materials and Methods, overview section) in predefined ROIs surviving small volume correction for multiple comparisons at $p < 0.05$. Tonic response in dmPFC **(A)** and left LFC **(B)**. Linearly increasing response in dmPFC **(C)**, left LFC **(D)**, and right anterior LFC **(E)**. Linearly decreasing response in left LFC **(F)**. The Reappraisal main effects with a linearly decreasing profile are computed after multiplying single-subject parameter estimate images for the linearly increasing regressor with −1 (see also Materials and Methods, Data analysis). Activations are superimposed on a canonical structural image. Display threshold: $p < 0.01$ uncorrected. Left is upward in the coronal views. Bar graphs show corresponding group-averaged parameter estimates ("betas") in the voxel indicated by the haircross and the inserted coordinate. Values are normed to the first condition (NT/NR). In tonic contrasts, positive parameter estimates indicate tonic activation. In the other contrasts, positive parameter estimates indicate linearly increasing, negative parameter estimates indicate linearly decreasing response profiles. Error bars: SEM.

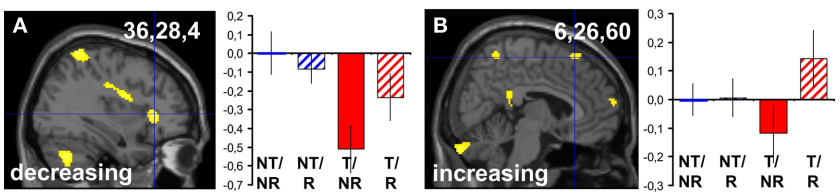

FIGURE 4 | Standard analysis: threat by reappraisal interactions. Threat by reappraisal interactions in predefined ROIs surviving small volume correction for multiple comparisons at $p < 0.05$ **(A)** or at trend-level, $p < 0.1$ **(B)**. In the right anterior insula **(A)**, a linearly decreasing response to threat (condition T/NR) is attenuated by reappraisal (condition T/R). The peak was identified from the interaction contrast [−1 1 1 −1] after multiplying the single-subject parameter estimate images for the linearly increasing regressor with −1 (see Materials and Methods). In the dmPFC **(B)**, reappraisal induces a linearly increasing response specifically when subjects are threatened (condition T/R) but not when they are safe (condition NT/R). The peak was identified from the interaction contrast [1 −1 −1 1] (see Materials and Methods). Activations are superimposed on a canonical structural image. Display threshold: $p < 0.01$ uncorrected. Bar graphs show corresponding group-averaged parameter estimates ("betas") in the voxel indicated by the haircross and the inserted coordinate. Values are normed to the first condition (NT/NR). Positive parameter estimates indicate linearly increasing, negative parameter estimate indicate linearly decreasing responses. Error bars: SEM.

posterior LFC activation and a later stage apparently marked by comparatively more right-sided and more anterior LFC activation. Our data do not provide evidence as to what types of cognitive processes operate during these two stages. We can therefore only assume that early processes subserve the implementation of the reappraisal strategy while late processes subserve its maintenance. Because subjects were instructed to use a specific, well-specified strategy and had received some training before the experiment, strategy selection mechanisms are unlikely to have played a major role during the putative implementation stage. This leaves memory retrieval as a likely candidate for the left posterior LFC processes active at that stage. Possible candidates for the later stage of

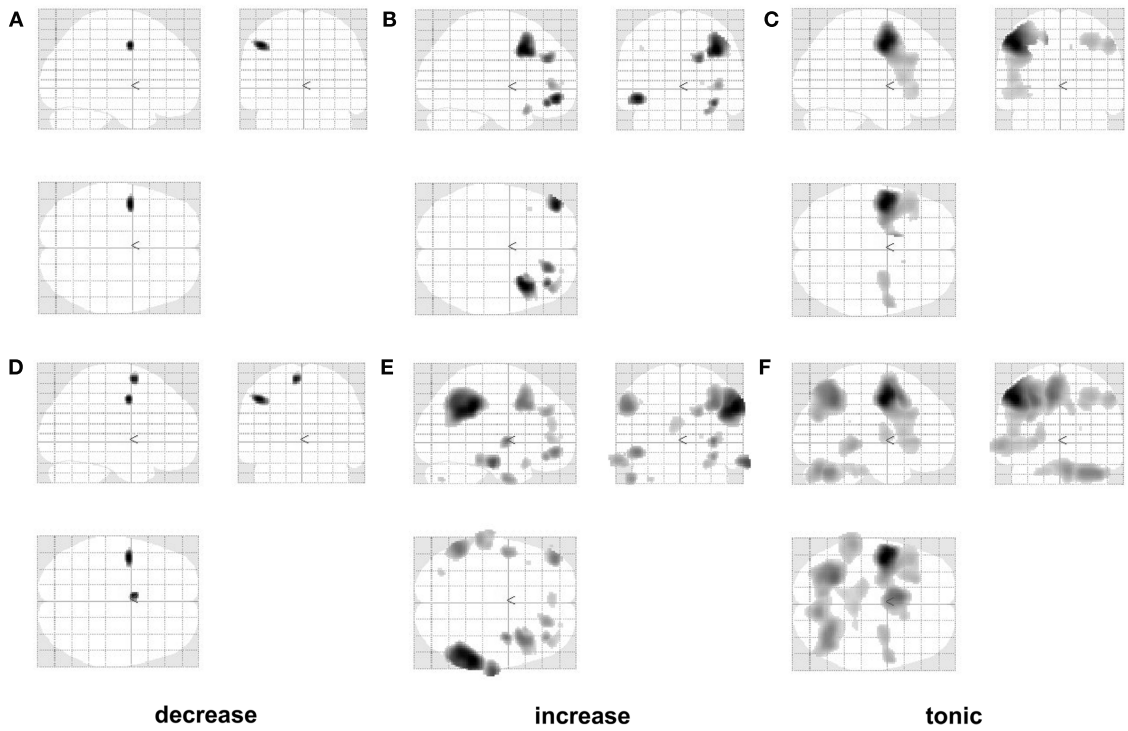

FIGURE 5 | Testing IMMO: glass brains. Reappraisal main effects (R–NR or [−1 1 −1 1]) with linearly decreasing **(A–D)**, increasing **(B–E)**, and tonic response profiles **(C–F)**. Glass brains in the upper row are masked by a bilateral LFC mask (see Materials and Methods). Glass brains in the lower row additionally show extra-LFC voxels and are otherwise identical. Threshold: $p < 0.001$ uncorrected. Left is left and right is right. Note the apparent shift from comparatively more left posterior to more right anterior activation sites between linearly decreasing and increasing profiles.

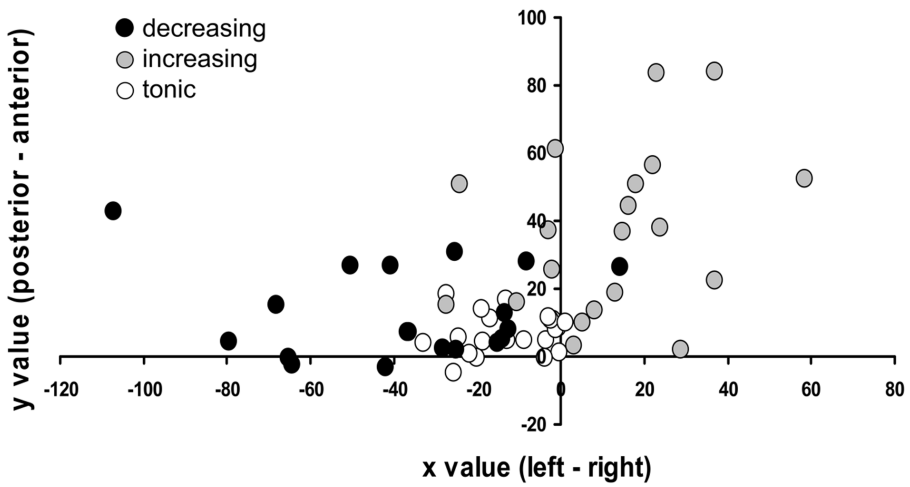

FIGURE 6 | Testing IMMO: lateral frontal average coordinates. In each subject, coordinates of all lateral frontal supra-threshold ($p < 0.01$ uncorrected) voxels from the reappraisal main effect (R–NR or [−1 1 −1 1]) were averaged into one single, virtual coordinate or "center of gravity." Reappraisal effects with a linearly decreasing response profile (black dots) are located comparatively more to the left (x value of average coordinate) and more posteriorly (y value) than reappraisal effects with a linearly increasing profile (gray dots). White dots: tonic profile.

maintenance are working memory and performance monitoring operations.

An important question for future research will be to what extent these findings can be generalized to other types of emotions, including positive ones, and other types of reappraisal strategies, such as those that reinterpret the causal structure of a situation ("situation-focused") rather than applying distancing ("self-focused"; Ochsner et al., 2004). Within the narrower context of the

paradigm used here, one can ask if the results would hold with a different No-reappraisal (NR) comparison condition. The issue of the comparison condition is critical because a low-level "attending to the emotion" condition like the one used here and in most other paradigms does not control for possible non-specific effects of general cognitive effort, mental imagery, linguistic processing (here: subvocal rehearsal), outcome expectation, and others. In an earlier study with a similar, though more complicated detachment-reappraisal strategy we had used a comparison condition that also included imagery and subvocal rehearsal, in order to more closely match R and NR conditions (Kalisch et al., 2005). The comparison condition had promoted immersion into the situation (compare Kross and Ayduk, 2008) in an attempt to model a natural and spontaneous appraisal of threat situations. One can predict that using such a control condition would abolish most reappraisal (R–NR) activations observed in this study, at least as far as these reflect the general executive processes needed to retrieve and maintain self-statement and imagery. Conversely, such a comparison should be more specific for performance monitoring operations. This prediction is based on the reasoning that subjects do not intend to down-regulate their anxiety in the control condition and do not expect it to attenuate their anxiety, making success monitoring in the control condition irrelevant and superfluous. IMMO conjectures performance monitoring to normally occur during the later stages of reappraisal trials, and we have speculated that the right anterior LFC might be specifically involved in this function (Kalisch, 2009).

Further factors can be predicted to influence activation patterns. In a situation where reappraisal demands vary from trial to trial (e.g., because each emotion-inducing stimulus, such as an affective picture, varies from preceding ones in content and intensity) and subjects thus have to select slightly different reappraisals in every trial, there should be an emphasis on implementation functions, and thus comparatively more left posterior LFC activity. This should be especially true when situation-focused strategies are used (see above) which often require individual solutions for every single emotional situation and compared to which self-focused reappraisal (that is, detachment) is a more generic, all-purpose type of strategy. Implementation operations should also be comparatively more dominant when subjects have not received prior training and/or are free to choose their own preferred reappraisal strategy. The same should apply to paradigms where subjects are only cued to begin to reappraise *after* the onset of the emotional stimulation. Finally, we have emphasized that a simple early/late distinction is only observable if reappraisal episodes are long enough (otherwise there is no time for maintenance) and if subjects are at least moderately successful in their reappraisal efforts (otherwise there will be frequent switching between performance monitoring and enhanced implementation and/or maintenance efforts and activation patterns should become largely unpredictable; Kalisch, 2009).

Our study is limited in that it cannot address this host of questions. Yet we believe it is an important first step toward better understanding the cognitive and neural architecture of reappraisal. A further limitation that needs to be mentioned is the use of an exclusively male, Caucasian sample that, moreover, comprised mainly university students. Male and female subjects recruit similar neural networks when reappraising, but to a different degree (McRae et al., 2008; Mak et al., 2009; Domes et al., 2010). While restricting this study to male subjects should have helped in reducing variance and thus identifying potentially subtle spatio-temporal activation effects, this measure clearly necessitates replication of findings in a more representative sample. Finally, it would be desirable to reproduce our fMRI findings with other imaging modalities such as EEG or MEG (e.g., Hajcak and Nieuwenhuis, 2006; Moser et al., 2006, 2009; Deveney and Pizzagalli, 2008). It should also be mentioned that other reappraisal studies have occasionally observed lateralized activation patterns in some contrasts (e.g., Ochsner et al., 2002, 2004; Phan et al., 2005; Urry et al., 2006; Eippert et al., 2007; Kim and Hamann, 2007; Walter et al., 2009; Erk et al., 2010), although these studies did not take into account time as a factor and generally did not calculate formal laterality analyses. We have earlier emphasized the possibility that other factors than time may affect the location of reappraisal-related frontal activations (Kalisch, 2009). We are however currently not aware of any single factor that is systematically associated with either predominantly left- or right-sided activation.

In the remaining paragraphs, we would like to discuss a number of side findings that might nevertheless be interesting for future theorizing. In our previous detachment-from-threat study with an immersion comparison condition (NR, see above), we had observed attenuation of threat-related activity in the rostral dmPFC/ACC (Kalisch et al., 2005) while, in the present study, we observed a corresponding interaction in the right anterior insula (see Results, standard analysis). We have meanwhile amassed evidence that the rostral dmPFC/ACC is involved in conscious threat appraisal (Kalisch et al., 2006; Mechias et al., 2010) and, in its extreme form, catastrophizing (Raczka et al., 2010). It is therefore conceivable that the use of an explicit immersion comparison condition that promotes negative reflection about the situation in the previous study had boosted rostral dmPFC/ACC responding to threat (T/NR condition), making it in turn sensitive for attenuation by reappraisal (T/R condition). Conversely, in the present study, subjects were simply asked to pay attention to their feelings and reactions in the NR comparison condition. This may have enhanced insula-dependent interoceptive awareness (Craig, 2009) and made the insula a primary neural target of reappraisal in the T/R condition.

Unexpectedly, reappraisal also induced left posterior LFC as well as dmPFC/ACC activations with a tonic response profile. While these again speak for reappraisal being a temporally extended process, we have nevertheless not predicted them. IMMO holds that subjects make renewed implementation or enhanced maintenance efforts, if performance monitoring signals insufficient reappraisal success. One could speculate that the current task of detaching from a threat of being shocked was difficult enough to engage such re-iterant processing to some extent. The small effect sizes in our subjective and physiological measures of anxiolysis could be taken to support this idea. The similarity of tonic and linearly decreasing main effects of reappraisal in terms of their anatomical distributions would suggest that subjects repeatedly recurred to retrieving the learned reappraisal material into working memory (implementation). Better previous training or a smaller anxiogenic challenge should then reduce this effect. An

alternative possibility that we cannot exclude is that the tonic left posterior LFC activation reflects working memory operations that function to continuously maintain the reappraisal material online. In this case, working memory maintenance would commence earlier in a trial than predicted by IMMO (perhaps because strategy implementation was made easy and quick due to prior training in this study) and leave only a monitoring function specifically to the late stage of reappraisal. Better previous training (resulting in easier implementation) should not reduce, but rather enhance, this effect. Use of a better matched control NR condition (see above) should abolish the effect, because in that case both the R and the NR condition would rely on working memory.

A final potentially interesting observation is that dorsomedial prefrontal reappraisal effects were confined primarily to comparatively posterior sites in the tonic and decreasing main effects (corresponding approximately to supplemental or pre-supplemental motor areas and mid parts of the dACC) but located comparatively more rostrally in the increasing main effect (see **Tables 1–3**), in a region previously linked with self-referential processing (Amodio and Frith, 2006). In a similar fashion, right anterior LFC activation, that we here observed again to increase over reappraisal episodes, has been associated with self-monitoring (reviewed in Kalisch, 2009). Together, these findings would be in accordance with later processing stages comprising a self-monitoring component that serves to determine reappraisal success and perhaps also the correct application of the given reappraisal strategy.

We would like to emphasize that we do not attempt to downplay the important roles that non-frontal areas may play in reappraisal but have chosen to focus this analysis and our theorizing on frontal areas, based on the assumption that these mediate the essential executive process that govern any reappraisal. It may also be useful to point out that IMMO has been developed to better understand the deliberate and effortful *regulation* of emotions, but we cannot exclude that IMMO might also be applicable to situations of deliberate and effortful emotion *generation*. While in the context of the current study, the distinction between emotion generation and regulation is straightforward, there may be instances where the distinction is less clear or perhaps impossible to make (see Gross et al., 2011). Notwithstanding this current debate, we believe that delineating the mental processes that construct an experience or behavior of interest is generally more useful than focusing on broad categories such as "generation" and "regulation" or also "emotion" and "cognition" (Etkin et al., 2011).

To conclude, we hope to have contributed to a further characterization of the functional architecture of reappraisal, both at an algorithmic (cognitive processes) and implementational (neural substrates) level. We are confident that a more precise mechanistic account of reappraisal is an important basis for investigating dysfunctionality in emotion regulation in patients and can give important hints as to how to best improve emotion regulation, be it with psychological, pharmacological, or neurotechnological instruments.

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Affective privilege: asymmetric interference by emotional distracters

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Numerous theories posit that affectively salient stimuli are privileged in their capacity to capture attention and disrupt ongoing cognition. Two underlying assumptions in this theoretical position are that the potency of affective stimuli transcends task boundaries (i.e., emotional distracters do not have to belong to a current task-set to disrupt processing) and that there is an asymmetry between emotional and cognitive processing (i.e., emotional distracters disrupt cognitive processing, but not *vice versa*). These assumptions have remained largely untested, as common experimental probes of emotion–cognition interaction rarely manipulate task-relevance and only examine one side of the presumed asymmetry of interference. To test these propositions directly, a face–word Stroop protocol was adapted to independently manipulate (a) the congruency between target and distracter stimulus features, (b) the affective salience of distracter features, and (c) the task-relevance of emotional compared to non-emotional target features. A three-way interaction revealed interdependent effects of distracter relevance, congruence, and affective salience. Compared to task-irrelevant distracters, task-relevant congruent distracters facilitated performance and task-relevant incongruent distracters impaired performance, but the latter effect depended on the nature of the target feature and task. Specifically, task-irrelevant emotional distracters resulted in equivalent performance costs as task-relevant non-emotional distracters, whereas task-irrelevant non-emotional distracters did not produce performance costs comparable to those generated by task-relevant emotional distracters. These results document asymmetric cross-task interference effects for affectively salient stimuli, supporting the notion of affective prioritization in human information processing.

Keywords: attention, affect, interference resolution, emotional conflict, executive function, conflict, cognitive control, Stroop

INTRODUCTION

Executive control enables organisms to act in accordance with internal goals, promoting the processing of information relevant to current objectives while mitigating distraction from irrelevant information. Several critical functions underlie this ability, including top-down attentional biasing that enhances the processing of goal- or task-relevant information relative to task-irrelevant information (Desimone and Duncan, 1995; Miller and Cohen, 2001). However, both physically salient (Yantis and Jonides, 1990) and affectively salient stimuli are adept at capturing attention in a bottom-up fashion, and may therefore disrupt ongoing goal-oriented processing (LeDoux, 2000; Ohman and Mineka, 2001; Vuilleumier and Huang, 2009). While the privileged access to processing resources exhibited by affectively salient stimuli is essential to rapid responding to stimuli that may convey a potential threat or possible reward, if affective stimuli are too adept at disrupting ongoing mental functions, harmful consequences may result. Indeed, many models of psychiatric disorders have disruptions in the interaction between executive function and affective processing at their core (Bishop, 2007; Banich et al., 2009), highlighting the necessity of characterizing the interactive influence between these two processes.

Theories positing that affectively salient items have privileged access during information processing (LeDoux, 2000; Ohman and Mineka, 2001; Vuilleumier and Huang, 2009) typically entail two key implicit assumptions regarding the properties of emotional stimuli. The first is that affective stimuli have the capacity to transcend task boundaries, disrupting ongoing processing regardless of whether they are relevant to the current task-set of the organism or not. The second is an assumption of asymmetry, whereby affective information interferes with non-affective task-sets more potently than non-affective information interferes with affective task-sets. Importantly, while these two presuppositions provide the foundation for the hypothesized privileged access of affective stimuli to attention, they are rarely ever tested, as common experimental probes of emotion–cognition interaction generally do not manipulate the task-relevance of affective stimuli or the potential for non-affective distracters to interfere with affective processing. The goal of the current experiment was to explicitly test these two assumptions.

The question of affective influences on cognitive processing has prompted the development of experimental paradigms seeking to examine affective modulation of executive function, particularly its influence on attention as gaged in classic conflict processing tasks, such as the Stroop paradigm (Stroop, 1935; Macleod, 1991). In the color–word Stroop task, color words (e.g., "red") are printed in an ink color (e.g., the color red), and the participant identifies the color of the ink while ignoring the semantic meaning of the word. Thus, the ink color is the target, whereas the semantic meaning of the word is the distracter. Typically, performance is both slower and more error prone when the distracter is semantically incongruent with the target (e.g., the word "green" printed in red ink), likely due to the fact that these two features are both semantically conflicting and prime mutually exclusive responses (Kornblum et al., 1990; Macleod, 1991). A well-known adaptation of the traditional Stroop paradigm to the affective domain includes non-affective (e.g., "car") and affectively salient (e.g., "death") words as irrelevant stimulus features, and the potency of emotional distracters is gaged by comparing response times (RTs) between neutral and affective distracter conditions (Mathews and Macleod, 1985; McKenna, 1986; Whalen et al., 1998; Isenberg et al., 1999; Compton et al., 2003). However, this adaptation suffers from several conceptual and practical limitations. First, it does not really constitute an affective equivalent of the classic Stroop task, as the inclusion of affectively salient stimuli in the distracting feature dimension may capture attention but it does not produce semantic or response conflict with the target feature (Algom et al., 2004). For instance, the semantic meaning of a negative affective word is not directly incongruent with the ink color in the same way that the semantic meaning of a color–word would be, nor do affective words prime competing responses in this case. Second, although this adaption could in principle gauge the capacity of task-irrelevant affective stimuli to interfere with an ongoing non-affective task-set (color-naming), it does not probe whether this type of interference is asymmetrical. Finally, if this protocol tests the capacity of affective stimuli to interfere with a non-affective task-set, results obtained with this task should actually raise doubts as to whether affective information is in fact capable of transcending task boundaries, as behavioral findings of an "emotional Stroop effect" have been highly inconsistent, especially in healthy subject populations (McKenna, 1986; Williams et al., 1996; Whalen et al., 1998; Isenberg et al., 1999; Compton et al., 2003).

An alternative approach has been to design tasks where an affective distracter could conflict directly with the target of the ongoing task-set, by combining affective distracters with affective targets and task-sets. One approach is a modified face–word Stroop task, in which participants must make a judgment about a visually presented face image while ignoring an overlaid word label. In affective versions of this task, the faces are modeling affective expressions and the participant must categorize the expression (e.g., happy or fearful) while ignoring an affective word label (e.g., "happy," "fear"; Etkin et al., 2006; Egner et al., 2008). Therefore, the distracter and the target are semantically related, and incongruent distracters would likely generate both stimulus and response conflict. Investigations using this type of paradigm reliably report significant behavioral slowing on incongruent trials (Etkin et al., 2006; Haas et al., 2006; Egner et al., 2008; Ochsner et al., 2009), indicative of a robust effect of emotional conflict. However, this type of protocol does not examine the interaction between affective salience and task-set relevance, leaving the assumption that affective stimuli asymmetrically interfere with non-affective task-sets unexamined. Thus, although tasks employing affective targets, distracters, and task-sets have expanded our knowledge about emotional conflict processing, they preclude the investigation of interactions between task-sets, target features, and affective salience.

In sum, previous paradigms for investigating the impact of affective distracters on non-affective task-sets have produced inconsistent results regarding the capacity of affective distracters to transcend task-set boundaries, whereas tasks employing affective task-sets have only shown a reliable capacity of affective distracters to disrupt performance during an affective task-set. Neither type of protocol has tested whether interference effects across task-sets are asymmetrical in nature, with affective stimuli having a more potent effect than non-affective information. The extent to which affective stimuli are prioritized in human information processing therefore remains unclear.

The current study sought to examine the effects of distracter task-set relevance and affective salience on behavioral performance. A variant of the face–word Stroop task was adapted to independently manipulate distracter congruence with the target stimulus as well as membership in the current task-set, which could involve either an affective or a non-affective judgment of the target. This manipulation enabled the examination of the relative capacity of both affective and non-affective stimuli to transcend task boundaries in altering performance.

MATERIALS AND METHODS

PARTICIPANTS

Thirty-eight healthy college students (Mean Age 19.0 years, SD 1.0; 27 women) participated in this study for course credit. All participants were fluent in English and had normal or corrected-to-normal vision. Participants were screened via self-report to exclude those with previous or current psychiatric or neurologic conditions. All participants gave informed consent and this study was approved for use in human subjects in accordance with the Duke University Medical Center Institutional Review Board.

MATERIALS

Stimuli consisted of black and white face images from the NimStim Set of Facial Expressions[1] (Tottenham et al., 2009) overlaid with word labels. The stimulus set consisted of four female and four male individuals posing both fearful and happy expressions, resulting in a total of 16 distinct face stimuli. Images were cropped to an oval that consisted of the main facial features to standardize image size and shape across the different identities and expressions. Additionally, image cropping removed non-face information, such as hair, which could facilitate performance during

[1]Development of the MacBrain Face Stimulus Set was overseen by Nim Tottenham and supported by the John D. and Catherine T. MacArthur Foundation Research Network on Early Experience and Brain Development. Please contact Nim Tottenham at tott0006@tc.umn.edu for more information concerning the stimulus set.

gender discrimination. There were eight different versions of each of the 16 distinct face stimuli, as each face was paired with gender ("male," "female") and expression ("fear," "happy") word labels that were superimposed on the center of the face images (in Helvetica font and red ink), and which could be displayed in lower or upper case lettering. Thus, the complete stimulus set consisted of 128 unique face–word compound stimuli. Four example stimuli are displayed in **Figure 1**. The face images subtended approximately 12.4˚ of visual angle vertically and 7.2˚ horizontally. The labels subtended approximately 1.4˚ of visual angle vertically and 3.8˚–8.1˚ horizontally. Stimulus presentation and data collection were performed using Psychophysics Toolbox Version 3 (Brainard, 1997; Pelli, 1997) on Dell Optiplex 960 computers with Dell 19″ LCD monitors. A chin rest was use to ensure that participants maintained a distance of approximately 60 cm from the monitor throughout the experiment.

PROCEDURE

After providing informed consent, participants were randomly assigned to either a gender task or an emotion expression task in a face–word Stroop paradigm adapted from previous work (Etkin et al., 2006; Egner et al., 2008). On each trial, participants were presented with a compound face–word stimulus and instructed to make a judgment of the face image as quickly and accurately as possible while ignoring the word label. Participants assigned to the gender task made a gender judgment (male or female) while those assigned to the emotion expression task made an expression judgment (fearful or happy). Responses were made via keyboard presses using the index and middle fingers of the right hand. Stimuli were presented for 1 s and followed by a variable inter-trial fixation interval of 2, 3, or 4 s drawn from a uniform distribution. Following a brief practice, participants completed three runs of 145 trials each, with the first trial in each run serving as a filler to mitigate any preparatory effects.

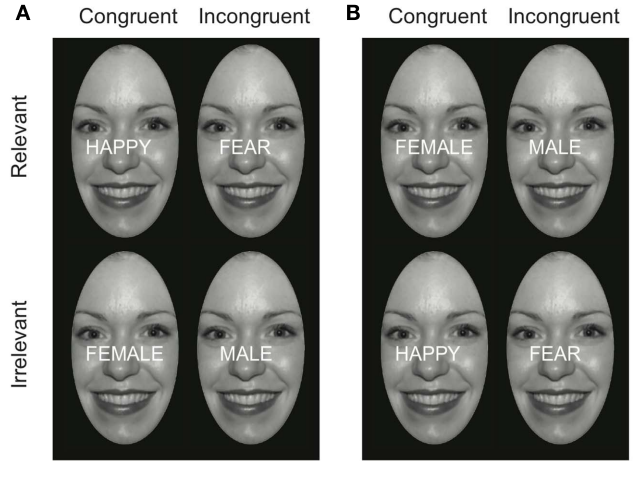

FIGURE 1 | Examples of experimental stimuli for (A) the emotion expression task and (B) the gender task. The experiment varied distracter congruency and relevance by manipulating the labels superimposed on the face stimuli. The labels were printed in red ink in the experiment, but have been presented in white ink here for display purposes.

Similar to previous versions of this task (Etkin et al., 2006; Egner et al., 2008), the labels presented superimposed on the images could be either semantically congruent or incongruent with the target face image. For instance, a fearful male face could be accompanied by a congruent "male" (or "fear") label or by an incongruent "female" (or "happy") label. In a departure from earlier versions, however, the labels were drawn not only from the semantic category that was relevant to the current task-set (e.g., gender labels presented during the gender task) but also from the category that was irrelevant to the current task-set (e.g., emotion labels presented during the gender task; **Figure 1**). We refer to the former as "task-relevant distracters" and to the latter as "task-irrelevant distracters." Importantly, task-relevant distracters could be associated with both (semantic) stimulus conflict and response conflict, because their semantic meaning could clash with the face gender/emotion (potentially inducing stimulus conflict), and in addition their meaning corresponded to a valid response option in the task-set (potentially eliciting response conflict). By contrast, task-irrelevant distracters could not generate response conflict, as they had no correspondence with valid response options in the task-set. Task-irrelevant distracters, however, could be associated with (task-irrelevant) semantic or stimulus conflict, and one of the main goals of this study was to determine whether task-irrelevant stimulus congruency could in fact affect responses to task-relevant stimulus features, and whether this type of effect would be dependent on the affective nature of the distracters. It should be emphasized that the potential semantic or stimulus conflict elicited in the current experiment would be due to completely task-irrelevant distracters and is thus different from task-relevant stimulus conflict that can be evoked, for example, by including response-ineligible colors in a regular color-naming Stroop task (Milham et al., 2001), or by employing many-to-few stimulus–response mappings in flanker or Stroop paradigms (De Houwer, 2003).

Each experimental run was balanced to include equal numbers of trials in each of the four congruence and relevance conditions (i.e., congruent–relevant, congruent–irrelevant, incongruent–relevant, incongruent–irrelevant). Face stimuli in each condition were equally likely to be male or female, fearful or happy. Additionally, the trial order was pseudo-randomized to ensure that there were an equal number of trial transitions between each of the four main conditions. Factors that could impact experimental effects of distracter congruence and relevance, such as repetition priming (Mayr et al., 2003) or cross-trial feature binding effects (Hommel, 1998), were controlled by preventing any repetitions of the same face identity on consecutive trials and alternating each trial between uppercase and lowercase distracter word labels. Thus, no exact perceptual features of either the target or the distracter ever repeated across successive trials.

ANALYSIS

Analyses of categorization accuracy and RT were conducted using SPSS (SPSS Inc., Chicago, IL, USA). RT data exclude error trials and post-error trials. Outlier RT values that were below or above 2 SDs from the participant's grand mean were also removed, resulting in the exclusion of 4.8% of trials on average (SD 0.9%). Two participants whose RTs were more than

2 SDs higher than the sample average in all four RT conditions were excluded from subsequent analyses. The final sample sizes were therefore 19 participants (13 female) completing the emotion expression task and 17 participants (12 female) completing the gender task. Response accuracy and RT data were submitted to separate 2 × 2 × 2 repeated-measures ANOVAs with distracter congruence (congruent versus incongruent) and task-relevance (relevant or irrelevant) as within-subjects factors and task (gender or emotion expression) as a between-subjects factor. Significant results in the main ANOVAs were examined using *t*-tests and ANOVAs. Means and SDs for participant RT and accuracy are reported for each condition in **Tables 1** and **2** respectively.

RESULTS

RESPONSE TIME DATA

Analyses of RT data revealed a main effect of distracter congruence [$F(1,34) = 24.4$, $p < 0.001$], with slower responses to incongruent than congruent stimuli. There were no significant main effects of either distracter relevance or task (p's > 0.8). However, distracter relevance interacted with distracter congruence [$F(1,34) = 26.1$, $p < 0.001$], as the congruency effect was significant only for relevant labels [$t(35) = 6.9$, $p < 0.001$] but not irrelevant labels ($p > 0.8$). Importantly, there was a three-way interaction between distracter congruence, distracter relevance, and task [$F(1,34) = 5.6$, $p = 0.024$, **Figure 2**]. To explore the three-way interaction, 2 × 2 repeated-measures ANOVAs were conducted with distracter relevance as a within-subjects factor and task as a between-subjects factor separately for congruent and incongruent trials. On congruent trials, the main effect of relevance was significant [$F(1,34) = 19.0$, $p < 0.001$], with relevant labels resulting in faster responses than irrelevant labels. Thus, for both tasks, congruent distracters facilitated performance to a greater extent when the distracter was relevant to the current task-set. Neither the main effect of task nor the interaction between distracter relevance and task was significant (p's > 0.1). On incongruent trials, the main effect of relevance was also significant [$F(1,34) = 13.0$, $p = 0.001$], as relevant labels resulted in slower responses than irrelevant labels. Critically, the interaction between distracter relevance and task was significant [$F(1,34) = 4.4$, $p = 0.043$]. Participants completing the emotion expression task exhibited slower RTs on incongruent trials with task-set relevant labels (i.e., affective labels) than those with task-set irrelevant labels (i.e., gender labels) [$t(18) = 3.5$, $p = 0.002$]. However, participants completing the gender task exhibited equally slow RTs on incongruent trials with task-set relevant and irrelevant labels ($p > 0.15$), indicating that the incongruent affective labels interfered with their performance despite their irrelevance to the task-set. Furthermore, RTs did not differ across the congruent and incongruent–irrelevant trials ($p > 0.4$), indicating that affective labels in general interfered with task performance, regardless of congruency. Thus, whereas gender labels did not affect performance during the emotion expression task, affective labels produced marked impairments of performance during the gender task, consistent with the capacity of affective stimuli to capture attention irrespective of their relevance to an ongoing task-set.

Table 1 | Means and SDs of participant response times (ms).

	Emotion expression task Mean (SD)	Gender task Mean (SD)
RELEVANT LABELS		
Congruent	671 (75)	683 (82)
Incongruent	705 (76)	702 (78)
IRRELEVANT LABELS		
Congruent	689 (70)	692 (76)
Incongruent	684 (84)	696 (84)

Table 2 | Means and SDs of participant accuracy.

	Emotion expression task Mean (SD)	Gender task Mean (SD)
RELEVANT LABELS		
Congruent	94.44% (6.42)	92.48% (5.52)
Incongruent	88.55% (10.63)	92.16% (5.56)
IRRELEVANT LABELS		
Congruent	91.67% (7.04)	90.47% (7.18)
Incongruent	93.03% (6.52)	91.50% (6.34)

ACCURACY

Analyses of response accuracy revealed no significant main effects of distracter relevance, distracter congruence, or task (p's > 0.05). There were significant interactions between distracter relevance and task [$F(1,34) = 4.2$, $p = 0.049$], distracter congruence and task [$F(1,34) = 7.0$, $p = 0.012$], and distracter relevance and congruence [$F(1,34) = 13.2$, $p = 0.001$]. However, these interactions were qualified by a significant three-way interaction of distracter relevance, distracter congruence, and task [$F(1,34) = 6.2$, $p = 0.018$, **Figure 3**]. To investigate this three-way interaction, 2 × 2 repeated-measures ANOVAs were conducted with distracter relevance as a within-subjects factor and task as a between-subjects factor separately for congruent and incongruent trials. On congruent trials, the main effect of relevance was significant [$F(1,34) = 14.0$, $p = 0.001$], with higher accuracy on relevant compared to irrelevant congruent trials. Relevant congruent distracters

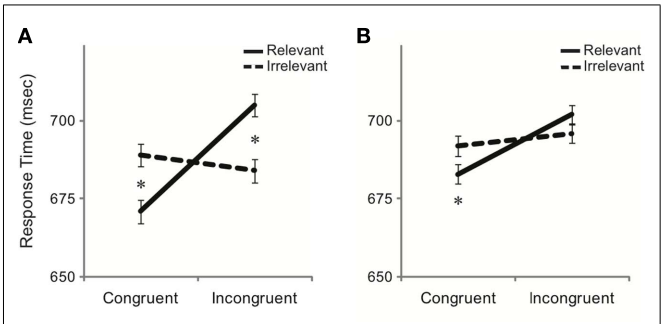

FIGURE 2 | Mean response times by distracter relevance and congruence presented separately for (A) the emotion expression task and (B) the gender task. Error bars represent SE of the mean.

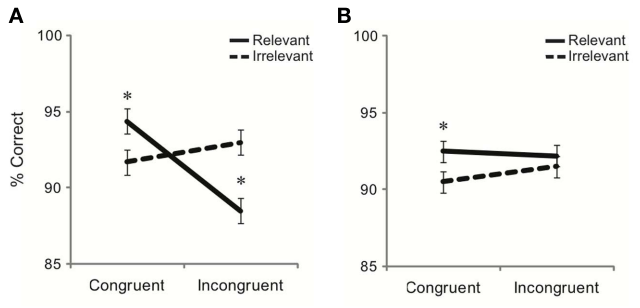

FIGURE 3 | Mean judgment accuracy by distracter relevance and congruence presented separately for (A) the emotion expression task and (B) the gender task. Error bars represent SE of the mean.

facilitated performance relative to irrelevant congruent distracters on both tasks. There was no main effect of task nor was there a significant interaction between distracter relevance and task (p's > 0.5). On incongruent trials, there was a main effect of relevance [$F(1,34) = 4.2$, $p = 0.047$], with lower accuracy in response to relevant than irrelevant incongruent distracters. Importantly, there was an interaction between distracter relevance and task [$F(1,34) = 7.6$, $p = 0.009$]. Paired t-tests comparing accuracy on irrelevant and relevant incongruent trials showed a significant difference for participants performing the emotion expression task [$t(18) = 2.9$, $p = 0.009$], such that accuracy was lower on relevant compared to irrelevant incongruent trials. No such difference was found for participants performing the gender task ($p > 0.5$). Accuracy on irrelevant congruent and incongruent stimuli did not differ ($p > 0.2$), indicating that affective labels interfered with performance, irrespective of their congruency, and despite being irrelevant to the current task-set. Consistent with the results from analyses of RT, these findings indicate that the irrelevant emotion labels in the gender task asymmetrically disrupt task performance compared to the irrelevant gender labels in the emotion expression task.

EFFECTS OF TARGET VALENCE

The observed interaction between task-set, distracter relevance, and affective salience could additionally be modulated by the affective content of the target stimulus. Although all the target face stimuli modeled an affective expression (either happiness or fear) and were counterbalanced across the cells of the factorial design, the specific affective valence of the target stimuli could nevertheless interact with observed effects of distracter relevance and affective salience. To interrogate this possibility, additional analyses were conducted in which trials were classified based on target valence (happy or fear expression) as well as distracter relevance and congruence. These analyses revealed no interactions between target valence and the distracter relevance by congruence by task-set interaction described above for either RT or accuracy (p's > 0.25). The present findings, therefore, appear to be driven by the affective nature of the distracters and their relevance to the current task-set as opposed to differences in affective valence of the targets in this task. The absence of an effect of target valence is consistent with the findings from a number of previous studies of emotion–cognition interactions (Compton et al., 2003; Etkin et al., 2006; Haas et al., 2006; Egner et al., 2008; Ochsner et al., 2009).

DISCUSSION

The present study adapted a face–word Stroop paradigm to investigate two underlying assumptions of theories postulating prioritized processing of affective stimuli: that affective stimuli can disrupt processing across task boundaries and that this capacity is not shared by non-affective stimuli. Analyses of the interaction between distracter congruence, distracter affective salience, and task-set relevance revealed that each of these factors modulates task performance. While the basic pattern of responses in the two task-sets was quite similar, the analyses revealed some significant distinctions: for both affective and non-affective task-sets, incongruent distracters relevant to the current task-set resulted in slower RTs and lower judgment accuracy compared to congruent distracters, due to the generation of semantic and response conflict. However, the influence of distracters outside the task-set on performance was driven by an interaction between affective salience and task-set. In the presence of a non-affective task-set, gender categorization, affective labels markedly slowed RTs and reduced judgment accuracy despite their irrelevance to the present task-set, demonstrating the capacity of affectively salient stimuli to disrupt ongoing processing and transcend task boundaries. This effect was specific to the task-irrelevant affective distracters, as non-affective, task-irrelevant distracters did not similarly impair performance in the presence of an affective task-set, the emotion expression categorization. Furthermore, this effect was not dependent on the congruency of the affective word distracter with respect to the equally task-irrelevant affective facial expression, indicating that task-irrelevant affective salience interfered with task performance regardless of congruency. In sum, affectively salient distracter stimuli, but not non-emotional distracter stimuli, interfered with ongoing performance despite their irrelevance to current task-set, indicating that affective stimuli have the capacity to transcend task boundaries and disrupt ongoing executive processes.

The present findings extend those from previous investigations of the interaction between affective processing and executive function. Previous adaptations of the Stroop paradigm utilizing task-irrelevant affective distracters and non-affective task-sets (such as adaptations of the color–word Stroop) examined whether affective stimuli could disrupt ongoing non-affective top-down processing but have met with mixed results (Mathews and Macleod, 1985; McKenna, 1986; Whalen et al., 1998; Isenberg et al., 1999; Compton et al., 2003). Other adaptations utilizing task-relevant affective distracters in the presences of affective task-sets exhibited robust interference effects but did not speak to the capacity of affective stimuli to disrupt non-affective processing (Etkin et al., 2006; Egner et al., 2008; Ochsner et al., 2009). Finally, neither of the sets of studies above addressed the question as to whether affective distracters have a greater potency for cross-task interference than non-affective distracters. By contrast, the present study manipulated the affective nature and task-relevance of target and distracter features independently, and demonstrated the asymmetry of these

interference effects. Affective stimuli appear to disrupt ongoing processing regardless of the task-set, while non-affective stimuli seem to impair performance only in the presence of relevant task-sets and response contingencies.

The capacity of affectively salient stimuli to override top-down biasing may be adaptive under certain circumstances. Affective stimuli may help direct attention to, and thus improve the detection of, stimuli that convey biologically relevant information, such as threat (Ohman and Mineka, 2001; Vuilleumier and Huang, 2009; Notebaert et al., 2011) or reward (Maunsell, 2004; Field et al., 2009; Krajbich et al., 2010). The prioritized processing of affective stimuli may also improve the speed and sensitivity of processing of related information (Anderson and Phelps, 2001; Vuilleumier et al., 2001; Phelps et al., 2006; Pourtois et al., 2006; Lim et al., 2009), therein enabling rapid, appropriate responding to salient information. Moreover, affectively salient stimuli can help mitigate the influence of factors that might otherwise impair executive function, such as by eliminating Stroop dilution effects (Chajut et al., 2010) and overcoming the attentional blink (Anderson and Phelps, 2001; Lim et al., 2009). Thus, the ability of affective stimuli to alter top-down processing can help promote adaptive responding.

The interaction between affective processing and executive function may, however, be disrupted in certain clinical disorders (Bishop, 2007; Banich et al., 2009). Heightened levels of anxiety, for instance, may result in hyper-vigilance for threat, and afflicted individuals may have difficulty disengaging from affective stimuli which disrupt ongoing top-down processing (Fox et al., 2001; Koster et al., 2004; Salemink et al., 2007), diminishing their ability to respond adaptively to subsequent stimuli and function normally. In the present study, all participants were screened for psychological and neurological disorders and subclinical variability in anxiety and depression were not assessed; however, the present findings anticipate that high-anxious individuals are likely to display greater cross-task interference from affective distracters than low-anxious individuals. Future studies should investigate the interactions between affective processing and executive function in relation to individual differences and clinical populations, ideally in conjunction with neuroimaging techniques, to elucidate the mechanisms underlying clinical disorders.

The present study documented the capacity of affectively salient distracters to interfere with ongoing processing despite irrelevance to the current task-set. This interference could be produced by rapid attentional orienting to the affective distracters, difficulty disengaging attention from the affective distracters, or some combination of both orienting and disengagement processes. Clarifying the differential roles of orienting and disengagement processes in selective attention has proved critical to elucidating the processes underlying selective attention to threat, particularly amongst anxious individuals (Fox et al., 2001; Koster et al., 2004; Salemink et al., 2007). However, in the present paradigm, the influences of these two processes cannot be distinguished. Future research should endeavor to characterize the unique contributions of orienting and disengagement to the type of affective interference documented in the current experiment. The present findings also indicate that the capacity of distracters to transcend task boundaries in generating interference is restricted to affectively salient distracters and not shared by non-affective distracters. Note though that non-affective distracters may be capable of crossing task-set boundaries in generating interference when the relevant task is non-affective in nature. The affective nature of a task-set may help buffer it from task-irrelevant interference generally, producing the lack of observed interference by non-affective task-irrelevant distracters. Either increased cross-task interference produced by affective distracters or enhanced task-set shielding for affective task-sets would support the asymmetrical effects of interference reported here.

In conclusion, through the adaptation of a face–word Stroop paradigm, the present study tested two key assumptions of theories of prioritized processing of affective stimuli, namely, that such stimuli can transcend task-set boundaries and that this capacity is not shared by non-affective stimuli. The current findings support both of these assumptions, demonstrating that affective stimuli disrupt top-down processing regardless of task-set, but that non-affective stimuli only produce interference if relevant for the task-set at hand. This asymmetric modulation of executive function confirms the prioritized processing of affective stimuli and highlights the importance of understanding the interactive influence of affect and cognition.

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A tribute to Charlie Chaplin: induced positive affect improves reward-based decision-learning in Parkinson's disease

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Reward-based decision-learning refers to the process of learning to select those actions that lead to rewards while avoiding actions that lead to punishments. This process, known to rely on dopaminergic activity in striatal brain regions, is compromised in Parkinson's disease (PD). We hypothesized that such decision-learning deficits are alleviated by induced positive affect, which is thought to incur transient boosts in midbrain and striatal dopaminergic activity. Computational measures of probabilistic reward-based decision-learning were determined for 51 patients diagnosed with PD. Previous work has shown these measures to rely on the nucleus caudatus (outcome evaluation during the early phases of learning) and the putamen (reward prediction during later phases of learning). We observed that induced positive affect facilitated learning, through its effects on reward prediction rather than outcome evaluation. Viewing a few minutes of comedy clips served to remedy dopamine-related problems associated with frontostriatal circuitry and, consequently, learning to predict which actions will yield reward.

Keywords: Parkinson's disease, positive affect, frontostriatal circuitry, probabilistic learning

INTRODUCTION

Parkinson's disease (PD) is a neurodegenerative process commencing in the midbrain, in particular affecting dopaminergic neurons of the substantia nigra projecting into the dorsolateral striatum (mostly the putamen; Bjorklund and Dunnett, 2007), resulting in motor deficits, such as tremor, bradykinesia, and rigidity (McAuley, 2003). As the disease progresses, dopamine (DA) depletion affecting cognitive circuits of the basal ganglia contribute to impairments in a range of cognitive domains, including reinforcement learning, reversal learning, risky decision-making, working memory, response inhibition, and speed/accuracy balancing (e.g., Cooper et al., 1992; Swainson et al., 2000; Cools et al., 2001; Frank, 2005; Moustafa et al., 2008; Wylie et al., 2009, 2010; Claassen et al., 2011). The purpose of the present investigation was to determine whether reward-based learning deficits in patients with PD might be remedied non-invasively by factors that induce positive affect.

Induced positive affect yields improved performance in a variety of tasks that rely on frontostriatal dopaminergic interactions, including antisaccade tasks, task switching, and varieties of Go/NoGo tasks such as the AX-CPT (Dreisbach and Goschke, 2004; Dreisbach et al., 2005; Dreisbach, 2006; Van der Stigchel et al., 2011; van Wouwe et al., 2011a). Interestingly, patients with PD show performance impairments in each of these tasks (e.g., Kitagawa et al., 1994), suggesting that performance improvements after positive affect might result from changes in dopaminergic levels in the brain. Before discussing how induced positive affect might remedy PD-related deficits in reward-based decision-learning, we first turn to a brief exposition of the neurocognitive bases of such reinforcement learning.

NEUROCOGNITIVE MECHANISMS UNDERLYING REWARD-BASED DECISION-LEARNING

Decisions about how best to respond in a situation are often guided by past learning of the relations between events, actions, and their outcomes. Probabilistic reward-based decision-learning paradigms enable us to measure the process of learning (through trial-and-error) associations between stimuli, actions, and their related rewards. Several brain areas have been linked to key aspects of reward-based decision-learning, including prefrontal regions (e.g., the dorsolateral and orbitofrontal cortices) and the basal ganglia. Additionally, the neurotransmitter DA plays a modulatory role in these functions through projections from midbrain DA nuclei to the striatum and cortical areas (Schultz, 2006).

Lesion and human imaging studies support a functional dissociation between the contributions of various regions within the striatum to reward-based decision-learning (for an overview, see Balleine et al., 2007). In addition to the role of dorsal versus ventral striatum in different aspects of reward-based learning (Knutson et al., 2001; McClure et al., 2003; O'Doherty et al., 2004; Seger and Cincotta, 2005; Seymour et al., 2007), recent fMRI work suggest

that distinct regions within dorsal striatum may contribute to different phases of learning (Haruno and Kawato, 2006a).

A *Q*-learning model can be used to generate individual parameters that reflect two important aspects of learning. First, the mismatch between anticipated rewards and actual rewards is computed as a reward prediction error (RPE), which learners use for adjusting decision-making on future trials, in particular in the early stages of learning when they rely on feedback to determine which actions maximize rewards. Haruno and Kawato (2006a) observed that higher RPE values were associated with activation of the caudate nucleus and ventral striatum and their associated frontal circuitry (orbitofrontal, dorsolateral prefrontal, and anterior cingulate cortex), involved in generating and testing hypotheses regarding reward optimization (c.f. Alexander et al., 1990; Oyama et al., 2010). Second, as learning progresses, participants attempt to forecast which actions will likely yield reward (or avoid punishment); this is computed as the stimulus-action-dependent reward prediction (SADRP). Higher SADRP values reflect more effective learning of stimulus-action-reward associations, and hence, are maximal at the later stages of the task. Haruno and Kawato (2006a) reported higher SADRP values to be associated with activation of the anterior putamen and its associated motor circuitry (supplementary motor area, premotor and primary motor cortex), involved in integrating information on the expectation of reward with processes that mediate the actions leading to the reward (c.f. Alexander et al., 1990; Gerardin et al., 2003).

To explain these patterns, the authors proposed that the caudate (embedded in the cortical striatal loop which includes the orbitofrontal cortex and dorsolateral prefrontal cortex) is involved in generating and testing hypotheses regarding reward optimization. Global reward-related features of the stimulus-action-reward associations are propagated from the caudate to motor loops (which include the putamen and premotor areas) by means of a dopamine signal that is subserved by reciprocal projections between the striatum and the substantia nigra (Haruno and Kawato, 2006b). During later stages of learning, putamen activity increases with reward predictions (i.e., with learning SADRPs). Activity in the putamen increases to incorporate more specific motor information with the associated stimuli and the expected reward; that is, the reward associated with a specific stimulus and a specific action becomes more predictable and learning is gradually fine-tuned (Haruno and Kawato, 2006b). As these SADRP values increase, the RPE is reduced as subjects more accurately anticipate the rewards associated with their actions. Note that the change in emphasis from RPE during early phases of learning to SADRP during later stages bears resemblance to the dynamics of phasic DA-activity as a function of learning. The phasic DA bursts displayed by striatal neurons in response to reward have been reported to shift in time from the presentation of unexpected reward during early phases of learning to the presentation of conditioned reward-predicting stimuli during later stages (Schultz et al., 2003; Balleine et al., 2007).

REMEDIES FOR COGNITIVE IMPAIRMENTS IN PARKINSON'S DISEASE

DA medication in Parkinson's patients, serving to increase dopaminergic influx into the striatum, improves the efficacy of using incoming response-relevant stimulus information to control behavior (Cools et al., 2001, 2007). Reward-based learning benefits from DA medication, specifically for learning that certain actions are likely to yield reward (Frank, 2005; Shohamy et al., 2005; Bodi et al., 2009). Because regions of the striatum are differentially affected by PD, DA medication may differentially affect these structures and their related functions. Using the *Q*-learning approach sketched above, van Wouwe et al. (2012) observed that DA medication improved SADRP (i.e., reward prediction, presumably supported by the anterior putamen and associated motor circuitry), but did not affect RPE (i.e., outcome evaluation, presumably supported by the caudate and ventral striatum and associated frontal circuitries). Similar effects were observed for the effects of deep brain stimulation of the subthalamic nucleus (van Wouwe et al., 2011b).

To the extent that impaired decision-learning in patients with PD follows from the decline in their striatal DA-system, one might suppose that *any* intervention that enhances dopaminergic functionality may serve to remedy the learning deficit. In fact, mild increases in DA-activity in the reward-processing system can be triggered by a broad spectrum of positive reinforcers (Burgdorf and Panksepp, 2006). One simple, non-invasive, and even agreeable means to trigger mild increases in DA levels is the induction of positive affect (a mood state characterized by subjective well-being and happiness; Ashby et al., 1999, 2002). Recent neuroimaging studies in humans have demonstrated that funny cartoons, implicit laughter, affectively positive music, and positive (as opposed to negative) emotional pictures can activate reward-related areas. According to a neurobiological theory on the influence of positive affect (Ashby et al., 1999; Ashby et al., 2002), induced positive affect leads to temporary increase of dopamine release in midbrain DA-generation centers. This dopamine release is subsequently propagated to dopaminergic projection sites in the prefrontal cortex and the striatum. Only a limited amount of DA transporters is available to remove DA from the synaptic cleft; hence, once boosted, DA levels will remain elevated for some period of time after affect induction. Together, these findings suggest a neurobiological link between positive affect and a transient but functional boost in DA.

Positive affect can be induced by commonplace methods, including watching comedy movie clips, experiencing success on an ambiguous task, self-recall of positive emotional states, and administering small unexpected rewards. These positive feelings last for approximately 30 min, a time course similar to that of DA-release in the ventral striatum induced by brief electrical stimulation (Floresco et al., 1998). Behavioral influences of positive affect are thought to be mediated by the same tonic dopaminergic neural mechanisms that mediate reward. We predict that PD-related impairments in reward-based decision-learning will be remedied by watching brief feel-good movie clips.

THE PRESENT STUDY

The present study investigates the effect of induced positive affect on reward-based decision-learning. PD patients performed the previously mentioned probabilistic learning task (Haruno and Kawato, 2006a) after watching either Charlie Chaplin slapstick movie clips (between-subjects) or affect-neutral documentary

clips. We determined the effect of induced affect on RPEs, in particular during the early phase of learning, and on formation of stimulus-action-reward associations (SADRP), in particular during more progressed phases of learning. Based on recent findings on the effects of DA medication on reward-based learning in this task (van Wouwe et al., 2012), we expect that positive affect will help improve the putamen-based process of predicting which action will yield reward (reflected by SADRP in late stages of learning) more than the caudate-based process of outcome evaluation (expressed in RPE early during learning).

MATERIALS AND METHODS

PARTICIPANTS

A total of 51 PD patients participated in the experiment after giving written informed consent. They were recruited through Dutch national websites dedicated to PD, and received a small present in return for their participation. All patients had normal or corrected-to normal vision, and no Parkinson-unrelated neurological or psychiatric history according to self-report. Patients were tested individually at their homes. They were asked to abstain from drinking coffee during the hour before testing, and to continue taking their medication at the required time on the day of testing. Tests were planned 60–90 min after regular medication intake. In addition to monoamine oxidase (MAO-B)/catechol-*O*-methyl transferase (COMT) inhibitors ($N = 29$), patients received either dopamine precursors only (levodopa; $N = 10$), agonists only (pramipexole, ropinirole, pergolide, amantadine, or apomorphine; $N = 9$), levodopa plus agonists ($N = 32$), or neither ($N = 1$). Explorative analyses indicated that there was no difference between the neutral and positive affect groups in terms of daily levodopa dosage [$t(49) = 0.129$, $p = 0.898$], agonists dosage [above- versus below-average versus no agonist, $\chi^2(1, 51) = 0.644$, $p = 0.725$], or years since formal diagnosis [$t(49) = 0.259$, $p = 0.797$].

Each of the patients was assigned randomly to one of two affect induction groups. The two groups ($N = 24/27$ for neutral and positive affect groups, respectively) did not differ in terms of age [$M = 62/59$, st.dev $= 9.7/10.5$, $t(49) = 0.914$, $p = 0.365$], years of disease since diagnosis [$M = 7.0/6.7$, st.dev $= 4.8/4.5$, $t(49) = 0.259$, $p = 0.797$], daily dosage (mg/day) of levodopa [$M = 404/393$, st.dev $= 311/327$, $t(49) = 0.129$, $p = 0.898$], level of education [$t(49) = 0.658$, $p = 0.514$], and male/female composition [$\chi^2(1, 51) = 1.663$, $p = 0.197$].

PROCEDURES

The experiment consisted of a training session (two practice blocks, see *Task*), a mood measurement, a film clip for affect induction, another mood measurement, an experimental session (two test blocks, see *Task*), a third mood measurement, and an exit session (consisting of general health questionnaires, a brief exit interview, and debriefing), all frequently interrupted with short breaks. The whole session lasted 60 min maximum. All experimental procedures were approved by a local ethics committee and by the board of the Dutch Parkinson Patiënten Vereniging, and were conducted in accordance with the Helsinki Declaration, international laws, and institutional guidelines.

AFFECT INDUCTION

Affect induction was operationalized by showing the patients film clips that lasted 8–9 min. One group of patients (referred to as the Positive Affect group) watched a slapstick clip from the Charlie Chaplin movie *City Lights* (the famous boxing scene, ending just prior to the part where Chaplin loses the fight). The other patients (referred to as the Neutral Affect group) watched a clip from a Dutch documentary on toll for heavy-traffic on the German Autobahn. The clips were played on a 17″-widescreen laptop computer. Mood was measured three times: immediately before and after affect induction, and immediately after the experimental session. We used a short Manekin test in which mood (valence, from negative to positive) and arousal (from not aroused to highly aroused) were scored on a 5-point Likert scale ranging from −2 to +2 (Hutchison et al., 1996).

TASK AND APPARATUS

A probabilistic learning task, adapted from Haruno and Kawato (2006a), was implemented on a 17″-widescreen laptop computer, placed at a distance of ∼60 cm in front of the participant. Stimuli consisted of colored fractal pictures against a white background. Responses to stimuli were right or left button presses registered by comfortable response keys (see **Figure 1**; the computer keyboard was shielded with a perspex plate such that hands and wrists could rest on the plate, which minimized tremor and prevented unintentional depressing of other keys).

Subjects were instructed that the goal of the task was to make as much money as possible by pressing a left or a right button to each picture stimulus that appeared on the computer screen. Each response provided the chance to either win or lose €0.25 in game

FIGURE 1 | Laptop computer with adjusted response buttons. The computer keyboard was shielded with a perspex plate such that hands and wrists could rest comfortably on the plate, which minimized tremor and prevented unintentional depressing of other keys.

money (note: participants were not remunerated for their participation). **Figure 2** depicts the sequence of a trial from the task. Each trial began with the presentation of one of three picture stimuli (colored fractals) in the center of the screen. The picture stimulus subtended visual angles of 5.67° × 4.41° (9 cm × 7 cm) and remained on the screen for 1000 ms. Participants were instructed to view the picture stimulus, but not to respond until the picture stimulus disappeared and was replaced by a response screen. The response screen consisted of the fixation cross and two blue boxes displayed at the bottom left and bottom right portions of the screen, respectively (see **Figure 2**). Upon the presentation of the response screen, the participant was instructed to make a left or a right button press, which would then be indicated on the screen by a change in color (from blue to green) of the box that corresponded to the response side that was chosen (left button press = left box turns green). The participant was given 2 s to issue a response. After the button press was indicated on the screen for 500 ms, a large box with feedback appeared in the center of the screen for 2000 ms. If the participant chose the correct response, the large box appeared in green, indicating that €0.25 had been won. If the incorrect response was chosen, the box appeared in red, indicating that the participant had lost €0.25. Throughout the entire trial, and throughout the entire block, a running tab of the total amount of money won by the participant was depicted in the upper center portion of the screen. Thus, if the participant won or lost €0.25 on a particular trial, the running total was immediately updated.

Participants completed a practice block in which they learned for each of the three picture stimuli which response led to reward, until they selected the correct button five subsequent times for all stimuli (max 60 trials). This practice block was non-probabilistic, so as to acquaint the patients with the general task and set-up. Next, participants completed a practice block of 21 trials in which the reward outcome of each response to a picture stimulus was determined. For each picture, one response hand was assigned as the optimal choice and the other response hand was designated as the non-optimal choice; selecting the optimal response hand resulted in a 90% probability of winning €0.25 and a 10% probability of losing €0.25; the probabilities of winning versus losing were reversed for the non-optimal response hand. As an example, a left response to fractal stimulus *X* yielded a €0.25 reward with a probability of 0.9 (90%) and a €0.25 loss with a probability of 0.1 (10%). A right response to the same stimulus yielded a €0.25 loss with a probability of 0.9 and a €0.25 with a probability of 0.1. Therefore, the optimal behavior for fractal stimulus *X* was to press the left button, which participants had to learn by trial-and-error. The dominant probabilities for optimal behavior regarding the other fractal stimuli were also 0.9; the optimal response for each fractal was pseudo-randomized over left and right hands, such that the left response was optimal for one or two stimuli whereas the right response was optimal for the remaining stimuli.

Next, after the affect induction session, participants completed two experimental blocks of 60 trials each. In the first block, the probabilities were 90:10 as described above; in the second block, the probabilities were 80:20. For each training and experimental block, a novel set of three picture stimuli was used, and the specific response options were randomly mapped onto each of the fractals. Across blocks and across participants, left and right hand dominant response patterns occurred equally often. Additionally, the fractal stimuli were presented pseudo-randomly (with equal frequency) within a block.

DATA ANALYSIS

Computational model to calculate SADRP and RPE

A reinforcement model (*Q*-learning; Sutton and Barto, 1998) was used to compute each participant's SADRP and RPE during learning. *Q*-learning is an implementation of a temporal difference model which assumes that stimulus-action-reward associations are acquired as a single representation during learning. The SADRP value (Q) consists of the predicted amount of reward for a certain decision (left or right response, r) made for a specific stimulus (one of three fractal stimuli, FS). Thus, the value of SADRP on trial t is the value of Q associated with the particular stimulus and response on that trial. This value thus relates reward to sensory input and actions. Individual predicted reward values (SADRPs) for each action (two possible responses) and each fractal stimulus (three different fractal stimuli) are calculated at time t, Q_t (FS, r) which adds up to six SADRP values per block. The RPE represents the actual reward received (R_t) minus the expected reward, $\text{RPE} = R_t - Q_t$ (FS, r). For the next occurrence of the same stimulus and action, SADRP and RPE values are updated according to the "*Q*-learning algorithm" to maximize reward (Sutton and Barto, 1998)

$$Q_{t+1}\,(\text{FS}, \text{r}) = Q_t\,(\text{FS}, \text{r}) + a_t^{\text{FS}}\,(R_t - (Q_t\,(\text{FS}, \text{r})))\,.$$

The learning rate a_t^{FS} is updated separately for each FS according to the following rule:

$$a_t^{\text{FS}} = \frac{\left(a_{t-1}^{\text{FS}}\right)}{1 + a_{t-1}^{\text{FS}}}.$$

The formula of this learning rate is often used in reinforcement learning studies or studies on adaptive control (Young, 1984; Bertsekas and Tsitsiklis, 1996; Dayan et al., 2000; Haruno and Kawato, 2006a,b). It provides an estimation of a learning parameter which is updated recurrently with the presentation of a stimulus. In the current study, a_t^{FS} reduces with the presentation of each fractal stimulus, but remains equal if a specific FS is not presented. The initial value of the learning rate was set to 1, and was previously observed not to affect the estimation of SADRP and RPE (Haruno and Kawato, 2006a).

The learning rate (a_t^{FS}) decreases toward the end of the learning stage (when SADRP becomes reliable). This is an important feature of a_t^{FS} because it means that, at the end of learning, the SADRP is less affected by an unexpected RPE (due to the probabilistic nature of the task).

Reward prediction errors are expected to be large early on during learning (i.e., first 20 trials), and small later on (i.e., the last 20 trials). By contrast, the SADRP value is expected to be small during initial phases of learning, but to increase and converge to an asymptotic value as learning progresses.

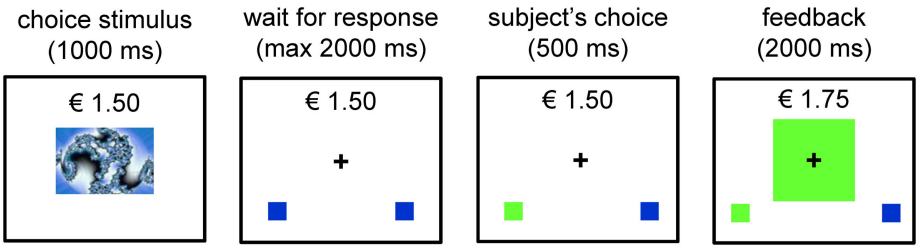

FIGURE 2 | Trial example of the probabilistic learning task adapted from Haruno and Kawato (2006a). In the example, the subject receives a reward by pressing the left button with this specific stimulus.

Statistical analyses

To test for the efficacy of affect induction, a Mann–Whitney U test was used to compare mood and arousal between groups across the three measurements.

For the probabilistic learning task, average reward per trial, average SADRP value per trial, and average RPE value per trial in each block were analyzed by a repeated-measures analysis of variance (RM-ANOVA) including the within-subject variables Stage (first, second, and third part of the block, comprising 20 trials each) and Probability (90/10, 80/20) and the between-subjects variable Affect Group (Positive, Neutral). Where appropriate, the effects of Stage were examined further using linear and quadratic contrast analyses.

RESULTS

AFFECT INDUCTION

Before the film clips, mood (as indexed by the Manekin score on the valence question) was comparable across Affect groups ($M_{POS} = 0.88$, $M_{NEU} = 0.83$; Mann–Whitney $U = 323.500$, $p = 0.992$). Immediately after the film clips, mood was elevated in the Positive compared to Neutral group ($M_{POS} = 1.16, M_{NEU} = 0.54$; Mann–Whitney $U = 174.000$, $p = 0.003$), and within the Positive group after compare to before affect induction [$F(1, 26) = 4.24, p = 0.05$]. After the experimental blocks, mood levels were equal again ($M_{POS} = 1.00, M_{NEU} = 1.04$; Mann–Whitney $U = 304.000, p = 0.672$). Thus, the positive affect induction was successful, albeit short-lived. Arousal was not different between groups at any stage (all $F < 1.8$).

PROBABILISTIC LEARNING: REWARD

Not surprisingly, average reward per trial (in €) increased as a function of learning Stage [$M_{first} = 0.059$, $M_{second} = 0.099$, $M_{third} = 0.110$; $F(2, 98) = 18.49$, $p < 0.001$; linear contrast: $p < 0.001$, quadratic contrast: $p = 0.023$]. Probability also produced a main effect on reward, such that better performance was seen in the 90:10 compared to 80:20 blocks [$M_{90:10} = 0.120$, $M_{80:20} = 0.058$; $F(1, 49) = 44.93$, $p < 0.001$]. This effect of Probability remained constant across Stages [$F(2, 98) = 0.69$].

Positive affect induction exerted a beneficial effect on reward per trial [$M_{POS} = 0.096$, $M_{NEU} = 0.082$; $F(1, 49) = 5.12$, $p < 0.028$]. This influence of Affect was seen for early, middle, and late Stages of learning alike [$F(2, 98) = 1.55$], remained constant across 90:10 and 80:20 Probabilities [$F(1, 49) = 2.11$], and did not engage in a three-way interaction with Stage and Probability [$F(2, 98) = 0.55$].

PROBABILISTIC LEARNING: RPE

As expected, the average RPE per trial (in €) was observed to diminish from early to later Stages of learning [$M_{first} = 0.191$, $M_{second} = 0.152$, $M_{third} = 0.148$; $F(2, 89) = 65.29$, $p < 0.001$; linear contrast: $p < 0.001$, quadratic contrast: $p < 0.001$]. Probability also affected RPE, such that smaller RPEs were observed in the 90:10 compared to 80:20 blocks [$M_{90:10} = 0.138$, $M_{80:20} = 0.192$; $F(1, 49) = 79.59, p < 0.001$]. The effect of Stage varied across Probabilities [$F(2, 98) = 4.14$, $p = 0.019$]; as can be seen in **Figure 3A**, the diminution of RPE as a function of learning was steeper in the 90:10 than 80:20 probability condition. Thus, patients learned to reduce their prediction errors over time, especially in the easier condition.

Positive affect induction failed to exert any effect on RPE [main effect of Affect: $F(1, 49) = 2.32$; Affect × Stage: $F(2, 98) = 0.65$; Affect × Probability: $F(1, 49) = 0.002$; Affect × Stage × Probability: $F(2, 98) = 0.66$].

PROBABILISTIC LEARNING: SADRP

In line with expectations, the average SADRP per trial (in €) was observed to increase from early to later Stages of learning [$M_{first} = 0.046$, $M_{second} = 0.081$, $M_{third} = 0.097$; $F(2, 89) = 64.29$, $p < 0.001$; linear contrast: $p < 0.001$, quadratic contrast: $p < 0.008$]. Probability also affected SADRP, such that smaller SADRPs were observed in the 80:20 compared to 90:10 blocks [$M_{90:10} = 0.099$, $M_{80:20} = 0.048$; $F(1, 49) = 30.53$, $p < 0.001$]. The effect of Stage varied across Probabilities [$F(2, 98) = 4.32, p = 0.016$]; as can be seen in **Figure 3B**, the 90:10 probability condition showed a steep increase of SADRP as a function of learning, whereas no such increase was seen in the 80:20 condition. Thus, patients learned to predict which stimulus-action combinations yielded reward, but only in the easier Probability condition.

Positive affect induction exerted a beneficial effect on SADRP [$M_{POS} = 0.096$, $M_{NEU} = 0.082$; $F(1, 49) = 4.48$, $p < 0.039$]. This influence of Affect differed across learning Stages [$F(2, 98) = 3.19$, $p = 0.045$]. As can be seen in **Figure 4**, SADRP was low in the early stage of learning for positive and neutral Affect groups alike, but the increase during later stages was steeper for the positive Affect group, suggesting that positive affect facilitates learning that response X to stimulus Y is likely to yield reward. The influence

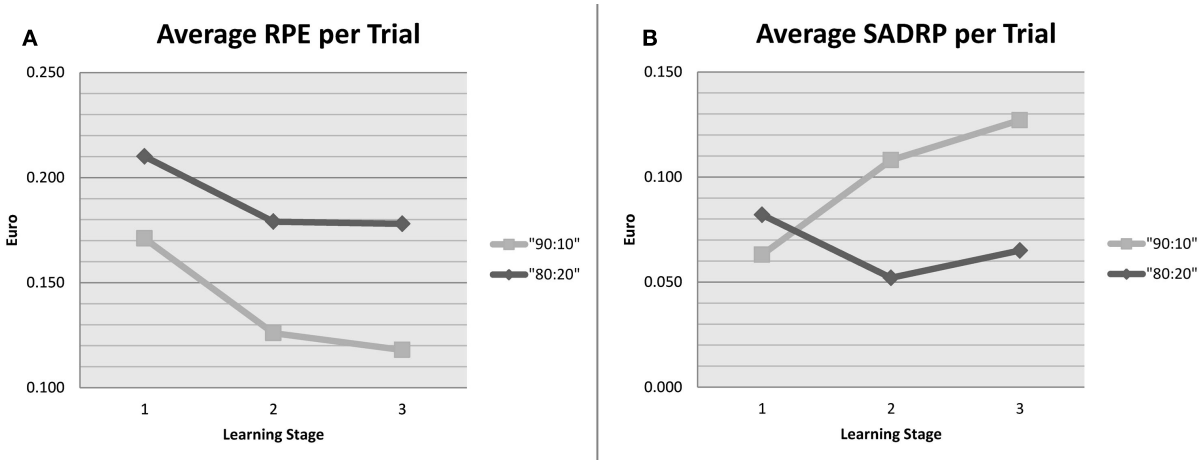

FIGURE 3 | *Q*-model parameters values during stages 1 (trial 1–20), 2 (trial 21–40), and 3 (trial 41–60) of learning, separately for each probability condition. (A) Average RPE values per trial. **(B)** Average SADRP values per trial.

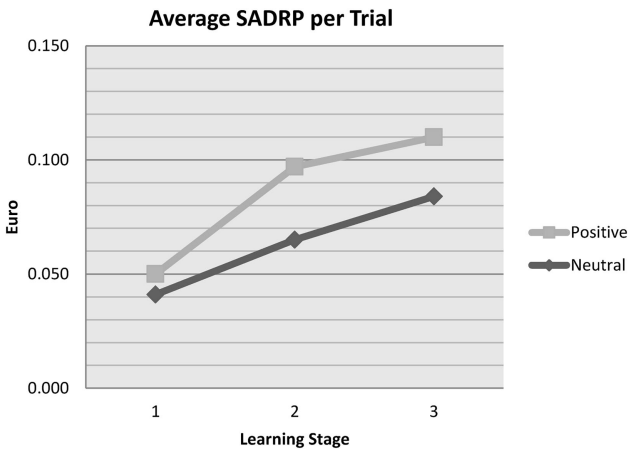

FIGURE 4 | Average SADRP values per trial, separately for each Affect group, during stages 1 (trial 1–20), 2 (trial 21–40), and 3 (trial 41–60) of learning, collapsed across the 90:10 and 80:20 conditions.

of Affect was similar for the 90:10 and 80:20 Probabilities [$F(1, 49) = 0.79$], and did not engage in a three-way interaction with Stage and Probability [$F(2, 98) = 0.13$].

DISCUSSION

Induced positive affect was expected to influence distinct components of reward-based learning in patients with PD. We investigated the effects of positive affect induction (i.e., watching Charlie Chaplin slapstick movie clips) on outcome evaluation (the processing of RPEs to update hypotheses) and reward anticipation (the formation of SADRP) that have been tied to distinct regions in the striatum and their associated circuitries. For learning to be successful, subjects must evaluate discrepancies between expected (or predicted) reward associated with a particular decision and the actual outcome of that decision. When an error occurs (i.e., predicted reward does not match the actual outcome), expectancies about possible outcomes associated with a decision can be updated to increase the likelihood of selecting a more optimal (i.e., reward-yielding) response in the future. As expectancies about the outcomes of particular decisions become more accurate, subjects are less swayed by the occasional violation of these reward expectancies and learn to optimize their selection of the most advantageous response to a stimulus. Behavioral findings typically reported for the probabilistic reward-based decision-learning task adopted here (e.g., Haruno and Kawato, 2006a; van Wouwe et al., 2012) were successfully reproduced in the current study. Participants' learning improved from the beginning to the end of the task: the formation of predictive stimulus-action-reward associations increased over time while prediction errors diminished.

Parkinson's disease patients were shown affectively neutral or positive film clips before participating in the learning task. We predicted that induced positive affect would improve the formation of stimulus-action-reward associations (as reflected in higher SADRP values), especially toward the end of the task, with less pronounced effects on outcome evaluation (expressed in RPE). Evidence that positive affect was induced was provided by pre- and post-test Likert scales in which participants in the positive affect condition confirmed that they felt more positive and amused after compared to before viewing the positive movie clip, whereas participants in the neutral condition reported no change in affect after the clip. Indeed, learning (as measured by earned rewards) benefited from induced positive affect. Consistent with our predictions, SADRP at the late stages of learning was larger in the positive compared to neutral affect group. Positive affect did not influence RPE values, not even when zooming in on the initial learning phase.

These findings present a striking parallel with recent findings on the effects of DA medication on reward-based learning in the same task. Using a within-subjects design, van Wouwe et al. (2012) observed that PD patients who were on compared to off their regular DA medication showed higher SADRP values, especially toward the later stages of learning, while RPEs remained unaltered, even in early stages of learning. Although by

no means conclusive, the present data are suggestive of the notion that induced positive affect incurs an increase in tonic DA which then modulates learning in much the same way as DA medication does.

Neuroimaging and computational studies (Haruno and Kawato, 2006a,b) have suggested that SADRP values are linked to activity in the anterior putamen (and its associated sensorimotor circuitry involved in action selection), whereas RPE values are linked to activity in the caudate and ventral striatum (and their associated circuitry involved in hypothesis generation and value updating). The present findings therefore provide consistent, albeit indirectly, with the idea that induced positive affect may benefit the action-oriented learning functions of the severely dopamine-depleted putamen in PD patients, while leaving the processing of RPEs in the less affected caudate and ventral striatum unaltered. During initial stages of mild PD, the disease is characterized by DA depletions in the striatum that produce motor deficits, involving the motor loop (including putamen and supplementary motor areas). During later stages of more progressed PD, these effects extend to the dorsolateral loop (including the dorsolateral prefrontal cortex and the dorsolateral head of the caudate) and still later to the orbitofrontal loop (lateral orbitofrontal cortex, ventromedial head of caudate) and the anterior cingulate loop (involving the anterior cingulate cortex and the ventral striatum, in particular the nucleus accumbens). Based on these differential effects of PD progression on striatal subregions and associated circuitries (Kaasinen and Rinne, 2002), it can be argued that SADRP should indeed benefit more from DA medication and positive affect than RPE, as the putamen is usually more depleted from DA than caudate and ventral striatum early in the disease. However, since the present data do not speak directly to the issue of striatal subcomponents, future work should confirm these speculations.

RELATION TO OTHER STUDIES: REINFORCEMENT LEARNING IN PARKINSON'S DISEASE

Studies of PD patients are important from a clinical perspective, but also provide a complementary approach to investigate the role of the basal ganglia and DA function in reward-based learning. The primary treatment to reduce PD motor symptoms such as tremor, bradykinesia, and rigidity, aims to increase DA availability and activity, including, most prominently, medication functioning as a DA precursor (typically levodopa) or as a DA agonist (Hornykiewicz, 1974). Because regions of the striatum are differentially affected by the disease, DA medication differentially affects these structures and their related functions. Although DA pharmacotherapy successfully improves motor deficits in PD, its effects on cognitive processes are more ambivalent. For example, DA medication can have positive and negative consequences on cognitive performance among PD patients (Cools, 2006). Specific cognitive functions that rely on the heavily DA-depleted dorsolateral and motor loops improve with DA pharmacotherapy, whereas other aspects of cognition that depend on ventral circuitries of the basal ganglia and remain relatively spared in early PD are impaired by DA medication (Gotham et al., 1988; Swainson et al., 2000; Cools et al., 2001; Czernecki et al., 2002).

However, not all aspects of reward-based decision-learning are compromised by DA medication. For example, Shohamy et al. (2005) found that feedback-based learning improved when PD patients were ON DA medication compared to when they were OFF medication. Frank et al. (2004) showed that this benefit obtained specifically for learning that certain actions are likely to yield reward, whereas learning that certain other actions are likely to yield punishment was negatively affected by DA medication. This pattern of levodopa-induced improved incentive learning but impaired avoidance learning, replicated by Bodi et al. (2009), is taken to reflect strengthened disinhibition along the direct route and weakened inhibition along the indirect route within the basal ganglia.

Although studies converge on the notion that striatal regions play a key role in reward-based decision-learning (Knutson et al., 2001; McClure et al., 2003; Frank et al., 2004; O'Doherty et al., 2004; Tricomi et al., 2004; Seger and Cincotta, 2005; Haruno and Kawato, 2006a,b; Bodi et al., 2009; Cools et al., 2009), the modulatory role of DA in different structures within the striatum is not yet well established. DA might have dissociable effects on different component processes of reward-based decision-learning (*vis.* outcome evaluation, supported by caudate and ventral striatum, versus reward prediction, supported by anterior putamen).

While reinforcement learning appears to depend on phasic DA dynamics, the modulation of dopamine levels by medication (L-dopa) or, allegedly, by positive affect, is of more tonic nature, begging the question why and how tonic alterations of DA levels should influence reinforcement learning. First, there is evidence that administration of L-dopa yields an increase in presynaptic dopamine synthesis increases (Tedroff et al., 1996; Pavese et al., 2006) and in phasic (spike-dependent) DA bursts (Keller et al., 1988; Harden and Grace, 1995). Second, in a probabilistic reinforcement learning paradigm, Parkinson's patients learned better from positive feedback when they were ON compared to OFF their dopaminergic medication (Frank et al., 2004). It should be noted that in the latter study, learning from NEGATIVE feedback was impaired when PD patients learned were ON compared to OFF their dopaminergic medication; presumably, the continuous medication-induced stimulation of D2 receptors effectively precludes the detection of phasic dips in DA firing (Frank, 2005). Thus, Frank's patient and modeling work showed that PD patients OFF medication more effectively process negative feedback in comparison to positive feedback whereas PD patients ON medication show the opposite pattern. In the current task though, a RPE results from either unexpected positive or negative feedback, thus a preference for positive or negative feedback cannot be distinguished based on SADRP and RPE values.

RELATION TO OTHER STUDIES: POSITIVE AFFECT

Induced positive affect has been shown to yield improved performance in a variety of tasks that rely on frontostriatal dopaminergic interactions (e.g., Dreisbach and Goschke, 2004; Dreisbach, 2006; Van der Stigchel et al., 2011; van Wouwe et al., 2011a; for a recent review see Chiew and Braver, 2011). Findings that patients with PD show performance impairments in these tasks (e.g., Kitagawa et al., 1994) lend some suggestive credit to the notion that performance improvements after positive affect might result from changes in dopaminergic levels in the brain. Circumstantial evidence in support of this notion derives from similarities between

the effects of induced positive affect and those of genetic variations in DA polymorphisms as well as individual differences in spontaneous eye-blink rate. Compared to individuals with low blink rates, greater cognitive flexibility was observed in individuals with high blink rates (allegedly associated with high tonic DA levels), especially if they were carriers of the DRD4 exon III 4/7 genotype (associated with high levels of prefrontal DA; Dreisbach et al., 2005).

In a recent study (van Wouwe et al., 2011a), we provided indirect evidence for a modulatory effect of induced positive affect on the dynamics of subcortical dopamine. In accordance with the logic explicated in Frank's model (outlined above), if positive affect serves to increase striatal DA levels, then the increased availability of DA molecules in the synaptic cleft should limit the effects of phasic dips as triggered by negative feedback and errors. As a result, a less pronounced dopaminergic error signal should be carried to the dorsal medial frontal cortex, which should in turn give rise to a less pronounced error-related negativity as measured using scalp-EEG immediately after a performance error. Consistent with Ashby's notion that induced positive affect produced a transient boost in DA, van Wouwe et al. (2011a) observed reduced amplitudes for the error-related negativity after watching comedy clips compared to neutral film fragments.

The current study contributed insights beyond those reported above by focusing on component processes of reward-based decision-learning that rely on different striatal circuits, and by examining the effects of induced positive affect on model parameters representing these component processes. The evidence reported here likens the effects of induced positive effect directly to the effects of DA agonists in PD patients. Our results allow us to articulate with greater precision the effect of positive affect on the caudate and ventral striatum on the one hand and on the putamen on the other. While positive affect leaves outcome evaluation processes (supported by caudate and ventral striatum) unaffected, learning to predict which actions yield reward (supported by the anterior putamen and associated motor circuitry) is improved, at least transiently, after viewing movie clips containing positive and amusing content, such as Charlie Chaplin slapstick.

The present observations touch also on recent findings on the effect of motivational incentives on the efficiency of cognitive performance in patients with PD (Harsay et al., 2010). DA neurons in the striatal reward system respond with a phasic increase in firing to stimuli that cue the prospect of upcoming reward (Schultz et al., 1992; Kawagoe et al., 2004). Evidence from non-human primates suggests direct striatal dopaminergic modulation of reward-dependent improvements of performance (Nakamura and Hikosaka, 2006). Neural decrements in reward-processing among patients with PD presumably reflect degeneration of dopaminergic neurotransmission (Backman et al., 2006; Kaasinen et al., 2000), and may be remedied by increasing reward (Goerendt et al., 2004). Due perhaps to deterioration in dopaminergic striatal circuitry, antisaccade performance is subject to decline in individuals with PD; the prospect of future reward, however, provides a motivational incentive for optimizing oculomotor preparation (Harsay et al., 2010).

LIMITATIONS

Some limitations apply to the experimental paradigm adopted here. First, in our version of the reward-based learning task, the patients always received the 90:10 block before the 80:20 block. The finding that probability did not influence any of the effects of interest may therefore have been confounded by an order effect (e.g., the increased difficulty associated with lower probabilities might have been countered by increases in learning efficiency due to practice). We used a fixed easy-to-more-difficult order to ascertain successful learning and comfortable participation in all of our patients. We readily acknowledge that, had we not used such a fixed order, we might have been able to show that, for instance, positive affect benefits learning in difficult situations more than in easier conditions.

The between-subjects design used in the present study has some obvious disadvantages. However, as confirmed in an informal pilot, when we combined the two affect conditions into one within-subjects design, the experiment lasted too long, such that (1) some patients experienced substantial fatigue during the second subsession, and (2) the wear-out of dopamine medication started to play a role (with either decreased performance toward the end of the session, or the need to take medication during the second half of the session). Hence, we decided to opt for a between-subjects design; despite the increase in variance, the effects of induced effect turned out to be robust enough to counter this disadvantage. Yet, future studies should aim at replicating such findings in a within-subjects setting.

The addition of age-matched control groups might have supplemented the conclusions of this study in interesting ways. In particular, such an addition could confirm that the patients were more impaired than healthy controls in reinforcement learning (as documented in the literature), and could specify how much positive affect ameliorated deficits relative to performance in healthy controls. Yet, the key rationale of studying PD patients was that the nature of their specific impairment is such that, if the dopamine hypothesis were correct, their deficient reinforcement learning performance should benefit from induced positive affect. Such a finding (as we obtained) is in and of itself important and informative: DA-related deficits in PD can be remedied (at least transiently) by such a simple (and pleasant) measure as induced positive affect. The importance of that finding should in itself not depend on comparing this effect to age-matched controls, even though we recognize the potential supplemental value of such a comparison, and recommend such comparisons for future studies.

Stimulus-action-dependent reward prediction and RPE have been linked to the role of DA bursts at different time points and in different stages of learning. These distinctions notwithstanding, SADRP and RPE are not entirely independent at the behavioral level. By and large, increases in SADRP values are associated with decreases in RPE values. Thus, while our findings suggest that induced positive affect impacts putamen-based processes more than processes supported by caudate and ventral striatum, and that induced positive affect impacts late stages of learning more than early stages, these findings do not entirely exclude the possibility that the caudate and ventral striatum are modulated by positive affect altogether.

Finally, we did not obtain clinical measures of disease severity or progression, such as UPDRS scores or Hoehn–Yahr assessments, nor did we pursue formal diagnostic interviews by specialists. Thus, we cannot exclude that our positive and neutral affect groups differed in terms of relevant clinical variables. Yet, since the groups did not differ in years since disease onset, medication dosage, or other background variables, and since our samples included only patients who could ambulate independently (rendering it unlikely that they met criteria for Hoehn–Yahr stages IV or V), and since the sample sizes in our groups were rather considerable in comparison to typical studies in the field (for evidence on individual differences among PD patients revealed only in larger samples, see Wylie et al., 2009), we are confident that the results reported here are representative and robust. The finding that the SADRP values, previously associated with putamen function, benefit more from induced positive affect than the RPE values, previously associated with the function of the caudate and accumbens nuclei, appears to underline the suggestion that our patient groups were on average in relatively early stages of their disease, affecting the putamen more than other striatal areas. We acknowledge the lack of UPDRS scores or Hoehn–Yahr assessments limits the conclusiveness of our inferences (even though such measures do not provide direct measures of putamen versus caudate/accumbens involvement; in fact, identical UPDRS scores may actually reflect very different underlying patterns of striatal circuit dysfunction). However, we view the potential of our approach to differentiate these possible patterns (by incorporating the *Q*-model and the documented correspondence of the *Q*-learning model's key parameters to striatal substructures) as a major strength of our study approach. This approach allows us to articulate with greater precision which aspects of the striatal circuitry benefits from induced positive affect.

CONCLUSION

In conclusion, induced positive affect modulates computational measures of probabilistic reward-based decision-learning in patients diagnosed with PD. Previous work has shown these measures to rely on the nucleus caudatus and nucleus accumbens (outcome evaluation during the early phases of learning) and the anterior putamen (reward prediction during later phases of learning). We observed that positive affect facilitated learning, through its effects on reward prediction rather than outcome evaluation; these effects show a striking similarity to the effects of dopaminergic medication. Among PD patients who were on their regular medication regime, watching a few minutes of comedy clips apparently served to remedy dopamine-related problems associated with frontostriatal circuitry and, consequently, in learning to predict which actions will yield reward.

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Cognitive and affective control in insomnia

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Insomnia is a prevalent disabling chronic disorder. The aim of this paper is fourfold: (a) to review evidence suggesting that dysfunctional forms of cognitive control, such as thought suppression, worry, rumination, and imagery control, are associated with sleep disturbance; (b) to review a new budding field of scientific investigation – the role of dysfunctional affect control in sleep disturbance, such as problems with down-regulating negative and positive affective states; (c) to review evidence that sleep disturbance can impair next-day affect control; and (d) to outline, on the basis of the reviewed evidence, how the repetitive-thought literature and the affective science literature can be combined to further understanding of, and intervention for, insomnia.

Keywords: affect, emotion regulation, insomnia, repetitive thought, rumination, thought control, thought suppression, worry

INTRODUCTION

Insomnia is a prevalent disorder, with between 4 and 22% of people reporting chronic insomnia (Ancoli-Israel and Roth, 1999; Chevalier et al., 1999; Roth et al., 2011). The consequences of insomnia include complaints of impaired concentration and memory, elevated risk of accidents, more frequent use of medical services, and augmented work absenteeism (Ohayon et al., 1997; Roth and Ancoli-Israel, 1999). Furthermore, there is evidence that insomnia significantly heightens the risk of subsequently developing another psychiatric disorder, particularly an anxiety disorder, depression, or a substance-related disorder (Taylor et al., 2005; Johnson et al., 2006; Brower and Perron, 2010). Not surprisingly then, insomnia is regarded as a serious public health problem, with the direct and indirect costs associated with it in the United States estimated at between US$30 and 35 billion annually (Chilcott and Shapiro, 1996).

Over the last decades, behavioral and cognitive approaches have led to significant advances in the etiology and treatment of this disorder (e.g., Harvey et al., 2005; Bootzin and Epstein, 2011). Cognitively inspired accounts of insomnia assume that mental hyperarousal (e.g., Harvey, 2002) or problems with mental dearousal (e.g., Espie, 2002) play a key role in acute and chronic forms of this disorder. In support of such a role, it has been found that poor sleepers are cognitively more aroused at bedtime than normal sleepers are (e.g., Robertson et al., 2007) and that poor sleepers perceive their "racing mind" as causally related to their sleep problems (e.g., Lichstein and Rosenthal, 1980). Cognitive accounts of insomnia also generally assume that inadequate emotional processing during the day will result in a surge of affect-laden concerns during the presleep period, thereby fueling excessive negatively toned cognitive activity (e.g., Espie, 2002; Harvey, 2002). However, as will be reviewed in more detail below, empirical research into affective aspects of sleep-interfering arousal is still in its early stages.

A growing line of research suggests that poor sleepers typically have difficulties relinquishing control when trying to fall asleep (Espie et al., 2006). Specifically, in the face of unwanted mental activity at bedtime, which can take the form of verbal thought or visual imagery (Harvey, 2000), poor sleepers tend to rely on dysfunctional control strategies that maintain cognitive and affective arousal instead of helping them to wind down (Harvey, 2002). In this article, we will first review evidence suggesting that dysfunctional forms of cognitive control, such as thought suppression, worry, rumination, and imagery control, are associated with sleep disturbance. We will then review more recent research suggesting that dysfunctional affect control, such as problems with down-regulating negative and positive affective states, are also related to sleep disturbance. Moreover, we will review research indicating that sleep disturbance, in turn, can impair next-day affect control. Finally, we will outline how theoretical principles from the repetitive-thought literature could advance the study of cognitive control in insomnia, while principles and tools from the affective science literature could foster the study of affective control in insomnia. As will emerge from our analysis, a better understanding of the intricate interplay between cognitive and affective control could lead to the development of new intervention techniques that facilitate the transition to sleep.

EXPLORATIONS OF THE ROLE OF COGNITIVE CONTROL IN INSOMNIA

EXPERIMENTAL STUDIES ON THE ROLE OF THOUGHT SUPPRESSION IN INSOMNIA

Given that poor sleepers typically perceive their racing mind as causally related to their problems with sleep (e.g., Lichstein and Rosenthal, 1980), it seems inevitable that these individuals will do something in an attempt to prevent, modify, or suppress the thoughts that they perceive to be interfering with their sleep

(Harvey, 2002). In this section, we will review evidence suggesting that ironic effects of thought suppression (e.g., Wegner, 1989; Najmi and Wegner, 2008) may be involved in the etiology and persistence of unwanted intrusive thoughts in insomnia.

Wegner (1994) accounted for the counterproductive effects of thought suppression by suggesting that the level of mental control enjoyed by an individual at any one time is a function of the joint action of a monitoring and an operating process. Termed the ironic process theory, it maintains that attempts to suppress involve (a) a controlled operating process that directs attention toward a distracter, that is, a thought other than the unwanted one; and (b) an automatic monitoring process that searches for failures to achieve the desired state and that, if necessary, renews the first process. In many circumstances, these interrelated processes work in tandem to successfully achieve suppression. However, there are a number of situations in which the balance is undermined, resulting in the unwanted thought intruding into consciousness. The "rebound" of the suppressed thought is explained by the fact that if the controlled distracter search is voluntarily relinquished or disabled by other resource-demanding tasks, the automatic and therefore less resource-dependent monitoring lingers on, thereby enhancing the accessibility of the target thought (e.g., Najmi and Wegner, 2008). A series of studies highlight the importance of this theory to sleep and insomnia.

Harvey (2003) instructed individuals with insomnia and good sleepers either to suppress or to not suppress one issue/problem/thought while trying to get to sleep. On the night of the study, the participants who were told to suppress reported that they took longer to fall asleep and rated their sleep quality as poorer compared with participants given non-suppression instructions. This finding suggests that attempting to suppress a thought adversely affects both (a) self-reported sleep-onset latency and (b) sleep quality. The effect was detected for both the good sleepers and the patients with insomnia. Contrary to previous work (Wegner, 1989), there was no paradoxical increase in the frequency of the suppressed thought. However, it should be noted that the expected rebound of the suppressed thought might have occurred after sleep onset, which would have precluded it from emerging in the participants' reports about the presleep period. To test this hypothesis, Schmidt and Gendolla (2008) used an experimental design involving forced awakenings in a sleep laboratory. Half of the participants were instructed to suppress a target thought, whereas the other half freely thought of anything at all. Results revealed a reversal of target thought frequency at sleep onset: participants instructed to suppress reported fewer target thoughts than did controls before falling asleep, but more target thoughts afterward. In a related vein, three studies have found a suppression-induced increase of a target thought in dream mentation, especially if the target thought is laden with affect and if suppression is attempted under cognitive load (Wegner et al., 2004; Taylor and Bryant, 2007; Bryant et al., 2011). Although these studies on a "dream rebound" of suppressed thoughts did not assess sleep disturbance, they describe the mechanisms through which suppression of affect-laden content may entail a potentially sleep-interrupting rebound of the same content.

EXPERIMENTAL STUDIES ON THE ROLE OF WORRY IN INSOMNIA

Another strategy that is suggested to be commonly used by poor sleepers to manage unwanted thoughts at bedtime is worry (Borkovec, 1982; Watts et al., 1994). Research accumulated over several decades has shown that experimental manipulations designed to increase worry in good sleepers lengthen sleep-onset latency (Gross and Borkovec, 1982; Hall et al., 1996), whereas experimental manipulations designed to decrease worry in insomnia patients shorten sleep-onset latency (Haynes et al., 1981; Levey et al., 1991). An innovative set of investigations has been published more recently that confirms and extends these findings.

In one study, the approach to presleep worries was experimentally manipulated (Carney and Waters, 2006). Individuals with insomnia were asked either (a) to produce solutions to worries ("constructive worry") in the early evening, or (b) to list their worries and fill out worry questionnaires. The constructive worry group reported less cognitive arousal prior to bedtime and spent less time awake overall. These findings suggest that structured problem solving in the evening may reduce presleep cognitive arousal, thereby facilitating the transition to sleep.

In another recent study designed to test the relationship between sleeplessness and worry, undergraduate students grouped into high or low worriers on the basis of their responses on the Penn State Worry Questionnaire were given either 300 mg caffeine (to induce sleeplessness) or a placebo prior to sleep (Omvik et al., 2007). Results indicated that high worriers did *not* report increased worry thoughts compared with low worriers in the face of sleeplessness. However, total sleep time as measured by actigraphy was reduced relatively more in high worriers than in low worriers as a consequence of caffeine-induced sleeplessness. This result might suggest that worries are comparatively more negative or intense and therefore more sleep-interfering in high worriers.

Recent research has also begun to explore the relationship between worry and rumination, which still needs to be clarified. Rumination has been defined as a "mode of responding to distress that involves repetitively and passively focusing on symptoms of distress and on the possible causes and consequences of these symptoms" (Nolen-Hoeksema et al., 2008). Worry and rumination are generally distinguished on the basis of their temporal orientation: worry refers to distress regarding future events, whereas rumination concerns thoughts of past events and current symptoms (e.g., Kaplan et al., 2009). In the first experimental investigation into the role of rumination for sleep, Guastella and Moulds (2007) examined the relations between rumination and sleep quality following a stressful midsemester exam in an undergraduate sample. Participants were asked either to ruminate about the exam ("think about how you felt when you were taking the test today") or to distract ("think about clouds forming in the sky") before sleep. The following morning, participants completed ratings of presleep intrusions about the exam and of sleep quality. Results indicated that although the rumination instruction led to more exam intrusions during the presleep period in participants with high and low trait rumination scores, only individuals with a trait tendency to ruminate reported reduced sleep quality following the rumination instruction. According to the authors, this finding suggests that soon after the rumination task was completed, low ruminators returned to their "default" cognitive

style – that is, non-ruminative thought. In conjunction with correlational evidence that will be reviewed later (Carney et al., 2006, 2010), these results suggest that besides worry, rumination constitutes a form of cognitive control that may contribute to sleep disturbance.

CORRELATIONAL STUDIES ON THE ROLE OF THOUGHT-CONTROL STRATEGIES IN INSOMNIA

Apart from suppression, worry, and rumination, people may use a range of other thought-control strategies in an attempt to manage unwanted thoughts when trying to fall asleep. With the aim of exploring such strategies, Harvey (2001) adapted the Thought-Control Questionnaire (TCQ), designed by Wells and Davies (1994), for use with patients with insomnia. This questionnaire, the TCQ-Insomnia (TCQ-I), was administered to 30 individuals with insomnia and to 30 good sleepers (Harvey, 2001). The results indicated that thought suppression, reappraisal and worry were used significantly more by participants with insomnia than by good sleepers. The use of suppression and worry is consistent with the experimental evidence discussed earlier. Regarding reappraisal, it may be an effective daytime strategy ensuring effective resolution of hassles and concerns, but it makes intuitive sense that engaging in similar processes at night might interfere with sleep onset.

In a follow-up study, a refined version of the TCQ-I was elaborated, which was termed the Thought-Control Questionnaire Insomnia-Revised (TCQI-R; Ree et al., 2005). The TCQI-R comprises six subscales: (1) aggressive suppression (e.g., "I get angry at myself for having the thought"); (2) cognitive distraction/suppression (e.g., "I think pleasant thoughts instead"); (3) behavioral distraction/suppression (e.g., "I try to block them out by reading, watching TV, or listening to the radio"); (4) social avoidance (e.g., "I avoid discussing the thought"); (5) worry (e.g., "I worry about more minor things"); and (6) reappraisal (e.g., "I try to reinterpret the thought"). A comparison between good sleepers and individuals with insomnia revealed that the latter more frequently used every thought-control strategy except for cognitive distraction. In accordance with the experimental evidence reviewed earlier, the strategies of aggressive suppression and worry proved particularly unhelpful, with the use of these techniques predicting poorer sleep quality in a sample that included good sleepers, subthreshold insomniacs, and insomniacs. In contrast, the use of cognitive distraction predicted better sleep quality. The validation study of the French version of the TCQI-R essentially replicated these findings (Schmidt et al., 2009): all six thought-control strategies were significantly related to facets of insomnia, with worry and aggressive suppression being most strongly related to insomnia symptoms and cognitive distraction functioning as a potential buffer against the latter.

In a recent extension of this line of research, a study with a large sample of young adults has revealed how certain personality traits may predispose people to rely on dysfunctional strategies of mental control when confronted with thoughts that keep them awake at night (Schmidt et al., 2010). Specifically, a structural equation model analysis indicated that individuals scoring high on two facets of impulsivity, namely, urgency and lack of perseverance, are particularly prone to respond with aggressive suppression and worry to unwanted mental activity at night. Impulsive urgency can be defined as the tendency to act rashly, especially under conditions of negative affect (Whiteside and Lynam, 2001). This facet has been shown to reflect a relative inability to deliberately inhibit dominant, automatic, or prepotent responses (Gay et al., 2008). As for lack of perseverance, it refers to an individual's inability to remain focused on a task, especially if the latter is boring or difficult (Whiteside and Lynam, 2001). This facet is linked to a relative inability to inhibit recurrent and irrelevant thoughts or memories (Gay et al., 2008).

The pathways through which the two mentioned facets of impulsivity may lead to an increase of unwanted cognitive activity at bedtime and thereby to a more intense use of dysfunctional control are twofold. Impulsive behavior may lead to a buildup of cognitive and affective arousal in the course of the day, which will then interfere with sleep and evoke more intense efforts at mental control. Additionally, or alternatively, problems with cognitive inhibition, as involved in lack of perseverance, may render individuals particularly vulnerable to unwanted intrusive thoughts at bedtime, and problems with behavioral inhibition, as involved in urgency, may prompt individuals to immediately do something about the unwanted thoughts, be it by way of mental or real-life responses. Below, we will review evidence for the first pathway, which suggests that impulsive urgency is indeed related to a surge of negative thoughts and emotions at bedtime (e.g., feelings of regret) that hinder the process of falling asleep (e.g., Schmidt and Van der Linden, 2009). The question of whether urgency and lack of perseverance also act on sleep-interfering mental activity through the second pathway remains to be explored.

Another important question awaiting further research is that of how dysfunctional thought-control may contribute to the transition from acute to chronic insomnia. In a pioneering study in this field, Jansson and Linton (2006) examined the associations between worry and poor sleep in relatively new (poor sleep 3–7 months) and chronic (poor sleep 8–12 months) insomnia sufferers. The main finding was that worry about sleeplessness was associated with poorer perceived sleep in individuals with chronic, but not recent, insomnia. The authors interpreted this finding as suggesting that the impact of sleep-related worry becomes increasingly stronger over time, which supports the idea that worry contributes to the maintenance of sleep problems and the development of chronic insomnia.

STUDIES ON THE ROLE OF IMAGERY CONTROL IN INSOMNIA

In parallel to the studies on thought control in insomnia, a literature has emerged that focuses on the control of imagery, that is, the ability to manage the occurrence of mental images. As described in the introduction, unwanted intrusive thoughts at bedtime can take the form of verbal thought or visual imagery. Borkovec et al. (1998) proposed that thinking about an emotional topic in verbal mode results in a drop in physiological response, which hinders in-depth processing and resolution of the emotional topic. Conversely, the translation of a concern into an image is suggested to increase physiological response in the short term, but will ultimately facilitate successful processing and the resolution of the emotion (Borkovec et al., 1998; Sibrava and Borkovec, 2006). Application of this theorizing to insomnia leads to the

hypothesis that because presleep worry includes "the presence of active, picture-like images" (Coates et al., 1982), perhaps patients with insomnia spontaneously disengage from images to think about the same topic in verbal thought, thereby preventing emotional processing and contributing to the fueling of intrusive and worrisome thought (Harvey, 2002). Two published investigations have tested this proposal.

First, in an investigation of the imagery experienced during the presleep period in the natural home environment, 20 patients with insomnia and 20 good sleepers recorded when an image came to their mind by pressing a handheld counter (Nelson and Harvey, 2003). They then provided an oral description of the image and indicated whether the image was "pleasant," "unpleasant," or "neutral." This information was recorded on a voice-activated tape recorder. Analyses revealed that the insomnia group experienced more negatively valenced presleep images, but a lower number of images overall, relative to the good sleeper group. As negative imagery is likely to be associated with physiological and affective activation (Vrana et al., 1986), perhaps this activation motivates the quick and reflexive termination of images (hence the lower number of images overall reported by the insomnia group) in order to switch thought to the verbal mode.

Taking this line of investigation a step further, in an experimental manipulation of imagery control, individuals with insomnia were exposed to a stressor (speech threat) just prior to getting into bed and were instructed to think about the speech and its implications in either images (Image Group, $N = 14$) or verbal thought (Verbal Group, $N = 17$; Nelson and Harvey, 2002). Results indicated that in the short term, the Image Group reported more distress and arousal relative to the Verbal Group. In the longer term, the Image Group estimated that they fell asleep more quickly and, the following morning, reported less anxiety and more comfort about giving the speech compared with the Verbal Group. These findings are consistent with Borkovec et al.'s (1998) suggestion that the translation of negative-affect-laden concerns into imagery will ultimately facilitate emotional processing.

However, imagery may also be used in ways that divert attention from the processing of negative-affect-laden concerns and may in this way reduce unwanted presleep cognitive activity. In an experiment designed to test these assumptions (Harvey and Payne, 2002), individuals with insomnia were given one of three instructional sets to follow prior to sleep: instructions to distract using imagery, general instructions to distract, or no instructions. In the imagery distraction condition, participants were asked to distract themselves "by imagining a situation they found interesting and engaging, but also pleasant and relaxing." In support of the earlier mentioned assumptions, the imagery distraction group (a) reported shorter sleep-onset latency compared with the no instruction group and (b) rated their presleep thoughts, worries, and concerns as less uncomfortable and distressing than did the two other groups. Regarding the durability of this effect, the authors of the study note that although imagery distraction may be an effective short-term method to manage unwanted thoughts at bedtime, it may be less effective in the longer term because any "interesting and engaging" content will lose some of its attraction over time and will have to be replaced by new content.

In sum, the available evidence on imagery control and insomnia suggests that individuals with insomnia (a) may avoid negatively valenced imagery, which will hinder emotional processing and may thereby fuel negative cognitive activity at bedtime; and (b) may engage in positively valenced imagery to buffer negatively toned cognitive activity at bedtime, which may facilitate sleep in the short, but not long, term, unless the content of positive imagery is regularly renewed.

AFFECTIVE TURN IN INSOMNIA RESEARCH

As reviewed in the introduction, cognitive approaches to insomnia have led to the view that excessive mental activity at bedtime represents a central impediment to the process of falling asleep (e.g., Harvey, 2002). Although sleep-interfering mental activity is likely embedded in affective states, the latter have long received very little scientific attention. Recently, however, the importance of investigating affective processes in insomnia has been recognized (Harvey et al., 2009; Walker and Harvey, 2010) and a corresponding research agenda has been formulated (Baglioni et al., 2010a).

STUDIES ON THE ROLE OF AFFECTIVE EXPERIENCE AND CONTROL IN INSOMNIA

Initial empirical evidence for the role of emotion regulation came from two studies that investigated stress coping in good sleepers and insomniacs (Morin et al., 2003; LeBlanc et al., 2007). In both studies, stress coping skills were assessed with an inventory that distinguishes between three strategies, namely, task-oriented coping (e.g., coming up with several different solutions for a problem), emotion-oriented coping (e.g., blaming oneself for not knowing what to do), and avoidance-oriented coping (e.g., watching TV in order to distract oneself). Critically, it was found that patients with insomnia relied more often on emotion-oriented coping than good sleepers did, and that emotional coping increased perceived stress impact and presleep cognitive arousal, thereby contributing to sleep disturbance.

A number of other pioneering studies have in recent years investigated how specific affective states can impact sleep and how different control strategies may modify these affective states and their impact on sleep. In an extension of a line of research suggesting a negative impact of trait anger on sleep (Brissette and Cohen, 2002; Shin et al., 2005; Granö et al., 2008), Stoia-Caraballo et al. (2008) examined the relations between forgiveness of interpersonal transgressions, anger rumination, and sleep quality in a sample of undergraduates by means of questionnaires. Using structural equation modeling, the authors found that forgiveness attenuated anger rumination and negative affect, thereby enhancing sleep quality.

Just as forgiving others seems to benefit sleep, forgiving oneself for one's own "wrongdoings" may also improve sleep, as a line of research on counterfactual emotions and sleep disturbance suggests. Such emotions include regret, shame, and guilt, which are termed counterfactual because they involve comparing the facts of one's own behavior with counterfactual imaginations of what one might or should have done (e.g., "If I had [not] done X, I would be in a better situation now"; e.g., Zeelenberg and Pieters, 2007). Initial hints at a negative impact of counterfactual emotions on

sleep came from a questionnaire study by Wrosch et al. (2005), who found that regret intensity was associated with an omnibus measure of health problems, including chronic sleep problems, in a sample of older adults.

More recently, a series of studies have tested the idea that the time window prior to sleep might be particularly suitable for the emergence of counterfactual thoughts and emotions because bedtime is often the first quiet period available to review the day's events and one's own behavior. The results of an exploratory questionnaire study with undergraduate students lent initial support to this idea: counterfactual thoughts and emotions were often experienced at bedtime and their frequency was linked to self-reported insomnia severity (Schmidt and Van der Linden, 2009). Moreover, it was found that rash-action-prone students (with high scores on impulsive urgency) were particularly likely to engage in counterfactual processing at bedtime.

The findings obtained in the undergraduate sample (Schmidt and Van der Linden, 2009) were replicated and extended in a questionnaire study with elderly people (Schmidt et al., in press). In this study, participants were explicitly asked to indicate when in the course of the day they most often experienced regrets. The pattern of their responses clearly supported the idea that the presleep period is particularly suitable for the emergence of counterfactual emotions: while regret frequency remained at relatively low levels for most of the waking hours, a sharp rise occurred in the evening after going to bed. Moreover, analyses revealed that the nocturnal rise of regrets substantially contributed to insomnia severity independently of other well-known risk factors for late-life sleep disturbance, such as depression, sleep-interfering medical conditions, or medications. Finally, as with young adults, rash-action-prone older adults were particularly likely to experience regrets at bedtime and were therefore at a higher risk for sleep disturbances.

In the study with older adults (Schmidt et al., in press), participants were also asked to rate how often they relied on different thought-control strategies when trying to cope with their regrets at bedtime. It was found that the thought-control strategies of self-attacking (e.g., "I blame myself for having the regret"), thought suppression (e.g., "I try to stop thinking about the regret"), and worry (e.g., "Instead of the regret, I worry about more minor things") were positively associated with the frequency of nocturnal regrets and insomnia severity. These findings suggest that cognitive control may be exerted with the aim to achieve affective control, even if, as in the present study, the cognitive strategies turn out to be counterproductive.

Further evidence for sleep-interfering effects of counterfactual emotions has recently come from an experimental study by Schmidt and Van der Linden (in preparation). These authors asked a sample of undergraduate students to complete two questionnaires at home, one in the evening before going to bed, the other one in the morning after getting up. Participants were randomly assigned to one of three groups: prior to sleep, they were asked to describe the behavior they were most proud of, their most intense regret, or a standard working day (control condition). In the morning, they rated different dimensions of their sleep. In accordance with the earlier mentioned findings on counterfactual emotions and sleep (Schmidt and Van der Linden, 2009; Schmidt et al., in press), activation of preexisting regrets almost doubled the time taken to fall asleep in participants with high levels of habitual regrets when compared with the control condition.

With regard to the control of counterfactual emotions prior to sleep, the finding that self-attacking was closely associated with the frequency of nocturnal regrets and insomnia severity in the study by Schmidt et al. (in press) suggests that fostering *self-forgiveness* might also be beneficial for sleep, given that *forgiving others* for their wrongdoings has been shown to be beneficial for sleep quality (Stoia-Caraballo et al., 2008). Outside of the area of insomnia research, converging lines of evidence indicate that training in self-compassion or self-forgiveness may help down-regulate negative affective states that excessive self-criticism evokes (e.g., Ingersoll-Dayton and Krause, 2005; Gilbert and Procter, 2006).

An investigation by Wrosch et al. (2007, Study 2) provided preliminary evidence that regrets are amenable to intervention and that sleeping difficulties can in this way be reduced. In this study, participants were asked to report their most severe life regret and to assess its intensity twice with an interval of 3 months. At the same two time points, participants also rated their overall sleep quality during the past month. In the interim, they completed a writing intervention on three consecutive days. Participants in the experimental group were induced to engage in three regret–regulation strategies that have previously been shown to potentially serve a protective function: external attribution, downward social comparison, and disengagement from undoing the regret through selection of meaningful future goals (e.g., Heckhausen et al., 2010). Participants in the control group were instructed to write in a neutral, unemotional way about daily events and activities. Consistent with predictions, the authors found a general decline in regret intensity in the experimental group. Although the writing intervention did not show a main effect on changes in sleep problems, there was a significant interaction with initial regret intensity: participants in the experimental group who experienced high levels of regret intensity at the beginning of the study experienced fewer sleep problems over time, whereas the reverse was true of the participants in the control group.

Besides down-regulation of negative affective states, researchers have also begun to explore up-regulation of positive affective states for its effects on sleep. For instance, Steptoe et al. (2008) found in a large sample of civil servants that both hedonic well-being (positive affect), as captured by ecological momentary assessment, and eudaimonic well-being (purposeful engagement with life) were inversely related to sleep problems after adjustment for age, gender, household income, and self-rated health. Of note, hedonic and eudaimonic well-being dampened adverse effects of a number of negative psychosocial factors on sleep, including financial strain, social isolation, low emotional support, negative social interactions, and psychological distress. However, as the authors of the study emphasize, the relations between sleep and affect are likely bidirectional and the cross-sectional design of their investigation did not allow them to draw causal inferences.

Two groups of researchers have investigated how trait and state gratitude may influence affect and sleep, complementary to these findings on well-being and sleep. Emmons and McCullough (2003, Study 3) asked a sample of people with neuromuscular disease to complete "daily experience rating forms" in the early evening for

3 weeks. In these forms, participants rated, among other variables, their daily affect, subjective well-being, and sleep. Unbeknownst to the participants, they were assigned to one of two experimental conditions. In the control condition, they simply completed the mentioned forms. In the gratitude condition, they were asked to count their blessings, that is, to write down up to five things that they were grateful or thankful for. Analyses revealed that the gratitude induction increased positive affect, reduced negative affect, and improved sleep quantity and quality. Pursuing this line of research, Wood et al. (2009) examined the relations between trait gratitude and sleep in a cross-sectional questionnaire study with a large community sample. The authors found that trait gratitude predicted greater subjective sleep quantity and quality, shorter sleep latency, and less daytime dysfunction. Moreover, the relation between gratitude and each of the sleep variables was mediated by more positive and fewer negative presleep cognitions.

Although these studies seem to clearly suggest that up-regulation of positive affect is beneficial for sleep, some recent findings caution against too simplistic a view that would equate positive affect with good sleep and negative affect with bad sleep. For instance, Talbot et al. (2009) examined the effects of mood induction on sleep in a group of individuals with interepisode bipolar disorder and in a group of healthy controls. Participants spent two baseline nights in the sleep laboratory, followed by a happy mood induction night and a sad mood induction night. Results indicated a significant interaction whereby on the happy mood induction night, the bipolar group exhibited significantly longer sleep-onset latency than did the control group, whereas there was no difference on the baseline nights. Moreover, the bipolar group exhibited a (non-significantly) longer sleep-onset latency on the happy mood induction night compared with the baseline night, whereas the control group exhibited significantly shorter sleep-onset latency on the happy mood induction night. On the sad mood induction night, participants in both groups had shorter sleep-onset latency. These findings suggest that problems with the regulation of positive affect may contribute to sleep-onset insomnia in bipolar individuals, possibly because these individuals reacted to the happy mood induction with an activating focus on the pursuit of goals and rewards (cf. Johnson, 2005).

STUDIES ON THE ROLE OF SLEEP FOR AFFECTIVE EXPERIENCE AND CONTROL

Up to this point, we have considered how dysfunctions in affective control may adversely impact sleep. In addition, accumulating evidence suggests that lack of sleep, in turn, may adversely impact next-day affect. Thus, the relations between sleep and affect are best conceptualized as bidirectional (Walker and Harvey, 2010), with the potential of negative effects escalating into a vicious circle. For instance, Buysse et al. (2007) used ecological momentary assessment to obtain ratings of daytime symptoms from insomnia patients. Results indicated that, when compared with good sleepers, individuals with insomnia endorsed higher negative mood and lower positive mood. More recently, Baglioni et al. (2010b) presented blocks of neutral, negative, positive, sleep-related negative, and sleep-related positive pictures to individuals with primary insomnia and good sleepers. During the presentation, facial electro-myography and -cardiography were performed. Analyses revealed that individuals with primary insomnia exhibited greater inhibition of the corrugator activity in response to sleep-related positive stimuli as compared with the other blocks of stimuli, suggesting a heightened motivation and sensitivity toward good sleep. At the same time, individuals with insomnia rated sleep-related negative stimuli as more unpleasant and arousing and showed higher cardiac vagal tone in response to all stimuli as compared with good sleepers, suggesting subjective hyperarousal in the context of poor sleep and a general physiological hyperarousal.

Furthermore, converging lines of clinical evidence suggest that sleeping difficulties adversely impact affective disorders (including anxiety, depression, and bipolar disorder) and problems with anger, aggression, and impulse control, especially among children and adolescents (e.g., Jenni and Dahl, 2008). For example, in patients with bipolar disorder, the most common prodrome of mania is sleep disturbance (Jackson et al., 2003). And with regard to sleep and aggression, Haynes et al. (2006), for instance, examined the effects of a 6-week behavioral sleep intervention in adolescents who had recently been treated for substance abuse. It was found that increases in sleep time were associated with decreases in aggressive ideation and aggressive actions occurring during conflicts. Taken together, the available clinical evidence thus clearly suggests that poor sleep impairs affective and behavioral control – and that these effects may be reversed through sleep-promoting interventions.

Additional evidence for effects of sleep on next-day affect comes from sleep-deprivation studies. However, these studies must be interpreted with the caveat that findings from sleep-deprived normal sleepers may not readily be extrapolated to chronic poor sleepers because, among other reasons, insomnia involves adaptation processes and is not necessarily associated with reduced sleep quantity. In a recent sleep-deprivation study, Talbot et al. (2010) investigated the impact of experimentally shortened sleep in adolescents and adults. The main findings were that sleep-deprivation reduced positive affect and increased anxiety during a "catastrophizing task," in which an interviewer repeatedly asked questions about each participant's most threatening worry (for details, see Vasey and Borkovec, 1992). Analogous results have been reported for work-related sleep deprivation. For example, Zohar et al. (2005) investigated the longitudinal relations between night-shift-related sleep loss and emotional reactivity in medical residents. Analyses of ecological momentary assessment and actigraphic sleep data indicated that sleep loss intensified negative emotions following goal-disruptive daytime events, whereas positive emotions were attenuated following goal-conducive daytime events. Increased reactivity to negative stimuli following sleep deprivation has also been found in an experimental study with healthy young adults (Franzen et al., 2009): when compared with a normal sleep control group, the sleep-deprived group displayed a larger pupil diameter when viewing negative pictures.

In the first study that used functional brain imaging to investigate the neural underpinnings of affective dysregulation following sleep loss (Yoo et al., 2007), sleep-deprived individuals showed a 60% increase of amygdala activation in response to increasingly negative stimuli from the International Affective Picture System, a threefold increase in the extent of amygdala volume that was activated, and a loss of functional connectivity between the amygdala

and the medial-prefrontal cortex. This brain region exerts top-down control on the limbic area (including the amygdala) and regulates emotional responses to render them appropriate for the context. The study thus suggests that sufficient sleep is indispensable for maintaining prefrontal control over affective reactions and behavior (see also Walker and van der Helm, 2009).

TOWARD AN INTEGRATIVE VIEW OF COGNITIVE AND AFFECTIVE CONTROL IN INSOMNIA

As argued throughout this article, the stream of consciousness in the transition to sleep may be decomposed into cognitive aspects, such as thoughts and images, and affective aspects, such moods and emotions. Recent accounts of insomnia have proposed that both cognitive and affective aspects of mental hyperactivity at bedtime may contribute to sleeping difficulties (e.g., Espie, 2002; Harvey, 2002). However, as may be gathered from the preceding review of the literature, research has as yet mainly focused on cognitive aspects of sleep-interfering mental activity and only just begun to empirically explore affective aspects. In what follows, we will describe how an integrative view of cognitive and affective control in the transition to sleep could advance our understanding of insomnia and lead to the development of new prevention and intervention techniques.

NEW INSIGHTS FROM THE REPETITIVE-THOUGHT LITERATURE

Regarding cognitive control in insomnia, converging lines of correlational and experimental evidence suggest that two forms of thought control that are often used by poor sleepers are particularly dysfunctional, namely, thought suppression and worry. However, a question calling for conceptual and empirical clarification is that of the relation between worry and rumination. Rumination has also been found to be associated with poor sleep (Carney et al., 2006; Guastella and Moulds, 2007), and one recent study has suggested that rumination may in important ways contribute to clinical insomnia independently of worry and depressed mood states (Carney et al., 2010). In a similar vein, an earlier questionnaire study with students had revealed that rumination was associated with subjective sleep quality even after controlling for negative mood (Thomsen et al., 2003). Of importance, these findings suggest that problems with cognitive control may contribute to sleep problems independently of negative mood states.

To clarify the relation between worry and rumination in the context of insomnia, some researchers have suggested conceptualizing them as two forms of repetitive thought (e.g., Carney et al., 2010). Repetitive thought has come to be seen as an important perpetuating factor that pervades a wide range of psychological disorders (e.g., Harvey et al., 2004). Outside of the area of insomnia research, several researchers have in recent times proposed comprehensive approaches to repetitive thought that encompass worry and rumination (e.g., Segerstrom et al., 2003; Watkins, 2008). Of note, these approaches assume that repetitive thought is not necessarily unconstructive: depending on its characteristics, it may take constructive forms, such as planning and problem solving. This assumption accords with the earlier reviewed evidence that structured problem solving in the evening may reduce presleep cognitive arousal, thereby facilitating sleep (Carney and Waters, 2006).

According to Watkins (2008), three main features of repetitive thought account for differential outcomes: (a) the valence of the thought content, (b) the intrapersonal and situational context in which repetitive thought occurs, and (c) the level of construal (abstract vs. concrete processing). As to *valence*, a majority of studies have, unsurprisingly, found that repetitive thought amplifies negatively valenced content, thereby leading to unconstructive outcomes. However, as Watkins (2008) pointed out, positive content, when excessively repeated, may also lead to unconstructive outcomes, for example, in individuals vulnerable to hypomania and mania. This consideration is of particular importance in the context of insomnia because, as reviewed earlier, positive mood may contribute to sleep-onset insomnia, at least in certain clinical conditions, such as bipolar disorder (Talbot et al., 2009). For the "embeddedness" of repetitive thought, both the *intrapersonal* and the *situational context* can range from negatively valenced (e.g., intrapersonal: dysphoric mood, negative expectations, low self-esteem; situational: stressful, traumatic events) to positively valenced (intrapersonal: positive mood, positive expectations, high self-esteem; situational: successful, rewarding events). The available empirical evidence suggests that the prevailing valence of an individual's cognitive–affective system largely determines whether repetitive thought is helpful or unhelpful (Watkins, 2008). For instance, lack of self-esteem and self-criticism have been shown to moderate the effect of rumination on mood and cognition (e.g., Robinson and Alloy, 2003) – findings that are of direct relevance for insomnia because, as reported earlier, self-criticism and self-attacking have been associated with disturbed sleep in a number of studies (e.g., Ree et al., 2005; Schmidt et al., 2009; Vincent et al., 2009).

Although valence, as mentioned earlier, is a major factor in determining the consequences of repetitive thought, it cannot explain all empirical findings. For instance, repetitive thought focused on negative content has been found to have constructive consequences in some studies of depressive rumination and of defensive pessimism. Moreover, simply focusing on positive content is not necessarily the most constructive form of repetitive thought, as revealed by research on process and outcome simulations (Watkins, 2008). To account for the available evidence, Watkins (2008) therefore proposed *level of construal* as a third main characteristic of repetitive thought and hypothesized that, at least when repetitive thought is focused on negative content, low-level, more concrete construals have more constructive consequences than do high-level, more abstract construals (see also Watkins, 2011). Regarding the mechanisms by which level of construal may influence the consequences of repetitive thinking, Watkins (2008) mentions that lower level construals should provide more elaborated and contextual detail about means, alternatives, and actions, thereby promoting problem solving in difficult, novel, or complex situations. Moreover, concrete construals may facilitate self-regulation by focusing attention on the immediate demands, reducing anxiety, and requiring less effort and thus consuming fewer resources. Finally, in negative situations, more abstract construals could provoke negative overgeneralizations in the sense that a single failure is interpreted in terms of individual inadequacy (e.g., "I am a failure") rather than in terms of situational difficulties (e.g., "I failed this hard exam"). Indirect evidence for

the relevance of level of construal for sleep-interfering mentation has recently come from studies suggesting that mindfulness-based cognitive therapy (MBCT), which uses meditation techniques to train patients away from abstract levels of processing and into more concrete modes of processing (e.g., Carney and Segal, 2005), may be beneficial for sleep (e.g., Ong et al., 2008).

In sum, the literature on repetitive thought clearly suggests that it might be promising to conceptualize presleep worry and rumination within a larger theoretical framework and to examine sleep-interfering mental activity along the dimensions of valence, intrapersonal and situational context, and level of construal. In particular, such a conceptualization may help elucidate the relations between thought control, imagery control, and affective control in the context of insomnia. For instance, worry comprises mostly negatively valenced verbal thoughts, rather than visual images, and is more abstract and less concrete than other types of mental processing (Sibrava and Borkovec, 2006). The avoidance of more concrete imagery in worry is postulated to serve a protective function, whereby physiological activation and emotional processing are reduced at the expense of maintaining anxious meanings. A promising avenue for future research will be to determine whether promoting more concrete levels of construal, for example, by means of MBCT or exposure therapy, may foster imagery-related emotional processing in worry, thereby improving sleep. Another fruitful line of inquiry will be to investigate the links between suppression and different forms of repetitive thought, as exemplified in the study by Watkins and Moulds (2009), in the context of insomnia.

NEW INSIGHTS FROM THE AFFECTIVE SCIENCE LITERATURE

In this section, we will suggest that the theoretical framework for repetitive thought as elaborated by Watkins (2008) can also serve as a conceptual platform to investigate affective control in insomnia. As detailed in the previous section, Watkins' framework incorporates affective states in two ways: (a) by taking into account thought valence and (b) by taking into account subjective affective states (e.g., negative mood) within the intrapersonal context. However, it should be underscored that Watkins advocates a clear distinction between cognitive and affective aspects of repetitive thought at the conceptual level and recommends the development and use of repetitive-thought questionnaires that do not confound repetitive thought with the degree of negative affectivity.

As in the case of repetitive thought, the question naturally arises as to which dimensions of affective states may impact sleep. In a first empirical attempt to uncover sleep-relevant dimensions of affective states, Stewart et al. (2011) asked a sample of students to complete questionnaires assessing depressive symptoms, trait anxiety, trait anger, trait positive affect, trait rumination, and sleep quality. Structural equation modeling analyses revealed that negative affect (the shared variance among the measures of depression, trait anxiety, and trait anger) was related to poor sleep, whereas no unique effects were observed for any of the negative and positive affective factors. Of note in the present context, the relation between negative affect and sleep was independent of rumination. The latter finding suggests that affective factors can influence sleep in ways that are separable from the effects of cognitive hyperactivity, thereby highlighting the added value of an affective science approach to insomnia.

On the basis of their findings, Stewart et al. (2011) concluded that ". . . the shared, not unique, aspects of negative emotional factors may be key determinants of sleep quality" (p. 609). However, a limitation of their study resides in the fact that it almost exclusively used trait measures. Thus, certain transient affective states of negative valence, such as regrets (e.g., Schmidt and Van der Linden, in preparation), or of positive valence, such as gratitude (e.g., Emmons and McCullough, 2003), may well have unique effects on sleep. Of note, induction of regrets prior to sleep delayed sleep onset independently of levels of depressed mood, as assessed by the Beck Depression Inventory-II (Beck et al., 1996), in the study by Schmidt and Van der Linden (in preparation). Moreover, positive affective states may have opposite effects on sleep in normal and certain clinical populations, such as individuals with bipolar disorder (e.g., Talbot et al., 2009). In addition, as the study by Talbot and colleagues also revealed, specific affective states may influence certain sleep parameters but not others (e.g., sleep-onset latency vs. rapid eye movement density). Clearly, more research is needed to unravel the associations between the various dimensions of affective states and subjective as well as objective sleep parameters in normal and clinical populations.

Apart from valence, arousal, which may be further differentiated into bodily excitation and intensity of feeling, is another fundamental dimension of affective states (e.g., Scherer, 2005). For instance, intense anger is likely characterized by high bodily excitation, whereas intense sadness may be accompanied by very low bodily activation. A better understanding of affective arousal in the context of insomnia is all the more important as this dimension may account for findings suggesting that both positive and negative affect may impair sleep (e.g., Talbot et al., 2009). Regarding the assessment of presleep arousal, research has as yet mainly focused on the cognitive and somatic dimensions. For example, Nicassio et al.'s (1985) Presleep Arousal Scale comprises a cognitive arousal and a somatic arousal subscale; more recently, a refined instrument to assess presleep cognitive arousal has been developed (Harvey and Espie, 2004). However, to the best of our knowledge, no instrument that specifically captures presleep affective arousal in its two subdimensions is currently available. Yet, outside of insomnia research, recent advances in affective sciences have led to the development of new assessment tools that could be adapted for investigations of sleep. For example, the Geneva Emotion Wheel (Scherer, 2005) allows one to rate the valence and intensity of a wide range of emotions by checking corresponding circles in a graphical display.

A further complication arises from the fact that, in the area of insomnia research, the terms "emotion" and "affect" have often been used interchangeably although the two concepts should be clearly distinguished. Within the field of affective sciences, emotions are generally considered as one particular type of affective state that features the following components (e.g., Scherer, 2005): (a) a cognitive component (appraisals); (b) a subjective feeling component (emotional experience); (c) a neurophysiological component (bodily symptoms); (d) a motor expression component (vocal, facial, and postural display); and (e) a motivational component (action tendencies). As compared with affect

dispositions (e.g., hostility) and moods (e.g., depressed mood), which have often been investigated in connection with sleep, emotions are: (a) typically linked to a stimulus event, (b) of shorter duration, and (c) of greater intensity. In view of their greater intensity, emotions deserve special scientific attention in the context of insomnia. As reviewed earlier, a number of recent investigations have provided evidence for sleep-interfering effects of specific emotions, such as regret, shame, and guilt (e.g., Schmidt and Van der Linden, 2009; Schmidt et al., in press). Given that emotions are endowed with specific action tendencies, emotions might have unique sleep-interfering effects that are not accounted for by general effects of valence or arousal. For instance, anger comprises action tendencies of opposition or assault (e.g., Frijda et al., 1989), whereas regret is typically accompanied by such action tendencies as to "kick oneself" and to correct one's mistake (e.g., Berndsen et al., 2004). Obviously, such action tendencies, if experienced at bedtime, have a strong potential to interfere with the process of falling asleep, especially in impulsive individuals with action–inhibition difficulties (cf. Schmidt and Van der Linden, 2009). Exploration of sleep-interfering action tendencies in the context of affective control might thus be a worthy endeavor.

Analysis of presleep mental activity in terms of these emotion components reveals yet another underinvestigated area in the context of affective control and insomnia, namely, the effects of (non-)expression of emotions. In a pioneering study in this field, Caska et al. (2009) found in a large sample of patients with coronary heart disease that self-rated suppression of anger expression was substantially associated with poor sleep quality. This association remained strong even after adjusting for demographics, comorbidities, lifestyle factors, medications, cardiac function, depressive symptoms, and trait anger. Apart from cognitive control strategies, such as thought suppression and worry, behavioral forms of affect control may thus have an important impact on sleep, which clearly calls for further investigation.

Also underinvestigated in the context of insomnia is what Gross and Thompson (2007) termed "antecedent-focused emotion regulation." This term refers to anticipatory forms of regulation that intervene before a full-blown emotional response occurs. For instance, an individual may selectively approach situations that are likely to evoke positive emotions or avoid situations that are liable to provoke negative emotions. To date, most studies in clinical research have focused on "response-focused emotion regulation," that is, retrospective forms of regulation, such as the cognitive strategies of thought suppression and worry, which are typically used to deal with emotions once they have occurred. In the first study to investigate the relations between antecedent-focused emotion regulation and sleep, Schmidt and Van der Linden (2011) hypothesized that individuals who have difficulties anticipating potential future regrets are at an increased risk of blindly running into situations or adopting behaviors that are prone to evoke regrets. As a consequence, they should experience more actual regrets and related counterfactual emotions, which have been shown to interfere with sleep (Schmidt and Van der Linden, 2009; Schmidt et al., in press). To test these ideas, Schmidt and Van der Linden (2011) developed the Regret Anticipation Failures Scale and administered it, within a battery of other questionnaires, to university students. Results supported the predictions mentioned: regret anticipation failures in the course of the day were associated with the frequency of regret-related counterfactual thoughts and emotions at bedtime, and the latter mediated the effect of regret anticipation failures on self-rated insomnia severity. These preliminary findings encourage further investigation of the implications of antecedent-focused emotion regulation for sleep. For instance, an intriguing question is that of whether training in affect anticipation might help people avoid negative emotional incidents in the course of the day, thereby obviating the need to engage in response-focused emotion regulation (e.g., thought suppression, worry) at the end of the day.

SUMMARY AND OUTLOOK

In the first part of this article, we reviewed a coherent body of evidence suggesting that dysfunctional forms of cognitive control, such as thought suppression, worry, rumination, and imagery control, are associated with sleep disturbance, as they are with other forms of psychopathology. In the second part of this article, we presented more recent lines of evidence indicating that dysfunctional forms of affective control, such as problems with down-regulating negative and positive affective states, are also related to sleep disturbance, and that sleep disturbance, in turn, can impair next-day affect control. In the third part of this article, we outlined how principles from the repetitive-thought literature and the affective science literature can inspire new lines of investigation and help develop a theoretical framework that integrates the roles of cognitive and affective control in insomnia.

An important theoretical question that calls for further research is that of whether dysfunctional forms of cognitive and affective control may be viewed as separate, though related, mechanisms, or may rather stem from a unitary, core mechanism. As reviewed earlier, some of the available suggests that dysfunctional forms of cognitive control, such as thought suppression, are often used to down-regulate negative affective states, for example regrets (e.g., Schmidt et al., in press). However, there is also evidence suggesting that cognitive factors and affective factors may independently impact sleep. For instance, in the previously mentioned study by Stewart et al. (2011), the relation between negative affect (as captured by the shared variance among measures of depression, trait anxiety, and trait anger) and sleep was independent of rumination. Conversely, another study has revealed that rumination may in important ways contribute to clinical insomnia independently of depressed mood states (Carney et al., 2010). Taken together, these preliminary findings suggest that affective factors might contribute to insomnia over and above the influence of well-established cognitive factors.

Regarding methodology, a limitation of the reviewed literatures on cognitive and affective control in insomnia resides in the fact that most studies have as yet relied on self-report. However, conclusions from these investigations continue to be strengthened and extended by the use of behavioral measures, including actigraphy (e.g., Omvik et al., 2007), physiological measures (e.g., Baglioni et al., 2010b), functional brain imaging (e.g., Yoo et al., 2007), and clinical intervention studies (e.g., Haynes et al., 2006). Clearly, more research with these methods is needed to advance our understanding of the interplay between cognitive and affective factors in insomnia.

With respect to the treatment of insomnia, the reviewed literatures contain a number of indications of how the standard cognitive–behavioral therapy (e.g., Bootzin and Epstein, 2011) could be complemented with interventions that specifically address dysfunctional forms of cognitive and affective control. For instance, training in structured problem solving might counteract non-constructive forms of repetitive-thought prior to sleep (e.g., Carney and Waters, 2006), or training in self-compassion (e.g., Gilbert and Procter, 2006) might defuse sleep-interfering effects of self-attacking (e.g., Schmidt et al., in press). These techniques exemplify how basic research into cognitive and affective control in insomnia may ultimately translate into new forms of intervention.

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Reward and punishment effects on error processing and conflict control

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Recently, positive affect has been reported to reduce cognitive conflicts and adaptations related to conflict control. van Steenbergen et al. (2009) proposed that the aversive quality of conflicts drives short-term adaptations following a conflict. They reasoned that monetary gain and its positive emotional consequences might counteract the aversive quality of conflict and hence reduce subsequent adaptations. In two experiments, we combined Simon-type conflicts with monetary gains and losses in between trials and analyzed event-related brain potentials. In Experiment 1, gains and losses occurred randomly between trials as a lottery, whereas in Experiment 2 gains and losses were contingent upon performance, either rewarding the 25% fastest responses or penalizing the 25% slowest responses. In Experiment 1, conflict adaptation was completely unaffected by gains or losses; contrary to predictions, in Experiment 2, conflict adaptation in reward blocks was more pronounced after a gain. In Experiment 2 we also investigated the error-related negativity (ERN) – a brain signal proposed to be related to performance monitoring. The ERN and behavioral post-error slowing were enlarged in the context of reward; therefore, reward increases error adaptation, possibly by enhancing the subjective value of errors. In conclusion, affective modulations of conflict adaptations seem to be much more limited than previously asserted and adaptive mechanisms triggered by errors and conflicts dissociate.

Keywords: conflict adaptation, error processing, reinforcement, reward, punishment, event-related brain potentials, error-related negativity

INTRODUCTION

Despite many years of research on cognitive conflicts, the potential role of emotions or affects in these situations is taken into account only recently. The present study investigates the effect of reward-induced motivational states on one element of cognitive control during conflict processing – action monitoring. Monitoring one's own actions is a critical precondition for adaptive behavior in general and for handling cognitive conflicts in particular.

The original conflict monitoring account (Botvinick et al., 2001) postulated that the dorsal anterior cingulate cortex (ACC) responds to conflicts, arising during various tasks, by issuing a conflict signal. This conflict signal triggers strategic adjustments in cognitive control by redirecting attention according to the task demands. Thus, the ACC would serve as a conflict monitoring device. The conflict monitoring theory stimulated research interests in sequential dependencies during conflict processing. In typical interference tasks like the Stroop, Flanker, or Simon task, a set of multidimensional stimuli is assigned to a set of responses. Usually, only one stimulus dimension is task-relevant, while other stimulus dimensions are task-irrelevant but at least one of them shares features with the relevant dimension (Lu and Proctor, 1995). In the Simon task, for example, left or right-hand responses are performed as a function of a non-spatial stimulus feature (S; e.g., shape or color) while the stimuli are presented either on the left or right-side. Responses (R) are faster and more accurate when S and R locations correspond (C, compatible events) than when they do not correspond (IC, incompatible events). In general, incompatible trials provoke conflicts in information processing when at least one feature of S or R contradicts the correct response. For example, in the Simon task, the stimulus may activate a right-side response by virtue of its (irrelevant) location, which contradicts a left-side response demanded by the relevant stimulus dimension, resulting in slower and more error-prone responses.

In most kinds of tasks with S–R conflicts, conflict-strength in the current trials depends on the correspondence condition of the preceding trials: after non-corresponding events conflicts are much smaller than after corresponding events (Gratton et al., 1992; Stürmer et al., 2002; Egner et al., 2007). According to the conflict monitoring approach, conflicts redirect the attentional focus to task-relevant features, diminishing the influence of task-irrelevant features, hence reducing the conflicts between these features and the response on the next trial.

In a recent extension of his conflict monitoring account, Botvinick (2007) suggested that the processing of conflicts is effortful and therefore aversive. Hence, conflicts should bias decision-making toward more efficient task strategies. The presumable redirection of attention after a conflict trial may be a direct consequence of the aversive efforts in dealing with this conflict. If conflict adaptation behavior is triggered by the negative affect elicited by conflicts, one may presume that positive affect would counteract conflict adaptation. This assumption is supported by findings of Kuhl and Kazen (1999), showing that the

Stroop effect is largely reduced by the short-term induction of positive affect. Following the suggestion that conflicts are experienced as negative, van Steenbergen et al. (2009) reasoned that monetary gain and its positive emotional consequences might counteract the aversive quality of the preceding conflict and hence reduce subsequent conflict-driven adaptation processes. Indeed, in a flanker task a small but significant reduction of conflict adaptation was found after monetary gain. In a follow-up study, van Steenbergen et al. (2010) applied mood induction and showed that as a trend-less positive mood induction tended to be associated with larger conflict-related adaptation. Taken together, the authors concluded that affect adaptively regulates cognitive control.

A direct link between affective and cognitive processing in conflict control was already implied in the seminal model by Miller and Cohen (2001). In this account, the prefrontal cortex (PFC) establishes S–R mappings by biasing competition between conflicting sensory inputs or motor outputs to favor relevant aspects for current task-performance. Phasic dopamine (DA) release by the midbrain DA system plays a major role in gating the appropriate update of task-relevant goal representations in the PFC. Thus, reward-driven DA release related to the reward prediction error (Schultz, 1998) is proposed to strengthen top-down control over bottom-up processing. One could therefore assume that conflict adaptation as a top-down control process should be enhanced after a reward when DA is released.

This assumption is in line with studies investigating conflict adaptation in Parkinson's disease (PD) patients who suffer from a low level of midbrain DA. Conflict adaptation in a Simon interference task was much reduced in PD patients (Praamstra and Plat, 2001; Fielding et al., 2005). These findings contrast with the view of van Steenbergen et al. (2009) who claimed that DA bursts were responsible for the reduced conflict adaptation observed in the context of reward.

A further problematic point for the idea that conflict adaptation is triggered by the aversiveness of the efforts involved in cognitive conflict processing is the lack of evidence for the purported negative emotional valence of cognitive conflicts. As shown by Schacht et al. (2010) in a direct comparison of Go/Nogo and Simon tasks, the emotions elicited by conflicts are task specific and not necessarily aversive. Emotional responses, indicated in a number of psychophysiological parameters, were only present in Go/Nogo conflicts but not in incompatible Simon task trials. Moreover, the construal of conflicts as aversive, adaptation-driving events was further called into question by indications that the emotions elicited in Nogo conflicts seem to be appetitive rather than aversive (cf. Schacht et al., 2009).

In sum, the theoretical predictions for the relationship between affect and conflict processing are controversial. Whereas van Steenbergen et al. (2009) hold that conflict adaptation is triggered by the aversive nature of conflict processing, other findings indicate that conflict adaptation should be facilitated by emotionally positive, DA-releasing events or states.

Here we tested in two experiments with the Simon task whether reward counteracts the presumably negative experience of a conflict and hence reduces conflict adaptation as predicted by van Steenbergen et al. (2009). In Experiment 1, reward was presented non-contingent to behavior, attempting to replicate the study of van Steenbergen et al. (2009) with a different conflict task. Because the predicted effect was absent, Experiment 2 explored the effects of presenting reward and punishment contingent upon performance.

EXPERIMENT 1

In the first experiment, a Simon task was combined with wins or losses in between two Simon trials. Wins and losses were not related to participant's performance but were presented at random, closely replicating the flanker task study van Steenbergen et al. (2009). In the present experiment, one of two stimuli appeared above or below fixation and required a choice response according to the stimulus shape on an upper or lower key. This procedure was similar to that of Stürmer et al. (2002) where context-driven adaptation had been present also when direct trial repetitions were excluded. Usually, responses to direct trial repetitions are very fast and confound with sequential effects related to conflict adaptation. In order to avoid these simple priming mechanisms unrelated to cognitive control (Mayr et al., 2003), we excluded direct repetitions by experimental design.

To control whether the affective manipulation by gain signals was effective, we recorded event-related brain potentials (ERPs) to the win and loss signals presented in between Simon trials (Holroyd et al., 2008). In contrast to van Steenbergen et al.'s (2009) study where gain signals directly followed the responses, we inserted a 500-ms interval after responses to avoid an overlap of response-related and gain signal-related ERPs. To guarantee a suitable baseline for ERP analyses we extended the inter-trial interval from 200 to 400 ms, as used by van Steenbergen and colleagues, to 1000 ms in the present study. Moreover, to ensure a positive payoff at the end of the experiment – maintaining participant's motivation – monetary gains per trials exceeded the losses by 0.05 €. Slightly higher wins than losses had the additional advantage that an influence of rewards on conflict adaptation was emphasized.

METHOD AND MATERIAL

Participants

Twenty-one neurotypical adults (age range = 20–49 years, $M = 28.5$ years, SD = 9.2 years; 4 males) participated in the experiment. All were right-handed (handedness score = 75.6) with normal or corrected-to-normal vision. Data of one further participant had to be discarded due to excessive error rates (>30%). Prior to testing, participants provided written consent according to the declaration of Helsinki and completed a handedness questionnaire (Oldfield, 1971).

Experimental setup and design

Participants were seated in a sound-attenuated and dim lit electrically shielded chamber. All stimuli were presented on a 17″ monitor of a Pentium processor using Presentation software, at a viewing distance of approximately 80 cm.

Participants responded with left and right index fingers to the shape of the stimuli using two vertically aligned response keys. In compatible trials stimulus and response position corresponded whereas in incompatible trials they did not. The stimuli consisted of a white square and rhombus, presented randomly above or below a central fixation point against a gray background.

Each trial started with the presentation of a central fixation point for 1000 ms, followed by the Simon stimulus shown for 200 ms approximately 1.5 cm above or below the fixation point. After a practice block participants were informed that they could earn between 10 and 20 € via a lottery algorithm that would provide gains or losses independently of their performance. The gain signals appeared 500 ms after the response (or 1.5 s after the stimulus in case of an omission) and consisted of a green, red, or blue circle (all 1.2 cm in diameter) displaying a monetary win (+0.25 €), a loss (−0.20 €), or a blank (0.00 €), respectively. Gain signals (win, loss, or blank) were presented centrally for 500 ms. Win, loss, and blank feedbacks appeared randomly with equal probabilities. After 60 practice trials, eight blocks of 120 trials were presented.

EEG recording and processing

The EEG data was recorded from 60 electrodes placed in an electrode cap and referenced to the left mastoid with a band-pass of 0.01–250 Hz at a sampling rate of 500 Hz. Vertical and horizontal electro-oculograms (EOG) were recorded from external electrodes. Electrode impedance was kept below 5 kΩ, using ECI electrode gel. Offline, continuous data were down-sampled to 250 Hz, re-referenced to an average mastoid reference; blink correction was applied using independent component analyses as implemented in Brain Vision Analyzer. Artifacts were automatically rejected, eliminating epochs with voltage steps per sampling point >50 μV, and low activity (<0.5 μV) within a 100-ms window. Offline, data were filtered, using Butterworth Zero Phase Filters (Time Constant: 3.18 s, 48 dB/oct; High Cut-off: 30 Hz, 48 dB/oct). ERPs related to the gain signals were segmented into 1200 ms epochs, starting 200 ms before stimulus presentation; a 100-ms pre-stimulus baseline was applied.

RESULTS

Performance

Errors occurred in 4.1% of all trials (compatible = 2.4%; incompatible = 5.7%). An ANOVA on error rate including the factors compatibility (2 – C, IC) and gain signal (3 – win, blank, or loss) yielded a significant main effect of compatibility; $F(1,20) = 32.72$, $p < 0.001$. Neither the main effect of gain signal, nor the interaction of feedback and compatibility were significant, Fs < 1.

In order to test the influence of different gain signals on conflict adaptation, we analyzed correct trials following correct trials and separated them according to their current compatibility, the compatibility of the preceding trial, and the feedback following the preceding trial. Only RTs > 100 ms entered into subsequent analyses. An ANOVA on RTs with factors compatibility (2 – C, IC), predecessor (2 – C, IC), and gain signal (3 – win, blank, or loss) yielded a significant main effect of compatibility, $F(1,20) = 47.69$, $p < 0.001$, a main effect of predecessor, $F(1,20) = 25.18$, $p < 0.001$, and a significant interaction of compatibility and predecessor, $F(1,20) = 117.64$, $p < 0.001$. However, there was neither a main effect of feedback nor an interaction with any of the other factors (see **Table 1**), Fs < 1. Following van Steenbergen et al. (2009), we calculated a conflict adaptation measure that integrates the speed-up and slow-down of compatible and incompatible trials using the formula [(CI − CC) − (II − IC)]. Mean conflict adaptation scores (see **Table 1**) for trials after loss, win, and blank signals were 73, 70, and 81 ms, respectively. An ANOVA on the conflict adaptation measure with the factor preceding gain signal (3 – win, blank, or loss) did not yield any significant differences, $F(2,40) = 1$.

Table 1 | Reaction times (RT) and error rates (ER) as a function of condition in Experiment 1.

Trial type	RT in ms (SD)	ER (SD)
WIN SIGNAL		
Compatible preceding compatible (cC)	420 (74.9)	0.7 (1.8)
Incompatible preceding compatible (iC)	470 (81.9)	3.7 (3.2)
Compatible preceding incompatible (cI)	487 (80.7)	7.2 (5.4)
Incompatible preceding incompatible (iI)	466 (85.7)	3.9 (3.5)
Simon effect	32 (21.5)	3.4 (3.0)
Conflict adaptation effect	70 (29.0)	6.2 (5.9)
BLANK SIGNAL		
Compatible preceding compatible (cC)	417 (75.5)	1.5 (2.2)
Incompatible preceding compatible (iC)	471 (87.8)	3.8 (4.5)
Compatible preceding incompatible (cI)	494 (84.3)	7.9 (6.1)
Incompatible preceding incompatible (iI)	467 (88.0)	3.7 (3.7)
Simon effect	37 (25.0)	3.2 (2.7)
Conflict adaptation effect	81 (37.9)	6.5 (5.8)
LOSS SIGNAL		
Compatible preceding compatible (cC)	420 (76.6)	1.0 (1.7)
Incompatible preceding compatible (iC)	472 (80.6)	3.8 (4.3)
Compatible preceding incompatible (cI)	487 (82.4)	7.6 (6.6)
Incompatible preceding incompatible (iI)	466 (88.7)	3.5 (3.3)
Simon effect	30 (21.1)	3.1 (3.1)
Conflict adaptation effect	73 (44.8)	6.9 (7.7)

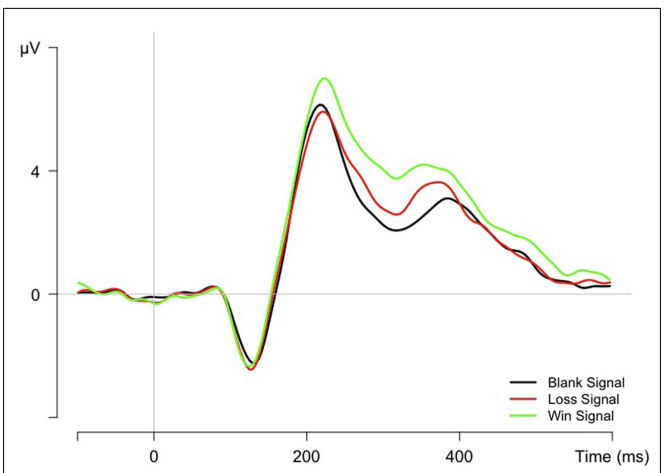

FIGURE 1 | Grand average ERPs at FCz electrode time-locked to gain signal presentation after correct trials.

Event-related potentials

Feedback-related ERPs were analyzed on mean amplitudes at the FCz electrode between 225 and 275 ms after gain signal onset. In this time interval win signals elicited a significantly larger positivity (2.3 μV) as compared to blank (1.8 μV) or loss signals (1.7 μV), $F(2,22) = 7.3$, $p < 0.01$ (see **Figure 1**).

DISCUSSION

In the present Simon task, we found a reliable conflict adaptation effect of 75 ms in RTs. Although the conflict adaptation effect in the present study was far bigger than in the studies by van Steenbergen et al. (2009, 2010), in direct contradiction to their findings, it was unaffected by the type of preceding gain signal.

Our feedback manipulation was efficient as demonstrated by differential effects on the gain signal-related ERPs. Wins elicited more positive-going ERP deflections following feedback onset as compared to losses or blank feedback. This finding is in line with the feedback-correct related positivity as reported for example by Holroyd et al. (2008). Obviously, at least win signals were registered by the cognitive system of our participants. Therefore, the absence of affective modulation of conflict adaptation in the present experiment cannot be ascribed to an ineffective affective manipulation.

Possible reasons as to the discrepancy with the findings of van Steenbergen et al. (2009, 2010) will be elaborated in the General Discussion. In brief, one reason why motivationally significant stimuli were ineffective to modulate conflict adaptation here might be due to their unrelatedness to performance. It is conceivable that affect modulates conflict processing only if a direct connection between prior performance and reward/punishment can be made. Therefore, we conducted a second experiment where monetary gain and loss was contingent upon performance.

EXPERIMENT 2

In the two halves of this experiment, we either rewarded fast and correct responses or penalized slow responses. In case the proposed relationship between affect and conflict processing holds we assumed that affective modulations of behavioral adaptation effects would be present if reward and punishment were contingent upon performance. In addition, the specific influence of feedback-induced affect on reinforcement monitoring and error processing was investigated by analyzing the feedback-related negativity (FRN) and the error-related negativity (ERN) in the ERP.

The FRN in response to the reinforcement signal should indicate whether the performance-contingent feedback was registered by the participants. The FRN was first demonstrated in response to external feedback about incorrect responses (Miltner et al., 1997). It is elicited approximately 250 ms after the feedback stimulus with a fronto-medial scalp distribution. However the FRN was also present for feedback stimuli about losses and was larger than to gain signals (see Simons, 2010, for an overview).

Additionally, Experiment 2 investigated the influence of motivational states on cognitive control in error processing, which was possible here because error rates were larger than in Experiment 1 due to higher response speeds. Errors usually result in slower responses in the following correct trial. This so-called post-error slowing (PES) is seen as a compensatory control mechanism improving performance in subsequent trials (Gehring and Fencsik, 2001). According to the conflict monitoring account (Botvinick et al., 2001) errors result in strategic adaptations by increasing the response threshold in the next trial in order to reduce the likelihood of an upcoming error. Errors and its consequences are, hence, seen to affect cognitive control processes related to performance monitoring, similar to response conflicts in interference tasks (Yeung et al., 2004; but see Masaki et al., 2007; Notebaert et al., 2009, for an alternative view).

A prominent ERP component related to error processing is the ERN (Ne or ERN). The ERN is tightly linked to the incorrect response, starts with the erroneous response and peaks about 60 ms later (Leuthold and Sommer, 1999; Falkenstein et al., 2000). The main generator of the ERN is probably located within the ACC (Ridderinkhof et al., 2004) and closely related to the midbrain DA system. Holroyd and Coles (2002) argue that, like negative feedback, response errors induce a dip in DA-cell firing (Schultz, 1998) which transmits to the ACC. The resulting disinhibition of ACC neurons is proposed to generate the ERN. Usually, larger ERN amplitudes are associated with improvements in performance monitoring (Larson et al., 2007, 2009; Olvet and Hajcak, 2008). Findings of Larson and Clayson (2011) suggest that increases in ERN amplitude are related to more focused attention that improves executive functions.

The involvement of the midbrain DA system in the processing of motivationally salient events implicates a relation between ERN and affective processing as confirmed by numerous reports. Larson et al. (2006) showed that the ERN is larger to errors within a background of pleasant emotional pictures as compared to neutral or unpleasant pictures. They suggest that the positive affective context creates a mismatch to task errors, thus enlarging the ERN. The ERN amplitude might reflect the subjective value of an error as derived from the recent reward history (Holroyd and Coles, 2008). Ogawa et al. (2011) used feedback signals of personal relevance (the trainer's voice in members of a university tennis team) and found that verbal admonishments significantly reduced ERN amplitude. However, there are also reports of enlarged ERNs in the context of negative affect. Wiswede and colleagues induced emotions either by presenting IAPS pictures (Wiswede et al., 2009a) or by embodied emotions, that is, participants had to hold a chop stick with their lips making them either smile or not smile (Wiswede et al., 2009b). The ERN was reduced in the no-smile conditions and in a context of negative IAPS pictures. However, embodied emotions did not show any effects on behavioral performance, casting doubt on whether performance monitoring was affected.

To sum up, when affective manipulations showed an influence on behavioral performance in conflict and error processing (e.g., Larson et al., 2006), the ERN was enlarged in a positive affective context. In the present experiment, feedback was provided according to participants' performance, which should strengthen the link between affective and cognitive processing. We therefore expected an affective modulation of conflict control and error processing. The ERN as an indicator of performance monitoring in errors should be enlarged in the context of rewards. Moreover, PES as a consequence of performance monitoring after preceding errors should increase as well. Predictions for the modulation of conflict adaptation by affective states are not univocal. Assuming that the aversiveness of a conflict triggers conflict adaptation (van Steenbergen et al., 2009), we should observe reduced conflict adaptation in the context of reward when the positive experience counteracts the conflict experience.

METHOD AND MATERIAL

Participants

Twenty-six neurotypical participants took part in the experiment; four of them were excluded (one had received wrong instructions and the others produced error rates >20% in one of the experimental conditions). Of the remaining 22 participants (mean age = 24.6 years), 12 were female and all were right-handed (mean handedness score = 92.2; Oldfield, 1971). All participants reported normal or corrected-to-normal vision. Participants were informed in advance that they would receive at least 25 € for the 3.5-h session and all gave their informed consent to the study.

Experimental setup and design

The experimental setup was identical to Experiment 1 with the following exceptions. The participants' head rested on a forehead and chin rest at a distance of 80 cm to a computer screen within an eye-tracking device, providing a constant viewing distance. Participants always started with a practice block of 240 Simon trials without any feedback. Afterward either the punishment or reward block followed; each contained 720 trials with self-paced breaks after every 60 trials. The timing of stimuli within a trial was identical to Experiment 1.

The order of punishment and reward blocks, the mapping of hands to response keys (index fingers of right and left hand on the upper or lower response keys) as well as the stimulus–response mapping in the Simon task (pressing the upper or lower key in response to the square or diamond) were counter-balanced across participants. For a given participant, the hands-to-key assignment and the stimulus–response mapping were constant throughout the experiment. Stimuli were presented in randomized order and direct repetitions of both stimulus form and stimulus location were excluded by design. All four possible sequences of compatible and incompatible trials and compatibility of their predecessors were equiprobable.

A staircase algorithm controlled that the 25% slowest responses were penalized in the punishment block and the 25% fastest responses received a bonus in the reward block. Four separate algorithms were calculated for compatible and incompatible trials and each response hand, respectively. To this end, reaction times were monitored online over the last 40 trials by a staircase algorithm starting at the 25%-values of the practice block. This allowed calculating immediately after each response whether RT was among the 25% slowest or among the 25% fastest responses, respectively. An additional algorithm controlled how often the participant had received a gain or a loss on the 8 most recent trials. Whenever gain or loss signals were presented more or less often than in 25% of these 8 trials (i.e., 2 trials out of 8), the gain or loss rate was temporarily adjusted, so that every participant was continually rewarded or penalized in almost exactly 25% of the trials.

In the punishment block, participants started with a virtual sum of 50 €.In the punishment block, they lost 15 €-cent when responding too slow or committing an error. In the reward block, participants started without any seed money and could earn a bonus of 15 €-cent for fast responses or loose 15 €-cent after an error. Loss and gain were indicated by a pink or green disk marked with "−0.15" or "+0.15," respectively. An orange disk, marked with "−0.15," indicated the commission of an error. A blue disk, marked with "0.00," indicated blank feedback in trials where participants neither won nor lost. When the balance at the end of the experiment exceeded the regular participation fee of 25 €, the extra money was paid in addition ($M = 24.59$ €, gain range = 3.80–40.55 €).

EEG recording and processing

In general, EEG recording and preprocessing were identical to Experiment 1. The duration of feedback-locked ERP epoch was 1200 ms, starting 200 ms before feedback onset. The duration of response-locked segments was 1200 ms, starting 200 ms before the key press. All ERP segments were baseline-corrected with a 100-ms pre-event baseline. ERPs were averaged separately for each participant, electrode, and condition.

RESULTS

Performance

To test feedback effects on conflict processing we calculated the magnitude of the Simon effect depending on preceding feedback for each experimental block. Overall error rate was 11.7% (C: 7.6%, IC: 15.8%). Mean error rates in the punishment and reward blocks were 12.3 and 11.1%, respectively, and did not differ significantly, $t(21) = 1.1$, $p = 0.285$. Compatibility (2 – C, IC) by feedback (2 – gain/loss, blank) ANOVAs on error rates were run separately for the reward and punishment block and yielded a significant main effects of compatibility in the punishment block, $F(1,21) = 32.4$, $p < 0.001$, as well as in the reward block, $F(1,21) = 31.2$, $p < 0.001$. No main effects of feedback and no interactions were significant, neither in the punishment nor in the reward block, Fs < 1.1.

Only trials with correct responses preceded by correct ones and RTs > 200 ms entered into the following RT analyses. To examine the influence of performance-contingent feedback on conflict adaptation, we applied an overall ANOVA with repeated measures on factors compatibility (2 – C, IC), predecessor (2 – C, IC), reinforcement type (2 – reward block, punishment block), and feedback (2 – gain/loss, blank). This ANOVA revealed a main effect of compatibility, $F(1,21) = 87.95$, $p < 0.001$, a conflict adaptation effect, reflected in a significant compatibility × predecessor interaction, $F(1,21) = 103.55$, $p < 0.001$, and a four-way compatibility × predecessor × reinforcement type × feedback interaction, $F(1,21) = 11.33$, $p < 0.01$. The three-way interaction of compatibility × predecessor × reinforcement type was not significant, $F < 1$.

Following up on the four-way interaction, we calculated ANOVAs for each reinforcement type (reward and punishment block) with the factors compatibility (2 – C, IC), predecessor (2 – C, IC), and feedback (2 – gain/loss, blank). In the reward block, this analysis yielded a significant main effect of compatibility, $F(1,21) = 40.2$, $p < 0.001$, and a conflict adaptation effect as expressed in the interaction of compatibility and predecessor, $F(1,21) = 88.6$, $p < 0.001$. The three-way interaction of predecessor × compatibility × feedback was significant as well, $F(1,21) = 6.3$, $p < 0.05$, indicating that conflict adaptation was more pronounced after gain feedback than after blank feedback (see **Table 2**), $t(21) = 2.5$, $p < 0.05$.

Table 2 | Reaction times (RT) and error rates (ER) as a function of condition in Experiment 2.

Trial type	RT in ms (SD)	ER (SD)
REWARD BLOCK; AFTER BLANK FEEDBACK		
Compatible preceding compatible (cC)	302 (38.4)	3.4 (3.3)
Incompatible preceding compatible (iC)	345 (35.6)	10.9 (7.3)
Compatible preceding incompatible (cI)	366 (44.7)	21.5 (8.7)
Incompatible preceding incompatible (iI)	330 (48.6)	8.0 (4.0)
Simon effect	25 (20.5)	8.1 (6.5)
Conflict adaptation effect	79 (35.5)	21.0 (13.0)
REWARD BLOCK; AFTER GAIN FEEDBACK		
Compatible preceding compatible (cC)	299 (36.6)	3.0 (4.1)
Incompatible preceding compatible (iC)	347 (34.6)	13.0 (8.4)
Compatible preceding incompatible (cI)	372 (38.5)	24.6 (13.6)
Incompatible preceding incompatible (iI)	325 (45.1)	6.6 (4.3)
Simon effect	25 (18.3)	8.0 (7.6)
Conflict adaptation effect	95 (54.6)	27.9 (16.4)
PUNISHMENT BLOCK; AFTER BLANK FEEDBACK		
Compatible preceding compatible (cC)	297 (32.4)	3.9 (4.0)
Incompatible preceding compatible (iC)	344 (34.9)	13.3 (9.2)
Compatible preceding incompatible (cI)	371 (37.1)	24.4 (10.2)
Incompatible preceding incompatible (iI)	326 (38.9)	7.0 (4.7)
Simon effect	29 (17.8)	7.7 (6.3)
Conflict adaptation effect	93 (33.9)	26.8 (14.4)
PUNISHMENT BLOCK; AFTER LOSS FEEDBACK		
Compatible preceding compatible (cC)	303 (41.7)	3.5 (5.8)
Incompatible preceding compatible (iC)	350 (38.3)	13.8 (9.1)
Compatible preceding incompatible (cI)	373 (38.3)	25.1 (9.3)
Incompatible preceding incompatible (iI)	334 (42.8)	8.8 (8.9)
Simon effect	29 (16.5)	9.1 (8.7)
Conflict adaptation effect	86 (52.1)	26.6 (16.0)

The ANOVAs for the punishment block yielded a main effect of compatibility, $F(1,21) = 93.7$, $p < 0.001$, and a significant conflict adaptation effect (compatibility × predecessor), $F(1,21) = 88.6$, $p < 0.001$. Furthermore, a main effect of feedback was present, $F(1,21) = 7.6$, $p = 0.012$, reflecting generally faster RTs following blank feedback than after punishment. Importantly and in contrast to the reward block no interaction of conflict adaptation with feedback was apparent, $F < 1$.

As compared to blank feedback (79 ms) conflict adaptation after gain feedback (95 ms) was more pronounced in the reward block; however, conflict adaptation after gain feedback in the reward block did not differ significantly from that after loss feedback in the punishment block (86 ms), $t(21) = 1.3$, $p = 0.22$. Comparing gain feedback in the reward block with loss feedback in the punishment block refers to different portions of the RT distribution, hence, the 25% fastest response in the reward block were compared to the 25% slowest responses in the punishment block. To test whether conflict adaptation is generally enlarged for fast responses in the current trial we directly compared the 25% fastest responses between reinforcement blocks and did not find a significant difference in conflict adaptation, $F < 1$.

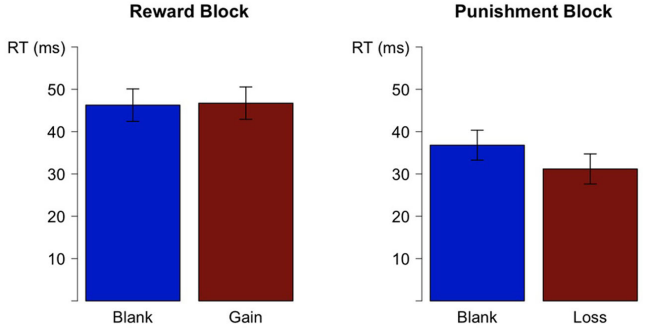

FIGURE 2 | Post-error slowing (PES = $RT_{N-1error} - RT_{N-1correct}$) for blanks and valent feedback preceding the correct response displayed separately for the reward and the punishment block.

We also tested whether PES was affected by a context of punishment or reward. PES (PES = $RT_{N-1error} - RT_{N-1correct}$) was calculated by subtracting trials with correct responses preceded by a correct response from those preceded by an error. In order to avoid that the directly preceding feedback confounds with general block effects, we compared trials following correct and erroneous trials after blank feedback. PES was larger in the reward block as compared to the punishment block (see **Figure 2**), $t(21) = 2.2$, $p < 0.05$. The within-blocks comparisons of PES after gain or loss feedback, respectively, with PES after blank feedback (see **Figure 2**) were neither significant in the punishment block, $t(20) = 1.5$, $p = 0.16$, nor in the reward block, $t(21) = 1.7$, $p = 0.088$.

Event-related potentials

The FRN was calculated as a peak-to-peak measure at FCz electrode (see **Figure 3**), following the procedure of Holroyd et al. (2003). We determined the negative peak between 150 and 300 ms following stimulus onset and marked the preceding positive peak as the beginning of the FRN. We calculated the peak-to-peak amplitude for all four conditions and applied a repeated measures ANOVA with the factors reinforcement type (2 – reward block, punishment block) and feedback (2 – gain/loss, blank). There was a main effect of reinforcement type, $F(1,21) = 8.96$, $p < 0.01$, and an interaction of reinforcement type and feedback, $F(1,21) = 13.49$, $p = 0.001$. *Post hoc* t-tests indicated larger FRN amplitudes to loss than blank feedback within the punishment block, $t(21) = 2.11$, $p < 0.05$, as well as smaller FRN amplitudes to gain than blank feedback within the reward block, $t(21) = 4.5$, $p < 0.001$. Further, gain feedback led to smaller amplitudes compared to loss feedback, $t(21) = 3.87$, $p = 0.001$.

The ERN was quantified in ERPs synchronized to incorrect button presses by detecting the minimum at FCz within a time-window from 0 to 100 ms. ERN amplitudes were larger in the reward block as compared to the punishment block (see **Figure 4**), $t(21) = 2.31$, $p < 0.05$.

DISCUSSION

Gains and losses showed differential effects on feedback-related brain potentials in Experiment 2. We can, therefore, safely

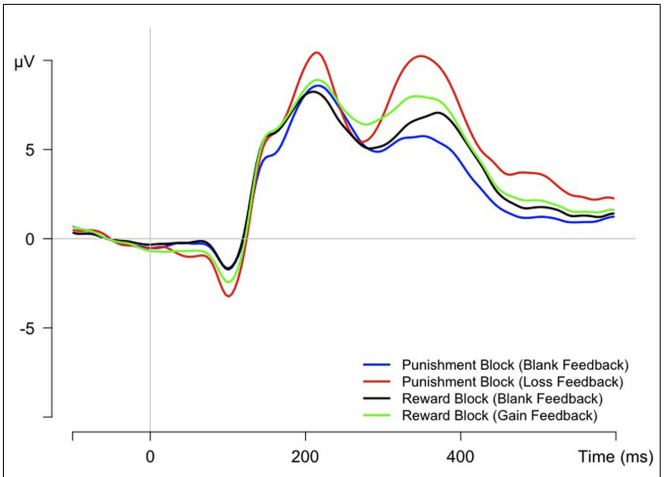

FIGURE 3 | Feedback-related grand average ERPs at FCz electrode time-locked to feedback signal presentation after correct trials.

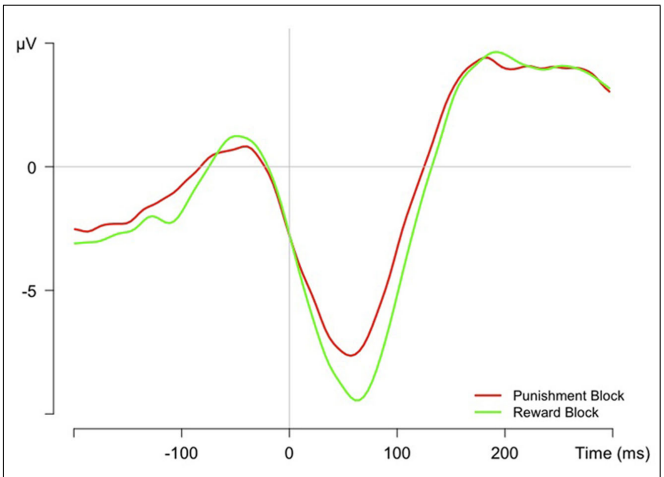

FIGURE 4 | The error-related negativity as grand average ERPs at FCz electrode time-locked to the error response.

conclude that motivationally salient stimuli were effective. In contrast to Experiment 1, we observed an affective modulation of conflict adaptation. Importantly, conflict adaptation was enhanced after gains in the reward block, whereas in the punishment block conflict adaptation was unaffected by feedback. This finding is at variance with the reports by van Steenbergen et al. (2009, 2010) that conflict adaptation is reduced by positive affect but is in line with the suggestion that top-down cognitive control is enhanced by DA bursts (Miller and Cohen, 2001) induced by reward. Although we observed a short-term effect of reward the overall motivational state – that is the context of reward or punishment, which varied between experimental halves – did not alter conflict adaptation.

Moreover, the ERN was enlarged and PES was enhanced in the reward as compared to the punishment block. The enlarged ERN with reward is in line with some previous studies (Larson et al., 2006; Holroyd and Coles, 2008; Ogawa et al., 2011) but is at variance with others that reported smaller ERNs under positive affect as induced by embodied emotions (Wiswede et al., 2009b) or larger ERNs with negative affect induced by IAPs pictures (Wiswede et al., 2009a). The latter studies, however, had not shown effects of affective induction on behavior, casting doubt on whether performance monitoring was involved.

Ogawa et al. (2011) who found reduced ERN amplitudes when feedback consisted in admonishments suggested that the personal relevance of the feedback might be a modulating factor for the processing of the errors. In their study, the generator of the ERN in the admonishment condition was located in a more rostral portion within the ACC, which has been related to the affective aspects of error processing. Activation in the rostral ACC has been suggested to inhibit processes in the dorsal ACC (Bush et al., 2000). Such interpretation is supported by assumptions of current appraisal theories of emotion, as for instance the Component Process Model by Scherer (2001, 2010). In these frameworks, personal relevance serves as a major criterion for the elicitation and differentiation of emotions.

GENERAL DISCUSSION

The present study aimed at testing whether motivational states and short-term effects of reward and punishments affect conflict control and performance monitoring. According to the conflict monitoring account (Botvinick, 2007), conflicts are negative experiences that trigger strategic adjustments in cognitive processing in order to avoid future conflicts.

For Flanker tasks van Steenbergen et al. (2009) reported that positive affect, presumably elicited by non-contingent gain signals, reduces conflict adaptation. Against the background of the conflict monitoring account, these authors suggested that conflict adaptation was reduced because positive affect counteracts the negative experience of a conflict. In a closely related design, using the Simon interference task, we did not replicate the findings of van Steenbergen and colleagues. Conflict adaptation was not at all modulated by motivational state of the context although gain signal-related ERPs clearly showed that win and loss were differentially registered.

In our second experiment, gain and loss were contingent upon performance. Here, they reliably influenced conflict adaptation, which was in the reward block larger after a gain relative to a blank feedback. In the punishment block conflict adaptation did not differ after a loss compared with blank feedback. Moreover, conflict adaptation after gain in the reward block and after loss in the punishment block did not differ. One could therefore as well assume that blank feedback reduced conflict adaptation in the reward block. Anyway, this finding contradicts those of van Steenbergen et al. (2009) who reported less adaptation after gain signals.

CONFLICT ADAPTATION

There might be several reasons for the discrepancy between our findings and those of van Steenbergen et al. (2009, 2010). First, effects of emotion on conflict adaptation might be as task specific as the affective consequences of cognitive conflicts, as shown by Schacht et al. (2010) who found psychophysiological emotion

effects only for the Go/Nogo but not for the Simon task. Second, the discrepancy might be due to the fact that in our experiments direct repetitions were excluded in the conflict adaptation analysis, whereas – according to their description – this seems not have been the case in the studies of van Steenbergen et al. (2009). Not excluding conditions with especially fast responses due to direct repetitions includes a confound with conflict adaptation (Mayr et al., 2003). Third, differences in the timing of feedback signals between van Steenbergen et al. (2009) study and ours may have contributed to the discrepancies. Whereas van Steenbergen et al. (2009) presented their feedback directly after the response, we inserted a 500-ms interval. These differences in timing of feedback might have failed to interfere with conflict adaptation in the present study, although the ERP measures in our study indicated that positive feedback differed in processing from neutral or negative feedback. Inconsistent findings between the reports by van Steenbergen et al. (2009, 2010) and the present study do preclude strict conclusions about the role of positive affect in conflict adaptation.

ERROR-RELATED ADAPTATION

In addition to the immediate impact of rewards on conflict adaptation, the second experiment revealed an effect of general motivational state on performance monitoring in error processing. Positive affect increased the ERN and PES. The enlarged ERN under positive affect confirms previous reports (Larson et al., 2006; Holroyd and Coles, 2008) and might be due to a mismatch between a positive affective context and the error, which, in turn, might induce more conservative response strategies. An increased ERN is often associated with improvements in performance monitoring (Larson et al., 2007, 2009; Olvet and Hajcak, 2008). Better monitoring of errors might facilitate compensatory control mechanism improving performance in the subsequent trial (Gehring and Fencsik, 2001) resulting in increased PES. Although a relationship between ERN, performance monitoring, and subsequent PES seems to be plausible, this is – to our knowledge – the first study, which shows that positive affect modulates behavioral measures of error processing such as PES.

CONFLICT VS. ERROR-RELATED ADAPTATION

The results of Experiment 2 indicate increased performance monitoring in errors and conflict control under positive affect. The original conflict monitoring account (Botvinick et al., 2001) suggested one common mechanism underlying both conflict and error monitoring. Thus, both should result in identical or at least similar strategic adjustments. Recently, this suggestion is being called into question by reports in support of independent adaptive mechanisms. For instance, Notebaert and Verguts (2010) investigated conflict and error adaptation in a task-switching paradigm using two interference tasks. They showed that PES generalized across tasks whereas conflict adaptation did not, arguing against a unitary adaptation mechanism.

Hikosaka and Isoda (2010) suggested that adjacent medial–frontal brain areas are involved in two complementary modes of cognitive control. One operates on performance failures consisting in errors or unexpectedly high rewards ("reward prediction error"). Both situations seem to alter midbrain DA release and, hence, modulate activation of ACC neurons. This mode was called retroactive because the control processes were triggered by behavioral performance. The other control mode was called proactive because here an external cue indicated a new task context for response selection. It was suggested that the proactive control mode is mediated by the pre-supplementary motor area (pre-SMA). In contrast to the ACC, pre-SMA processing is not directly related to the midbrain DA system; only indirect relations exist via basal ganglia output targeting the pre-SMA.

Ullsperger and King (2010) extended this approach by assuming that the proactive control mode selectively prepares appropriate task sets and triggers conflict adaptation. Reactive control, however, enhances responsiveness to any potentially relevant stimulus. Reactive control is recruited by performance errors and triggers adaptation after errors (e.g., PES). Following up, midbrain DA is directly related to error adaptation via ACC processing but it is not to conflict adaptation via the pre-SMA.

Our finding that error processing is affected by motivational states could be accounted for by the proposed midbrain DA and dorsal ACC circuitries involved in reactive control. At the same time, modulations of conflict adaptation by motivational states were inconsistent between studies. Evidence for affective modulation of conflict adaptation is, therefore, only sparse. These less conclusive reports of affective modulations in conflict adaptation could be accounted for by the pre-SMA involvement in proactive control, which is not (directly) linked to the midbrain DA system probably crucial for cognitive–affective interactions.

Even when errors and conflicts show similar consequences on subsequent behavior by generally slowing responses in the upcoming event (Verguts et al., 2011), some aftereffects do dissociate. Conflicts result in more focused processing in the next trial thereby reducing potential conflicts. Errors, however, did not reduce upcoming conflicts.

CONCLUSION

Recent findings clearly speak for different adaptation mechanisms triggered by errors and conflicts. An increasing number of studies support cognitive–affective interactions in error processing. Affective modulations of conflict control are, however, less clear. We observed a temporary modulation of conflict adaptation effects only in blocks in which gain was achieved contingent upon task-performance. In contrast to van Steenbergen et al. (2009), we did not observe any effects of motivational state on conflict adaptation when gains and losses were applied non-contingent to task-performance as a lottery. Importantly, the overall context of reward or punishment did not alter conflict adaptation.

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Glancing and then looking: on the role of body, affect, and meaning in cognitive control

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In humans, there is a trade-off between the need to respond optimally to the salient environmental stimuli and the need to meet our long-term goals. This implies that a system of salience sensitive control exists, which trades task-directed processing off against monitoring and responding to potentially high salience stimuli that are irrelevant to the current task. Much cognitive control research has attempted to understand these mechanisms using non-affective stimuli. However, recent research has emphasized the importance of emotions, which are a major factor in the prioritization of competing stimuli and in directing attention. While relatively mature theories of cognitive control exist for non-affective settings, exactly how emotions modulate cognitive processes is less well understood. The attentional blink (AB) task is a useful experimental paradigm to reveal the dynamics of both cognitive and affective control in humans. Hence, we have developed the glance–look model, which has replicated a broad profile of data on the semantic AB task and characterized how attentional deployment is modulated by emotion. Taking inspiration from Barnard's Interacting Cognitive Subsystems, the model relies on a distinction between two levels of meaning: implicational and propositional, which are supported by two corresponding mental subsystems: the glance and the look respectively. In our model, these two subsystems reflect the central engine of cognitive control and executive function. In particular, the interaction within the central engine dynamically establishes a task filter for salient stimuli using a neurobiologically inspired learning mechanism. In addition, the somatic contribution of emotional effects is modeled by a body-state subsystem. We argue that stimulus-driven interaction among these three subsystems governs the movement of control between them. The model also predicts attenuation effects and fringe awareness during the AB.

Keywords: computational modeling, attentional blink, emotion, cognitive control, body-state

INTRODUCTION

Cognitive control is typically defined as the biasing of cognitive functions, perhaps especially perception and response, to promote "task-appropriate" behavior, and particularly to override pre-potent responses. While a valuable working hypothesis, such a definition poses several questions: what constitutes task-appropriate, indeed, what constitutes a task and, ultimately, what constitutes an organism's goals? Due partially to the constraints imposed by experimental method, the notions of task, goal and, thus, cognitive control, have tended to be narrowly prescribed. For example, the concept of task has, if only tacitly, been directly associated with the set of task instructions that can be easily and unambiguously imposed in well-controlled laboratory experiments; e.g., a participant might be instructed to report a letter in the color red.

This definition of cognitive control is, of course, limiting, artificial, and not fully reflective of the diversity of goal-driven control processes to be found beyond the sphere of traditional experimental work. For example, many psycholinguistic phenomena, such as, the Moses illusion (Erickson and Mattson, 1981), suggest that task set does not enforce strict categorical boundaries. In particular, the trajectory of task-focused processing seems unperturbed by small semantic inconsistencies; that is, when processing demands are high, the central executive seems content with a broad schematic consistency of meaning. In addition, although only relatively recently considered in the laboratory, it would seem clear that affect and body-state feedback in general, has a major role in guiding perception and action over and above its immediate goals. To take a very obvious example, "flight or fight" responses to threatening stimuli are surely prioritized, and accordingly bias attentional and response processes. In the extreme case, Ohman and Soares (1994) have shown that, compared to healthy controls, phobics have larger skin conductance responses to masked fear related pictures, such as snakes, even when they were unaware of their presentation. In addition, it has been reported that anxiety can modulate attentional control (Koster et al., 2006), or delay the disengagement of visual attention away from threatening stimuli (Fox et al., 2001; Yiend and Mathews, 2001; Georgiou et al., 2005). Bishop et al. (2004) have also shown an interaction between anxiety state and attentional focus on threatening stimuli.

Moreover, Leyman et al. (2007) have reported that patients with major depressive disorders show enhanced attention to angry faces compared to controls.

The literature's restricted perspective on cognitive control is particularly apparent in neural modeling of task set. Neural network models that address the issue at all, typically realize cognitive control as a statically configured task-demand system (Cohen et al., 1990; Houghton and Tipper, 1994; Bowman and Wyble, 2007; Zylberberg et al., 2010), which simply foregrounds task relevant pathways and backgrounds others. In particular, in such models, there is little consideration for how such a task-demand system knows what to foreground and what to background; how it might, indeed, configure such biasing; how these configurations may change according to performance; and the interaction between such configurations and affective/body-state influences. Modeling work focused on notions of conflict and entropy (Botvinick et al., 2001; Davelaar, 2008; Wyble et al., 2008), have brought a richer perspective on cognitive control, but the interrelationship between representation of meaning, affect, body-state, and dynamic reconfiguration of task set, remains only superficially explored.

Our central tenet is, then, that cognitive control does not provide a perfectly delineated task filter, which enforces absolute, affect-immune, categorical boundaries between target and non-target. In addition, we argue that this "imprecision" ought, in fact, to be adaptive and, thus, of functional value for the organism. There are a number of ways in which this imprecision may manifest itself.

1. Enforcement of task set may, to a significant degree, be reliant upon schematic (categorically loose) representations; what might be called *gist* meaning.
2. Affect and body-state in general may play a major role in guiding task-focus; and they interfere with goal-directed processing via two different pathways, i.e., by a body-state route or by a fast and direct route that bypasses body-state. In addition, as often demonstrated, anxiety impacts the reconfiguration of task set.
3. The benefit of (more schematic) gist-based filtering may be observed when the attentional system is challenged to the point of near-overload, as exemplified by phenomena such as fringe awareness (Mangan, 2001; May, 2004) and improved attentional blink (AB) performance in the presence of distraction (Olivers and Nieuwenhuis, 2005, 2006; Taatgen et al., 2007).

Our *glance–look* model realizes this broader notion of cognitive control by partitioning central executive mediated salience detection into two stages. The first of these, the *glance*, undertakes a schematic glimpse at meaning and is, also, the site at which affective and bodily evaluations guide attentional focus. In contrast, the second stage, the *look*, operates in a fashion more consistent with classic perspectives on salience detection and task-focus. That is, it performs a more detailed (referentially specific) analysis of meaning. These two stages map directly onto the *propositional* and *implicational* central executive subsystems in Barnard's (1985) interacting cognitive subsystems (ICS).

We will present the glance–look model and its interpretation of cognitive control as follows. Firstly, we will provide background on the experimental paradigm, i.e., the AB task (Raymond et al., 1992), which is well suited to revealing the dynamics of both cognitive and affective control in humans. In particular, it has been observed with ERP (Flaisch et al., 2007) and psychophysiologically (Phelps et al., 2006) that emotion does not only affect the processing of the affective stimulus itself, but also following stimuli, emphasizing the importance of the temporal profile of affective salience. We will argue that the AB task provides a suitable platform to study the complicated temporal structure when cognition and emotion interact. And then, we will review and highlight the structure and principles of the model's realization of salience detection and attentional control. The theory of the glance–look model is inherited from ICS; however, this particular computational implementation and its parameter setting are systematically justified here. In particular, a unique modeling approach with mathematical formalization of the model parameters is detailed in Appendix.

Secondly, we will model several experimental results from the literature covering semantic and affective influences on attentional control and AB attenuation effects due to distraction. In Experiment 1, we will describe how the glance–look model explains the semantic key-distractor AB phenomenon (Barnard et al., 2004). This will demonstrate the model's two levels of meaning: implicational (when glancing) and propositional (when looking). In particular, we will explain the finding of a classic AB in the semantic key-distractor task in terms of the glance subsystem's focus on (implicational) gist meaning. Furthermore, this meaning is represented in a self-organizing statistical learning framework: latent semantic analysis (LSA, Landauer and Dumais, 1997; Landauer et al., 1998, 2007). In Experiment 2, and again in a key-distractor AB setting, we consider the role of affective salience in guiding attentional focus. This involves adding a body-state subsystem to the glance–look model. In this way, we model the capacity for affectively charged key-distractors to generate a variety of AB profiles (Barnard et al., 2005; Arnell et al., 2007), dependent upon intrinsic salience of the affective key-distractor and participant group (anxious vs. non-anxious). In Experiment 3, we consider how guide of cognitive control by gist meaning can be functionally beneficial. We do this by exploring how the glance–look model exhibits a relatively graceful degradation in perception at high sensory loads, generating fringe awareness. In addition, we argue that, somewhat counter-intuitively, an increased reliance on implicational (gist) meaning can improve behavioral performance, consistent with overinvestment theories of temporal attention and the beneficial effect of distraction upon AB performance (Olivers and Nieuwenhuis, 2005, 2006; Taatgen et al., 2007).

Finally, we will draw general conclusions on the glance–look model's contributions in broadening the notion of cognitive and affective control. We will also suggest some possible neural correlates of our model, in particular, relating it to several cognitive neuroscience models of cognitive and affective interaction (Pessoa, 2008).

BACKGROUND

ATTENTIONAL BLINK TASK

Humans have an exceptional capacity for assessing the salience of the stimuli that arise in their environment and for adjusting

processing accordingly. For example, when standing on a street corner we are subject to a plethora of stimuli: cars passing, conversations amongst pedestrians, and street vendors plying their trade. When placed in such environments, humans are very good at prioritizing these competing stimuli: directing attention toward the highest priority events and ignoring the rest. Furthermore, when we perceive a significant event, such as a car careening off the road, the current task is interrupted and attention is redirected to reacting to the new event. It is also clear that there is a trade-off between the need to meet (potentially long-term) goals and the need to respond optimally according to the salience level of environmental stimuli. This suggests that a system of salience sensitive control exists, which trades goal-directed processing off against monitoring and responding to (potentially high salience) stimuli that are irrelevant to the current task. In previous work, we have proposed the glance–look model, which formally specifies mental representations and processes that support salience detection and attentional control in the context of temporal attention (Su et al., 2009; Bowman et al., 2011).

A classic experimental paradigm that explores the temporal deployment of attention is the AB task. Following on from earlier work by Broadbent and Broadbent (1987), Raymond et al. (1992) were the first to use the term AB. The task they used involved letters being presented using rapid serial visual presentation (RSVP) at around 10 items a second at the same spatial location. One letter (T1) was presented in a distinct color and was the target whose identity was to be reported. A second target (T2) followed after a number of intervening items, presence or absence of T2 was to be reported. Typically, participants had to report whether the letter "X" was among the items that followed T1. The key finding was that report of T2 was impaired as a function of serial position. That is, T2s occurring immediately after T1 were accurately detected – a phenomenon typically described as lag-1 sparing (Wyble et al., 2009). Detection then declined across serial-positions 2, and also 3, and then recovered to baseline around lags 5 or 6 (corresponding to a target onset asynchrony in the order of 500–600 ms).

As research on the blink and RSVP in general has progressed, it has become evident that the allocation of attention over time is affected by the meaning of items (Maki et al., 1997) and their personal salience (Shapiro et al., 1997b). There is also evidence from electrophysiological recording that the meaning of a target is processed even when it is not reported (Shapiro and Luck, 1999).

In order to examine semantic effects, Barnard et al. (2004) used a variant of the AB paradigm in which no perceptual features were present to distinguish targets from background items. In this task, words were presented at fixation in RSVP format. Targets were only distinguishable from background items in terms of their meaning. This variant of the paradigm did not rely on dual target report. Rather, participants were simply asked to report a word if it refers to a job or profession for which people get paid, such as "waitress," and these targets were embedded in a list of background words that all belonged to the same category. In this case, they were inanimate things or phenomena encountered in natural environments; see **Figure 1**. However, streams also contained a key-distractor item, which, although not in the target category, was semantically related to that category. The serial position that the target appeared after the key-distractor was varied. We call this the key-distractor AB task, which, importantly, enables us to observe and quantify the semantic imprecision of the task filter. That is, the key-distractor is not in the target category. However, it is semantically related to that category. The critical question, then, is can key-distractors capture attention, even though strictly, they are task irrelevant.

Participants could report the target word (accurate report), say "Yes" if they were confident a job word had been there, but could not say exactly what it was (to capture some degree of awareness of meaning), or say "No" if they did not see a target, and there were, of course, trials on which no target was presented. When key-distractors were household items, a different category from both background and target words, there was little influence on target report. However, key-distractors that referenced a property of a human agent, but not one for which they were paid, like "tourist" or "husband," gave rise to a classic and deep blink, see **Figure 5**. We call household items low salient (LS) key-distractors

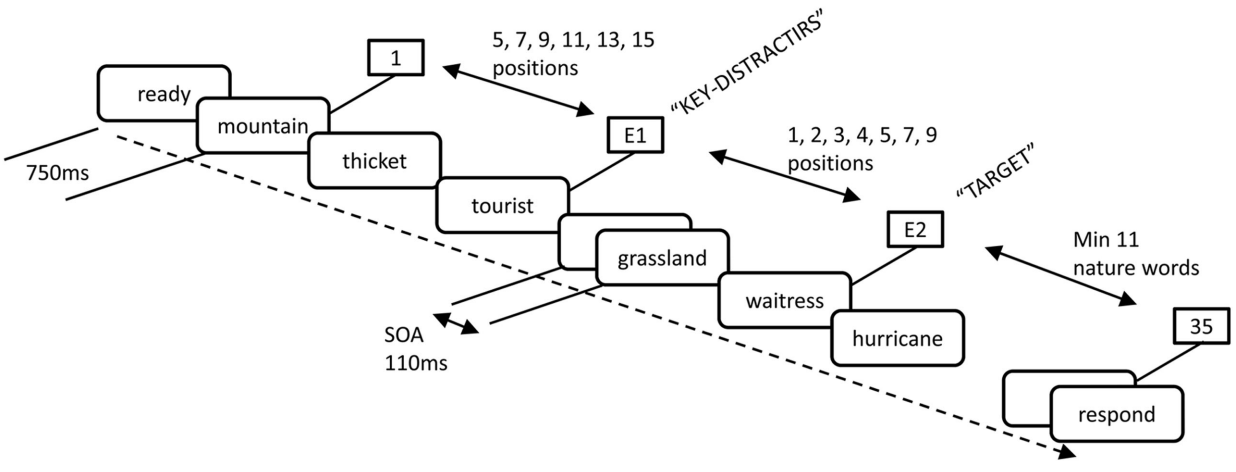

FIGURE 1 | Task schema for the key-distractor AB task; adapted from Barnard et al. (2004).

and human items high salient (HS) key-distractors. Thus, the task filter during an AB task can, at least partially, be "tricked" when facing semantically salient, but, in fact task irrelevant, stimuli.

THEORY OF THE GLANCE–LOOK MODEL

Over the last 20 years, the AB task has been the subject of very extensive empirical research, coupled with the development of a substantial body of theory (e.g., see Chun and Potter, 1995; Shapiro et al., 1997a; Visser et al., 1999; Bowman and Wyble, 2007). A specific focus for the glance–look model has been the key-distractor AB (Barnard et al., 2004), which it models using parallel distributed executive function. In this section, we will explain three principles that underlie the model and govern its perspective on cognitive control: sequential processing, two stages, and serial allocation of attention. Although the basic structure of the model has been proposed previously (Su et al., 2009; Bowman et al., 2011), parameter setting is only systematically justified here, and a unique modeling approach with mathematical formalization of the model is provided in Appendix. Importantly, in subsequent sections, where the model's scope is extended to other AB phenomena, e.g., the affective blink and the attenuation effect, the model parameters are unchanged.

Sequential processing

With any RSVP task, items arrive in sequence and need to be correspondingly processed. Thus, we require a basic method for representing this sequential arrival and processing of items. At one level, we can view our approach as implementing a pipeline. New items enter the front of the pipeline (in this case, from the visual system); they are then fed through until they reach the back of the pipeline (where they enter the response system). The key data structure that implements this pipeline metaphor is a *delay-line*. This is a simple mechanism for representing time constrained serial order. One can think of a delay-line as an abstraction for items passing (in turn) through a series of processing levels. In this sense, it could be viewed as a symbolic analog of a sequence of layers in a neural network; a particularly strong analog being with synfire chains (Abeles et al., 1993).

Every cycle, a new item enters the pipeline and all items currently in transit are pushed along one place. We shall refer to this as the *delay-line update cycle*, and assume that one cycle corresponds to 20 ms. This assumption is justified by the observation that underlying neural mechanisms can represent updates on a time scale of tens of milliseconds (Bond, 1999; Panzeri et al., 2001). Thus, in each delay-line update cycle, all delay-lines increment by one slot every 20 ms. Note, the update rate of the model is every 5 ms. This assumption is not constrained by neurobiology, but by the requirement of simulation, i.e., the sampling rate has to be faster than the update rate of constituent representations. This fine grain of time course allows us to be more discriminating with regard to the temporal properties of the AB. However, a high sampling rate would have implementation costs, in terms of how long simulations would take to run.

A delay-line is a very natural mechanism to use in order to capture the temporal properties of a blink experiment, which is inherently a time constrained order task. To illustrate the data structure, consider a delay-line of 10 elements, as shown in **Figure 2**, where

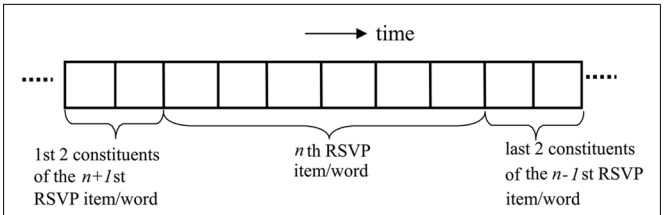

FIGURE 2 | A 10-slot delay-line with three RSVP items in progress through it.

indices indicate the position of the constituent representations of the corresponding RSVP item/word. We shall use this terminology throughout, i.e., a single RSVP item will be modeled by a number of constituents in a delay-line representation. We assume six constituent representations comprise one RSVP item/word, which approximates the 110-ms presentation used in most AB experiments (e.g., Barnard et al., 2004).

A constituent representation in the model contains three variables. The first one is the identity of the item. The second and the third elements are an implicational and a propositional salience assessment respectively. The origins of these terms are outlined in later sections. The salience assessments are initially set to un-interpreted.

Two stages

As noted earlier, a number of theoretical explanations and indeed computational models of the AB have been proposed; see Bowman and Wyble (2007) for a review. However, apart from the model discussed in Barnard and Bowman (2003), all these proposals seek to explain "basic" blink tasks, in which items in the RSVP stream are semantically primitive, e.g., letters or digits. Consequently, none of these "mainstream" theories or models is directly applicable to semantic and affective influences on the shape of the blink curves. However, of these previous theories, that introduced by Chun and Potter (1995) has some similarities to this model. Their theory assumes two stages of processing. The first stage performs an initial evaluation to determine featural properties of items, including "categorical" features. This stage is not capacity limited and is subject to rapid forgetting. The second stage builds upon and consolidates the results of the first in order to develop a representation of the target sufficient for subsequent report. This stage is capacity limited, invokes central conceptual representations and storage, and is only initiated by detection of the potential target on the first stage.

Like Chun and Potter (1995), we have argued elsewhere for a two-stage model (Barnard and Bowman, 2003; Barnard et al., 2004), but recast to focus exclusively on semantic analysis and executive processing. In particular, Barnard and Bowman (2003) modeled the key-distractor blink task using a two-stage model. In the context of modeling distributed control, we implemented the two-stage model as a dialog between two levels of meaning. In the first stage, a generic level of semantic representation is monitored and initially used to determine if an incoming item is salient. If it is found to be so, then, in the second stage, the specific referential meaning of the word is subjected to detailed semantic scrutiny in

order to access its salience in the context of the specific task set. In this stage, a word's meaning is actively evaluated in relation to the required referential properties of the target category. If this reveals a match, then the target is encoded for later report. The first of these stages is somewhat akin to first taking a *"glance"* at generic meaning, with the second akin to taking a closer *"look"* at the relationship between the meaning of the incoming item and the target category. These two stages are implemented in two distinct semantic subsystems proposed within our model for cognitive and affective control: the *implicational subsystem* or Implic (which supports the first stage) and the *propositional subsystem* or Prop (which supports the second; Barnard, 1999). Except for these two subsystems (Implic and Prop), the model, in its most basic form, also includes Source and Sink, which reflect the perceptual processing and response systems respectively, see **Figure 3**.

Implic and Prop process qualitatively distinct types of meaning. Implicational meaning, is holistic, abstract and schematic, and is where affect is represented and experienced (Barnard, 1999). Propositional meaning is classically "rational," being based upon propositional representation and captures referentially specific semantic properties and relationships. The exchanges between two levels of meaning reflect distributed executive functions, rather than a centralized executive control system, which might suffer from a homunculus problem.

There is significant evidence that a good deal of human semantic processing relies upon propositionally impoverished representations. It is this evidence that gives the clearest justification for the existence of a distinct implicational level of meaning. In particular, semantic errors make clear that sometimes we only have (referentially non-specific) semantic *gist* information available to us, e.g., false memories (Roediger and McDermott, 1995) and the Moses illusion (Erickson and Mattson, 1981). With respect to the latter, when comprehending sentences, participants often miss a semantic inconsistency if it does not dramatically conflict with the gist of the sentence, e.g., in a Noah specific sentence, such as "How many animals of each kind did Moses take into the Ark?" most people respond "two" even though, when questioned, they know that the relevant biblical character was really Noah rather than Moses. Substitution of Moses for Noah often fails to be noticed, while substitution with Nixon, or even Adam, is noticed. This is presumably because both Moses and Noah fit the generic (implicational) schema "aged male biblical figure," but Nixon and Adam do not.

In addition, Gaillard et al. (2006) reported that in a subliminal priming study, semantic gist information was available even when participants failed to correctly name masked emotional words. Specifically, in error, words semantically related to target words were often reported (e.g., target "war," response "danger"; target "bomb," response "death"). This suggests the availability of implicational meaning and the absence of veridical propositional meaning. In addition, deep dyslexia (Coltheart et al., 1987), in which sufferers generate incorrect referents (e.g., reading "lion" as "tiger"), can be regarded as a marker of broadly intact extraction of implicational meaning and significantly impaired attribution of referentially more stringent propositional meaning.

To tie this into the previous section, the implicational and propositional subsystems perform their corresponding salience assessments as items pass through them in the pipeline. We will talk in terms of the *overall delay-line* and *subsystem delay-lines*. The former of which describes the complete end-to-end pipeline, from the visual to the response subsystem, while the latter is used to describe the portion of the overall pipeline passing through a component subsystem, e.g., the propositional delay-line.

Serial allocation of attention

Our third principle is a mechanism of attentional engagement and cognitive control. It is only when attention is engaged at a subsystem that it can assess the salience of items passing through it. Furthermore, attention can only be engaged at one subsystem at a time. Consequently, semantic processes cannot *glance* at an incoming item, while *looking* at and scrutinizing another. This constraint will play an important role in generating a blink in our models.

When attention is engaged at a subsystem, we say that it is buffered (Barnard, 1999). In the context of this paper, the term buffer refers to a moving focus of attention. Thus, salience assignment can only be performed if the subsystem is buffered and only one subsystem can be buffered at a time. The buffer mechanism ensures that the central attentional resources are allocated serially, while data representations pass concurrently, in the sense that all

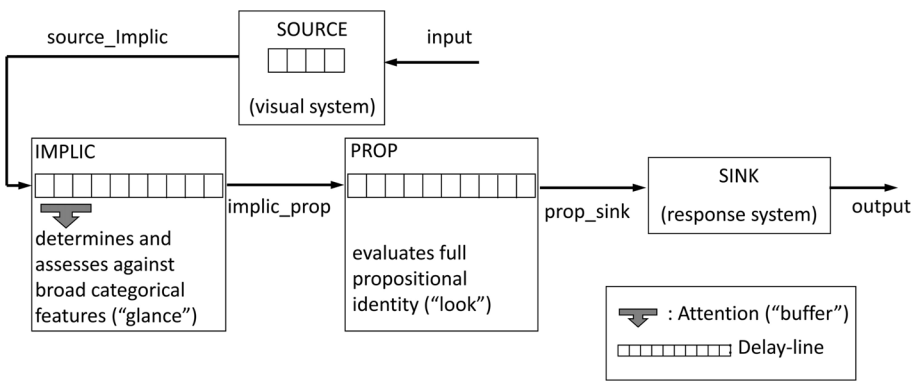

FIGURE 3 | Top-level structure of the glance–look model with Implic attended (buffered).

data representations throughout the overall delay-line are moved on one place every 20 ms.

Each subsystem assigns salience on the basis of the constituent representations entering it. Salience assignment is performed at the delay-line of the subsystem when it is buffered. As explained previously, an item (i.e., a word) in RSVP is composed of several constituent representations, six in the current simulation. Thus, the semantic meaning of a word builds up gradually through time. A subsystem accesses the meaning of a word by looking across several of its constituent representations. We assume the meaning of a word emerges from the first few representations. It is important to point out that we are not talking about letter by letter reading here, but the whole word forming an image that builds up gradually through time.

In relation to the time course associated with the extraction of meaning, we assume that three constituent time slots, amounting to 60 ms of presentation, are required for the extraction of useful meaning. Such an estimate is consistent with early research showing that the number of items reportable from a visual array rises rapidly with exposures up to 50 ms, and plateaus thereafter (Mackworth, 1963). A 60-ms integration time also equates closely with a finding recently reported by Grill-Spector and Kanwisher (2005). They show not only that detection increases with exposure durations up to 68 ms, but also that at exactly the time point that the simple detection of an object approximates maximum performance, the ability to report its category also approximates its maximal level, indicating that accurate generic semantic information can indeed be available on the same time scale as simple detection. Thus, the glance–look model specifies not just how attention relates to meaning and salience, but also the time course of meaning formation.

HOW THE MODEL BLINKS

The general idea that attention deployment is governed by an initial glance at generic meaning and then optionally pursued by more detailed scrutiny of referentially specific propositional meaning, is captured here by two stages of buffering with distributed control. The subsystem that is buffered decides when the buffer moves and where it moves to. In real life situations, stimuli do not arrive as rapidly as in AB experiments, so Implic and Prop will normally interpret the representation of the same item or event for an extended period. However, in laboratory situations, such as RSVP, items may fail to be implicationally processed as the buffer moves between subsystems. The buffer movement dynamic provides the underlying mechanism for the blink as follows.

- When in response to the key-distractor being found to be implicationally salient the buffer moves from Implic to Prop, salience assessment cannot be performed on a set of words (i.e., a portion of the RSVP stream) entering Implic following the key-distractor. Hence, when these implicationally un-interpreted words are passed to Prop, propositional meaning, which builds upon coherent detection of implicational meaning, cannot be accessed. If a target word falls within this window, it will not be detected as implicationally salient and thus will not be reported.
- There is normally lag-1 sparing in key-distractor AB experiments, i.e., a target word immediately following the key-distractor is likely to be reported. This arises in our model because buffer movement takes time, hence, the word immediately following the key-distractor may be implicationally interpreted before the buffer moves to Prop.
- When faced with an implicationally un-interpreted item, Prop is no longer able to assign salience and the buffer has to return to Implic to assess implicational meaning. Then, Implic assigns salience to its constituent representations again. After this, targets entering the system will be detected as implicationally and propositionally salient and thus will be reported. Hence, the blink recovers.

EXPERIMENT 1

In this section, we will demonstrate how key-distractors can capture attention through time, causing semantically prescribed targets to be missed. In addition, our model interfaces with statistical learning theories of meaning to demonstrate how attentional capture is modulated by the semantic salience of the eliciting key-distractor. In the course of this illustration, we will provide a concrete account of performance in the key-distractor AB paradigm where, as just discussed, attention is captured by meaning. The key principles that underlie this account are the division of the processing across two types of meaning, derived from the previously highlighted distinction made in the ICS architecture, between a generic form of meaning referred to as implicational meaning, and propositional meaning, which is referentially specific (Teasdale and Barnard, 1993).

METHODS

Modeling task set by semantic similarity

Barnard et al. (2004) used LSA (Landauer and Dumais, 1997; Landauer et al., 1998, 2007) to assess similarities between key-distractors and job targets. LSA is a statistical learning method, which inductively uses the co-occurrence of words in texts and principal component analysis to build a (compact) multidimensional representation of word meaning. In particular, an "objective" measure of the semantic distance between a pair of words or between a word and a pool of words can be extracted from LSA. The critical finding of Barnard et al. was an informal observation that the depth of the blink induced by a key-distractor was modulated by its proximity to the target category, i.e., its semantic salience. We seek here to build from this informal understanding to reproduce in a formal model the key effect of modulation of attentional capture by semantic salience and to explain that effect, again formally, using LSA.

Our model also reflects gradations in semantic salience. We assume that the human cognitive system has a space of semantic similarity available to it comparable to that derived from LSA. The link between principal component analysis (which is at the heart of LSA) and Hebbian learning (O'Reilly and Munakata, 2000), which remains the most biologically plausible learning algorithm, provides support for this hypothesis. Accordingly, we have characterized the assessment of semantic salience in terms of LSA.

To encapsulate the target category in LSA space, we identified five pools of words, for respectively, human relatedness, occupation relatedness, payment relatedness, household relatedness, and nature relatedness. Then, we calculated the center of each pool in

LSA space. We reasoned that the target category could be identified relative to these five semantic meanings (i.e., pool centers); see Appendix. This process can be seen as part of a more general categorization mechanism that works on all LSA dimensions. In the context of this experiment, it focuses on the five most strongly related components, as discussed above.

Next, we needed to determine the significance that the human system placed on proximity to each of these five meanings when making target category judgments. To do this, we trained a two-layer neural network to make what amounts to a "targetness" judgment from LSA distances (i.e., cosines) to each of the five meanings, cf. **Figure 4**. Specifically, we trained a single response node using the Delta rule (O'Reilly and Munakata, 2000) to classify targets from non-targets. The words used in Barnard et al.'s (2004) experiment were used as the training patterns. During training, for each target word, the five corresponding LSA distances were paired with an output (i.e., response node activation) of one, while the LSA distances for non-target words were paired with an output of zero. This analysis generated five weights: one for each LSA distance. These weights effectively characterize the significance that the target salience check ascribes to each of the five constituent meanings; thereby, skewing LSA space as required by implicational salience assessment, cf. Appendix.

Activation of our neural network response unit (denoted *m* in **Figure 4**) becomes the Implic salience assessment decision axis in our model. Thus, words that generate response unit activation above a prescribed threshold were interpreted as implicationally salient, while words generating activation below the threshold were interpreted as unsalient.

Parameter setting and multi-level modeling

Some parameters in our model are justified by neurophysiology, but others need to be set according to the human data observed in AB experiments. There are three sets of such parameters that are fitted using the behavioral curves: (1) salient assignment threshold at Implic; (2) the delay of buffer movement between Implic and Prop; and (3) the length of delay-lines in all subsystems. We fit these parameters using a *multi-level* approach (Su et al., 2007), which takes inspiration from the computer science notion of *refinement*. In the computational modeling of a particular cognitive phenomenon, the model development process can start with an abstract *black-box* analysis of the observable behavior arising from the phenomenon. For example, with the modeling of psychological phenomena, this may amount to a characterization of the pattern of stimulus–response data using a minimum of assumptions. Then, from this solid foundation, one could develop increasingly refined and concrete models, in a progression toward *white-box* models. Importantly though, this approach enables cross abstraction level validation, showing, for example, that the white-box model is correctly related to the black-box model, i.e., in computer science terms, is related by refinement (Bowman and Gomez, 2006).

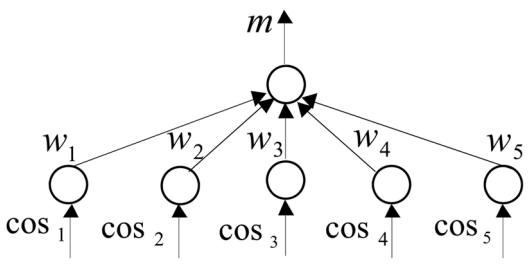

FIGURE 4 | A neural network that integrates five LSA cosines to classify targets from non-targets.

A central, and as yet largely unresolved, research question is how to gain the benefit of contained well-founded modeling in the context of structurally detailed descriptions on the one hand, and on the other hand, avoid the "*irrelevant specification problem*" (Newell, 1990). This problem is classically viewed as arising when a large number of assumptions are made during model implementation, such that it is unclear what assumptions correspond to known cognitive behavior. We provide an initial step in the direction of developing a progressive multi-level approach to cognitive modeling. In particular, all levels of our models occupy just part of the full trajectory of cognitive models (in particular, we regard the glance–look model as the white-box model, and will not consider the neural level). In addition, the relationships between levels that we highlight will be rather specific and will not be supported by formal reasoning. More complete instantiations of our methodological proposal awaits further computational theoretic work on how to relate the sorts of models developed in the cognitive modeling setting. The actual parameter setting and descriptions of black- to gray- and then to white-box modeling we explained in Appendix in order to make the main body more accessible for readers.

RESULTS

Simulation of the glance–look model has shown that high salience key-distractors were much more likely to generate above-threshold response unit activation than low salience items. This in turn ensured that HS items were more often judged to be implicationally salient, which ensured that the buffer moved from Implic to Prop more often for HS items. Since the blink deficit is caused by such buffer movement, targets following HS items were more likely to be missed, cf. **Figure 5**.

DISCUSSION

We have shown that in the context of Barnard et al.'s (2004) key-distractor blink task, attention is captured when a key-distractor is interpreted as implicationally salient and thus, in error, task relevant. This then causes attention (i.e., the buffer) to be redeployed to the propositional subsystem, in order to enable a more detailed (propositional) assessment of the salience of the key-distractor. Critically, this redeployment of attention leaves a temporal window in which implicational salience is not assessed. During this redeployment, the system is vulnerable to missing even highly salient items. It is through this mechanism that the model blinks. This instantiates the idea (Barnard and Bowman, 2003; Barnard et al., 2004) that semantic blink effects are mediated by first glancing at a form of meaning that supports a sense of relevance in the task context and then moving to a more stringent evaluation of the extent to which word meaning matches the referentially specific properties required by the task.

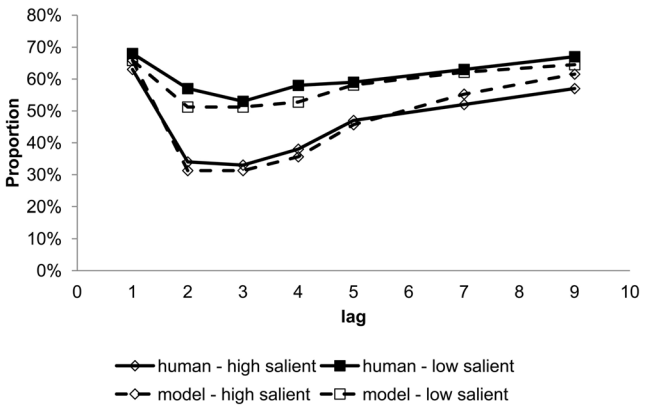

FIGURE 5 | Target report accuracy by serial position comparing human data (Barnard et al., 2004) and model simulation for both high and low salient key-distractors. Lag indicates the number of items intervening between the key-distractor and the target.

In addition, we have provided a further case study for the utility of LSA as a means of modeling word meanings. Although the LSA space did not furnish a direct route to distinguishing high and low salience key-distractors, a weighted sum of five attributes did model generic meaning and we established empirically that this could form a basis for discriminating our key-distractors. The effectiveness of LSA depends on the appropriateness of the corpora used to derive the semantic space employed (Landauer et al., 1998). Nevertheless, we have shown that measures of semantic distance derived from LSA, which we take as a useful approximation of implicational salience assessment, can reproduce the key-distractor blink and semantic modulations of blink depth.

This model has its origins in work on emotional disorders (e.g., see Teasdale, 1999 for an extended discussion). In this respect, the broader mode of processing meaning bears some resemblance to recent suggestions from the experimental literature that emotion, body-state, and task manipulations can modulate blink effects (Barnard et al., 2005; Olivers and Nieuwenhuis, 2005, 2006; Arend et al., 2006; Arnell et al., 2007). We next move to model these effects.

EXPERIMENT 2

There are now several reports of specific effects of affective variables during the AB (e.g., Arnell et al., 2007 and Barnard et al., 2005). In particular, (Anderson, 2005) has shown that the blink is markedly attenuated when the second target is an aversive word. These findings are consistent with the perspective that emotions have a major influence on salience sensitive control. Accordingly, the interaction between emotional salience and temporal attention is being actively investigated in the AB literature. Consequently, we have incorporated emotional salience into the glance–look model. We have particularly focused on modeling the effect of emotional stimuli in two data sets collected using Barnard's key-distractor AB tasks. Similarly to the previous section, in these tasks, participants search an RSVP stream of words for an item in a target category. Again, performance on the target identification task is investigated as a function of the lag that the target item appears relative to a key-distractor. However, rather than being semantically salient, in these tasks, the key-distractor is emotionally charged.

METHODS

Modeling intrinsic salience due to affect

Arnell et al. (2007) have reported a characteristic blink effect when the key-distractors are emotionally charged words. Specifically, sexual words captured attention more significantly than mildly threatening, anxiety-related, or other emotional words. A deeper blink occurs in the sexual key-distractor condition than control conditions, see **Figure 7**. In addition, sexual words were better encoded as reflected by heightened performance in a subsequent memory test. This effect suggests that stimulus emotionality is a cue of intrinsic salience used by cognitive control. In particular, perception of high priority emotionally salient stimuli can override the task filter, in this case a specific set of target words defined by semantic category. There is also neurobiological evidence that supports the modulation of cognitive control by affect. For instance, patients with damage to specific emotional centers in the brain (unilateral damage to the left Amygdala) show no differential effect to aversive compared to emotionally neutral words (Anderson and Phelps, 2001). The implication is that this region plays a central role in the pathway by which affect-driven salience is assessed.

Arnell et al. (2007) has argued that it is the arousal, rather than valence, of these key-distractors that correlates with the reduced accuracy in target identification. In particular, the participants' accuracy of reporting the targets reduced significantly when key-distractors were taboo words. In order, then, to model attentional capture by emotional words, in particular "tabooness," in the context of LSA, we have identified 10 reference words that we view as representing a schema of a taboo–sexual condition; see Appendix. The choice of these 10 reference words is inspired by Jay (2009), which has addressed the utility and ubiquity of taboo words in the context of how they carry emotional information and what makes these words taboo. Our reference words do not occur as key-distractors in Arnell's experiments, but most of them (or their synonyms) are used in Jay's article for defining what taboo words are.

We calculated the semantic distance in LSA space between each of Arnell et al.'s (2007) key-distractors (from both the arousal and control conditions) and the pool of (reference) taboo defining words. The high arousal key-distractors had the largest similarity to these reference words, while control condition words showed minimal similarity to the reference pool; see Appendix. Our glance–look model can thus be extended to describe both semantic and emotional salience by computing the semantic similarity of each word to the target set as well as the taboo defining references. We assume that if any of these dimensions has reached a certain threshold, the implicational subsystem will regard the item as salient, and trigger the buffer to move to the propositional subsystem. As a result of this, both task relevant and intrinsic emotionally salient key-distractors can cause the system to blink.

Modeling intrusion of body-state markers

Barnard et al. (2005) has shown another way in which emotion could interfere with cognitive control. The main finding in this

study was that although the threatening key-distractors do not capture attention of unselected participants as they did in Arnell et al. (2007), they can capture attention with participants that were both high state and high trait anxious. In addition, consistent with the notion that this phenomenon is not identical to that identified by Arnell et al. (2007) the blink exhibited in Barnard et al. (2005) was specifically late and short, cf. **Figure 8** solid lines (where it is only at lag-4 that the high state and high trait anxious group differs significantly from the low sate anxious group). State anxiety is defined as transitory anxiety experienced at a particular time (often in the recent past or during the experiment). On the other hand, trait anxiety refers to a more general and long-term experience of anxiety; and it often reflects individual differences in reaction to threat (Spielberger, 1972, 1983).

Consistent with the ICS framework, this attentional capture by threat was modeled through the addition of a body-state subsystem, cf. **Figure 6**. It is assumed that the body-state subsystem responds to the glance at meaning, i.e., to implicational meaning. A bodily evaluation of salience is then fed-back to Implic; thereby, enriching the representation. In effect, the body-state feeds back information in the form of a "somatic marker" (Damasio, 1994; Bechara et al., 2000), which, in the context of the task being considered here, would be a threat marker. Another assumption is that the body-state representation is built upon the implicational meaning with a delay, so the blink onset is positively shifted as shown in **Figure 8**. Huang et al. (2008) have demonstrated in a series of AB experiments with different task instructions, that emotional key-distractors only generate an AB if the task involves semantic judgments rather than more surface tasks, such as those based on visual features, rhyming patterns, or phonological cues. This evidence also suggests that emotional and body-state representations are activated in the processing of (implicational) meaning.

In addition, it is assumed that high anxiety levels (both state and trait) are required before this body-state feedback has sufficient strength to have a major effect on implicational salience. Such difference in sensitivity to affect between high and low anxious individuals is supported by neurophysiological findings (fMRI, Bishop et al., 2004). In their experiment, both high and low anxious people showed increased amygdala activation for fearful faces vs. neutral faces when the faces were attended. However, when the faces were unattended, only high anxious participants showed increased amygdala activation for fearful faces vs. neutral faces. This suggests that for high state and high trait anxious individuals, threatening key-distractors are implicationally interpreted as highly salient when body-state feedback enhances their implicational representation. This enhanced representation precipitates a detailed "look" at the meaning of these items by initiating a buffer move to Prop. Any new items, in particular targets, that arrive at Implic while the buffer is at Prop will be missed. However, since threatening key-distractors are not semantically salient, the buffer will move swiftly back to Implic and the blink is restricted in its length and depth. (In Arnell et al.'s (2007) experiment, although taboo words are not semantically salient, they are emotionally exceptionally salient, so the buffer does not move back to Implic faster than normal).

RESULTS

Without changing the model parameters set in Experiment 1, but by simply introducing the additional dimension of emotion salience, simulations of the glance–look model indeed reproduced the emotional AB phenomena in Experiment 2. For example, as shown in **Figure 7** dashed lines, the model reproduced a deeper blink for arousal (sexual-related) but not neutral key-distractors. In addition, as shown in **Figure 8** dashed lines, the model generated a characteristic late and short blink at around lag-4, which is uniquely observed in Barnard et al. (2005). In summary, the glance–look model has effectively broadened classical notions of task filtering that, in a wider sense, should embrace affect and body-state.

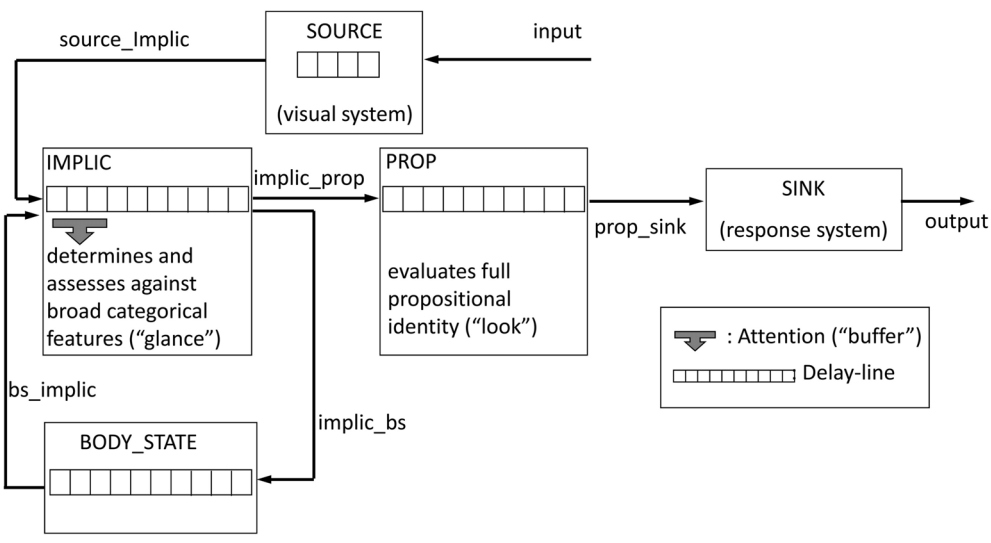

FIGURE 6 | The glance–look model extended with body-state subsystem.

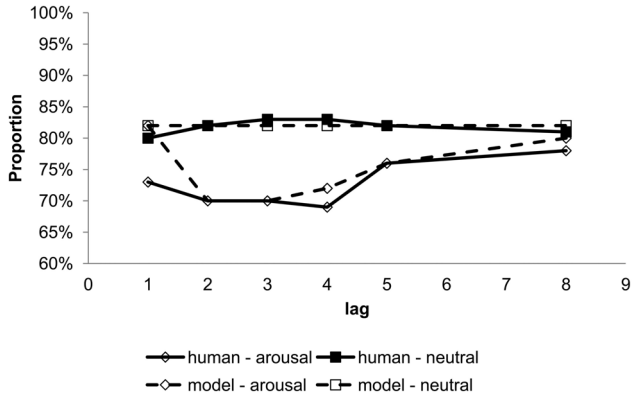

FIGURE 7 | Target report accuracy by serial position comparing human data (Arnell et al., 2007) and model simulation for arousal sexual-related and neutral key-distractors.

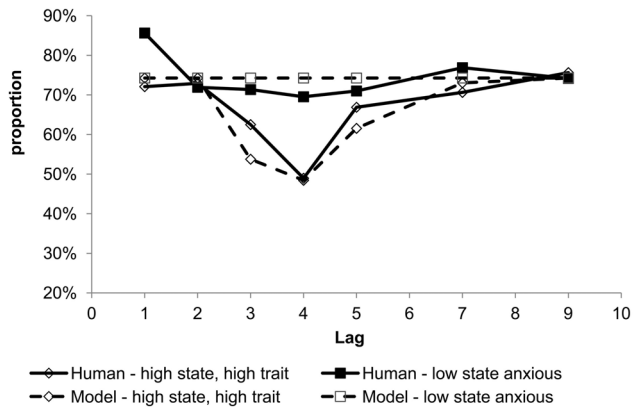

FIGURE 8 | Target report accuracy by serial position comparing human data (Barnard et al., 2005) and model simulations for high state and high trait anxious and low state anxious.

DISCUSSION

In this section, we have modeled emotional effects on the AB using the glance–look model. By reproducing two key experimental findings, we have proposed two distinct mechanisms by which affect may play a critical role in guiding temporal attention. The first mechanism takes a *direct path*, by which affect directly increases the salience of the stimuli to such a degree that control is redeployed from monitoring generic meaning to the more specific referential meaning. Hence, we see a somewhat classic blink curve as observed by Arnell et al. (2007). The second mechanism is via the *body-state feedback loop*, by which affect can influence cognitive control as seen in Barnard et al. (2005). These two mechanisms may occur simultaneously, but body-state feedback often has a delay of several hundred milliseconds. Hence, we argue that in Arnell et al. (2007), the body-state feedback arrives too late to affect the shifting of attention because the buffer has already committed to move from Implic to Prop. Thus, the effect of body-state feedback on cognitive control may only become important when the salience of emotional stimuli is not sufficient to trigger the buffer to move from Implic to Prop. We argue that this is the case in Barnard et al. (2005). Indeed, in high anxious individuals with hyperactive body-state subsystems, body-state feedback (although arriving with a delay) may still enhance the salience of the item sufficiently to trigger the buffer to move. Indeed, the glance–look model has reproduced the delayed blink curve in Barnard et al. (2005).

Although the focus of our modeling is the time course of blink onset, which is the key to distinguishing these two mechanisms of affective control, i.e., via the direct path (Arnell et al., 2007) or body-state feedback loop (Barnard et al., 2005), we have also noticed other differences between these two types of emotional effects in the AB. First, lag-1 sparing is markedly weaker in the taboo key-distractor condition compared to its control conditions, cf. Arnell et al. (2007) and classic semantic AB blink curves. Although not formally modeled in this paper, we argue that this may result from a faster reconfiguration when stimuli are exceptionally salient. In particular, the very presence of extreme taboo words and knowledge that they have a high likelihood of recurring in this task context may bias a rapid shift of attention toward them. The glance–look model predicts such reduced lag-1 sparing when the buffer movement delay from Implic to Prop is sampled from a negatively shifted distribution, i.e., the buffer moves faster from Implic to Prop. Such a shortened delay when switching attentional focus may leave a shorter window of time for the lag-1 item to be implicationally processed. This would lower the probability of reporting targets that immediately follow the key-distractor. There are plausible neurobiological mechanisms that may support such rapid orientation toward threatening stimuli. One of the most prominent theories is the fast sub-cortical route for emotion, proposed by LeDoux et al. (1986), LeDoux (1996). They showed, in a fear conditioning paradigm, that there exists a direct route between thalamus and amygdale, bypassing the cortex.

Second, the blink is shorter in Barnard et al. (2005) than in Arnell et al. (2007). As previously discussed, this is modeled by a reduced buffer movement delay from Prop to Implic when Prop is processing mildly threatening words. However, it is unlikely to be the case when participants are processing extremely salient taboo words. They not only rapidly capture our attention, but also engage extensively before releasing the control and allowing the buffer to return to Implic. Thus, the glance–look model naturally mimics our subjective experience of taboo words.

In summary, the glance–look model supports a broader perspective on cognitive and affective control. In particular, it has moved toward a schematic and embodied account by introducing a gist-based implicational subsystem, which is sensitive to body-state feedback. In this sense, it broadens classical theories of cognitive control. When moving to such a perspective of cognitive control, some commonly considered distinctions become somewhat undermined. For example, the difference between endogenous (top-down) salience and exogenous (bottom-up) salience is not as clear-cut as commonly considered. That is, the distinction between a stimulus that is viewed as salient on the basis of top-down influences (e.g., the ink color red when color-naming in a Stroop task) and on the basis of bottom-up influences (e.g., a threatening word when color-naming during an emotional Stroop task) is really

a distinction between salience prescribed by the experimenter (endogenous) and salience prescribed by the participant's longer-term goals (exogenous). Thus, both endogenous and exogenous reflect biases on stimulus processing due to organism goals, and, in that sense, are both top-down, it is simply that in the endogenous case, goals are short-term and artificially enforced, while in the exogenous case, goals are long-term and intrinsic to the organism.

EXPERIMENT 3

Recent findings also suggest beneficial effects of focusing on schematic and gist-based implicational meaning when the attentional system is under high cognitive load. Two important findings support this view. One is the fringe awareness phenomenon shown in the key-distractor AB, cf. (Barnard et al., 2004), where some level of awareness is preserved during the AB while full referential identity is apparently absent. There is also evidence for a counterintuitive pattern, in which distracting participants can in fact reduce blink depth (Olivers and Nieuwenhuis, 2005, 2006; Arend et al., 2006). Thus, in this context, reducing attentional focus seems to improve awareness. It has been argue that such distraction may counteract an overinvestment of attention. To elaborate further, in a typical laboratory setting, participants are encouraged to recall as accurately as possible. As previously argued by several authors (Olivers and Nieuwenhuis, 2005, 2006; Arend et al., 2006), this could well, in a very broad informal sense, result in more "investment of attention" than is strictly necessary to accomplish item report. Hence, task manipulations and emotional states (e.g., by using music, positive affect, or dynamic visual patterns) can attenuate blink effects (i.e., enhance awareness of the second target) by, it is argued, encouraging a more distributed state of attention. This section shows how the glance–look model can provide a more formal information processing account for the fringe awareness and overinvestment findings in the AB. In addition, we argue for a re-evaluation of conventional theories of cognitive function based on interactions between attention, emotion, and consciousness.

METHODS

Modeling fringe awareness in the attentional blink

As previously discussed, Barnard et al. (2004) used three types of response in their AB experiments: (1) report of the target identity, (2) "No job seen," and (3) "Yes, I saw a job, but could not report its identity." These responses reflect different degrees of awareness of target presence. The glance–look model suggests that the salience assignment of a target word can also be processed to three different degrees, cf. **Table 1**. We argue that different degrees of processing can potentially result in different types of response.

As shown in the first row of **Table 1**, targets that are found salient both at Prop and Implic can be reported correctly with their identity at the end of the sequence. As previously discussed, a subsystem needs to evaluate at least three constituent representations in order to access the salience of a word. In the second situation in **Table 1**, some items may be implicationally un-interpreted because Implic is not buffered when they are passed through the implicational delay-line. The model assumes that implicationally unprocessed items will not be evaluated for meaning at the propositional level.

Table 1 | Different degrees of processing and their corresponding responses from the model.

Implicational subsystem	Propositional subsystem	Responses
Fully processed	Fully processed	Correct report of identity
Unprocessed	Unprocessed	"No" responses
Partially processed	Any level of processing	"Yes" responses

Our model predicts that this will result in a situation where subjects are completely unaware of the presence of an incoming item, and will respond "no" at the end of the trial. Finally, as shown by the third situation in **Table 1**, some targets can be partially processed by Implic, but only for less than three constituent representations. Hence, we argue that when executive processes are reconfiguring, participants could be only fringe aware of salient stimuli. Although lacking the full referential identity, they are capable of reacting to at least some categorical information. This further suggests that gist-based implicational meaning may contribute to awareness of stimuli without extended propositional processing.

Modeling attenuation effects in the attentional blink

Given the existence of fringe awareness based on semantic "gist," the next question is whether schematic (implicational) representations alone are sufficient to identify items in RSVP streams when the capacity of the system is being pushed toward its limit, i.e., when there are distractions. Here, we model attenuation effects using the glance–look model and, thereby, provide a computational account of the overinvestment theory. In particular, overinvestment may reflect (functionally unnecessary) extended processing in our second propositional stage, delaying attention's return to a state in which implicational representations are evaluated. The implicational mode of attending to meaning has a broader focus on generic meaning, which we argue incorporates affect, and derivatives of multimodal or lower order inputs, such as music. When participants are exposed to dynamic patterns, being visual, musical, or internally generated, while performing the central AB task, there would be more changes in input to Implic. With our model of distributed control, these may well encourage the implicational mode of attending to meaning, perhaps "calling" the buffer back from Prop to Implic, and, thus, supporting more distributed awareness of this type of generic meaning.

The degree of, distraction-induced, attenuation reported in Arend et al. (2006), Olivers and Nieuwenhuis (2005, 2006) should, though, reflect two factors: the degree to which the ancillary task has direct consequences for the representation of generic (implicational) meaning and the extent to which the reporting of an item requires extended evaluation of propositional meanings. Crucially, when attenuation effects are observed, the paradigm often involves reporting letters in a background stream of digits (Olivers and Nieuwenhuis, 2005, 2006; Arend et al., 2006). Letters are drawn from a small and highly familiar set, and hence, in the limit, may require only the briefest "look" at a propositional representation, or in most cases, only a "glance" at the implicational representation, to support correct report. So, we assume in the model that the

buffer moves from Implic to Prop with a reduced probability when participants are distracted. And, we also assume that the majority of items can be reported when they have only been implicational processed.

RESULTS

As seen in **Figure 9A**, the glance–look model has reproduced the "No" response, which often occurs at serial position 3 and 4. And, these lags are the deepest points of the blink. Using the same parameter setting, our model generated partial processing at serial position 2, because it is the moment when the buffer is shifting from Implic to Prop. At this serial position, human participants often respond with "yes," confirming that they are aware of the presence of the target but unable to identify it, cf. **Figure 9B**. Hence, the glance–look model naturally captures the fringe awareness. Finally, the glance–look model also reproduced the attenuation effects, cf. **Figure 10**. Due to limitation of space, we only show the simulation result for Experiment 1 of Olivers and Nieuwenhuis (2005), in which the blink depth reduced when participants had additional tasks.

DISCUSSION

The glance–look model has shown that lack of awareness can be accounted for by the allocation of attention to different levels of meaning in a system where there is only distributed control of processing activity. Just as the focus of our attention may shift among entities in our visual and auditory scenery under the guidance of salient change, shifts in attention to different entities in our semantic scenery can lead to RSVP targets being either, (1) correctly identified; (2) "noticed" with fringe awareness of presence; or (3) overlooked. Salience states at each of two levels of meaning allow these three response patterns to be captured. Although the proposal, like that of Chun and Potter (1995), relies on two stages, both of our stages are semantic in nature and the temporal dynamic involves controlled changes in the focus of attention, rather than classic capacity or resource limitations. The idea of monitoring a generic form of meaning for implicational salience, the level at which affect is represented in the model, and switching only when required to evaluate propositional meaning, represent two "modes" of attending to meaning. The former mode has a broader focus on generic meaning (i.e., the "gist") and the latter a more evaluative focus on specific meanings, which can be verbally reported. This is similar to the distinction in the literature between "phenomenal" and "access" awareness (Lamme, 2003). Furthermore, the broader mode of processing meaning bears some resemblance to recent suggestions that task manipulations can attenuate blink effects, by encouraging a more distributed state of awareness, which would arise at our implicational level. In particular, music, positive affect, and dynamic visual patterns may counteract on overinvestment of attention (Olivers and Nieuwenhuis, 2005, 2006; Arend et al., 2006) and produce a fleeting conscious percept (Crick and Koch, 2003).

In summary, consciousness is modeled as an emergent property from the interaction among three subsystems: implicational, propositional, and body-state. In particular, we differentiate two types of consciousness. One is akin to full "access" awareness, i.e., conscious content can be verbally reported, and is supported by both implicational and propositional processing. In other words, it is a result of a detailed "look" and more extensive mental processing. The other is akin to "phenomenal" (or fringe) awareness. We argue that the latter is a result of attending to the implicational

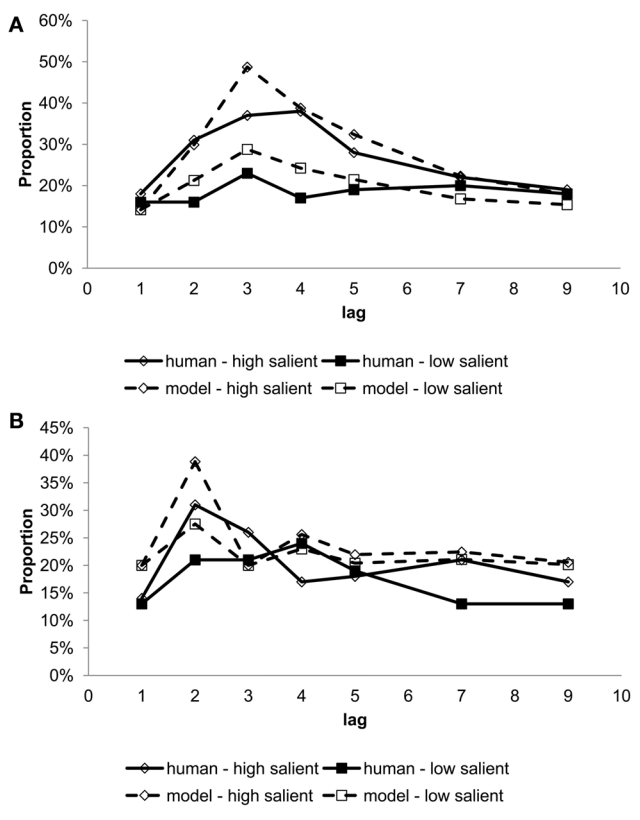

FIGURE 9 | Proportion of (A) "No" response and (B) "Yes" response (i.e., reflecting partial awareness) by serial position comparing human data (Barnard et al., 2004) and model simulation for high and low salient key-distractors.

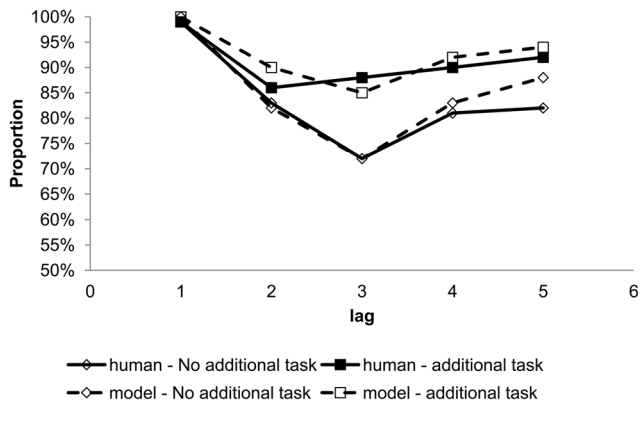

FIGURE 10 | Target report accuracy by serial position comparing human data (Olivers and Nieuwenhuis, 2006) and model simulation for with and without distraction, i.e., an additional task.

level or "glance." It is also notable that the implicational level is holistic, abstract and schematic, and is where multimodal inputs are integrated, and affect is represented and experienced (Barnard, 1999).

In addition, the glance–look model makes several predictions on the relationship between these two modes of consciousness. First, fringe awareness provides a basis for a more complete state of consciousness. Second, comparing to full access awareness, phenomenal, or fringe awareness is directly affected by emotional, multimodal, body-state, and lower order inputs. However, once propositional level information has been attended, a conscious percept is much less likely to be interrupted. The validation of these predictions awaits further experimental work.

The model however also predicts that attenuation should be less pronounced either with secondary tasks whose content does not directly influence the level of generic (implicational) meaning or, as with semantic blink effects, where a fuller evaluation of propositional meanings is required. Should such effects be found, it would provide an encouraging convergence between basic laboratory tasks and the literature on attention to meaning and affect in emotional disorders, using a non-computationally specified version of our current proposal (Teasdale, 1999).

GENERAL CONCLUSION

We started this paper with the observation that, as classically formulated and empirically studied, cognitive control has been rather narrowly delineated. In particular, studies have typically focused exclusively on the cognitive and on experimental materials that afford a precise discrete demarcation into task relevant and non-task relevant. One might, indeed, describe this as an all-or-none circumscription of task-focus: stimuli are either completely goal relevant or completely goal irrelevant. This problem is being partially addressed by a body of emerging cognitive control research that incorporates the affect dimension, the journal special topic that this paper is presented under being a case in point. Indeed, there is now a good deal of evidence that, even when task irrelevant, affect laden stimuli bias attentional focus and are prioritized (Anderson, 2005; Barnard et al., 2005; Arnell et al., 2007).

The other pillar of our argument to broaden the notion of goal-relevance, and which certainly remains underexplored, is the role of meaning representations (in their broadest sense) in cognitive control. Firstly, the space of meaning representations that the brain carries is likely to be inherently continuous and graded. This certainly is, for example, the perspective arising from statistical learning techniques, both in their supervised (O'Reilly and Munakata, 2000) and unsupervised (Landauer and Dumais, 1997; Landauer et al., 1998, 2007) formulations. Thus, it is just difficult for our brains to perfectly delineate one meaning category from another. The goal specifications, that we employ, and which are surely substantially driven by meaning, are likely to be graded in nature, rather than discrete.

In this context, we have proposed a model of central executive function based upon two levels of meaning and, correspondingly, two levels of filtering. The first of these, the glance, extracts a schematic, implicational, representation of meaning; and it is at this level that affect is encompassed. The second, the look, assesses a referentially bound propositional perspective on meaning. Using this framework, we were able to integrate graded representations of meaning, based upon LSA, with emotional and body-state influences. We illustrated this model in the context of the key-distractor AB task. We were able to model a spectrum of key-distractor AB phenomena, including, modulation of blink depth by key-distractor semantic salience, deep blink profiles with taboo key-distractors, smaller, and later concern associated blinks with milder affective key-distractors, fringe awareness patterns, and blink attenuation in the presence of distraction.

In the current trend of cognitive neuroscience, functional MRI is the primary method that maps cognitive functions to underlying neurobiology. However, in the context of AB, the poor temporal resolution of BOLD functional imaging is not sensitive to the very rapid switch between Implic and Prop in our glance–look model. Hence, one must be speculative when relating subsystems in our model to brain areas. Nonetheless, we argue that mechanisms implemented in our model fit within existing neuroscientific findings of cognitive and affective networks in human brain. For example, it is argued that neurons in the dorsolateral prefrontal cortex (DLPFC) encode task set (Miller and Cohen, 2001). DLPFC and parts of the multiple-demand (MD) system (Duncan, 2010) could be correlated with Prop, where the task filter derived from experimental instruction is implemented. It is also argued that animals have the ability to extract threatening information from the environment, and such ability is hard-wired through evolution. Threatening stimuli act as cues of potential danger and may trigger "flight or fight" responses, so it is important for all animals, including humans. Hence, threatening information needs to be rapidly extracted directly from sensory inputs, likely via the neural pathway from the sensory thalamus to the amygdala (LeDoux, 1996). This is consistent with our glance–look model that emotion induced representations are extracted directly from sensory inputs at Implic, cf. modeling intrinsic salience due to emotion in Section "Experiment 2." In addition, the body representations encoded in our body-state subsystem are likely to correlate with somatosensory cortex, insula, and hypothalamus (Bechara et al., 2005).

Implic as the central subsystem for the integration of cognition and emotion plays a critical role in the glance–look model account of cognitive and affective control theory. We believe a number of candidate regions in the brain are well situated to perform this function. Firstly, the amygdala is not only highly connected to both cortical and sub-cortical systems, but also participates in both cognitive processing, such as attention orientation, and emotional processing (Heller and Nitschke, 1997). Secondly, it has been shown that the orbitofrontal cortex (OFC), the ventromedial prefrontal cortex (VMPFC), and the anterior cingulated cortex (ACC) are also likely to be part of this integration network (Pessoa, 2008). (Also, see Taylor and Fragopanagos, 2005 for a review of neural correlates of attention and emotion systems, and for an alternative computational account for the time course of attentional control network. It is likely that the glance–look model also draws on resource in other areas of the brain that are sensitive to functional MRI. However, to get temporal information in the time frame of RSVP, time resolved techniques, such as Magnetoencephalography is highly desirable).

With respect to the interaction between emotion and cognition, the general effect of emotion in cognitive control has been experimentally studied, but related computational theories are not fully spelled out in the literature. Some successful computational models of emotion rely on statistical learning algorithms, e.g., reinforcement learning (Montague et al., 1996; Schultz et al., 1997). Others argue for competition between emotion and cognitive processing (Mathews et al., 1997; Mathews and Mackintosh, 1998; Taylor and Fragopanagos, 2005; Wyble et al., 2008). Our glance–look model fits within the latter bracket and arguing for competitive interaction between cognition and emotion, i.e., emotional salience can attract attention and impair (cognitive) task oriented processing. However, our model specifies how they compete in time, and predicts the complex temporal dynamics of cognitive and affective control. In addition, our model addresses the importance of processing at the implicational level.

From an evolutionary perspective, implicational meaning has its origins in the multimodal control of action (Barnard et al., 2007). The implicational subsystem, across the human line of decent, is where overt responses are selected on the basis of a blending and assessment of external (visual and auditory) and internal (body-state) stimuli. This is augmented by a propositional subsystem only in *Homo sapiens* and hence gives rise to a unique form of "cognitive" control. In some sense, most current theories of cognitive control lack a coherent behavioral grounding that goes beyond the fact that we are good at attending to stimuli that are relevant to what we are doing in intellectual tasks. However, the idea of implicational salience is that it can deal either with affective or non-affective salience. We argue that emotional blinks reflect "incidental" salience, which is partly due to processing of implicational meaning and partly due to a later body-state intervention. For emotional stimuli (specifically in the context of AB) there are only minimal requirements to do a propositional evaluation. So, in some sense, the essence of "cognitive" control is how much involvement of the propositional subsystem there is in evaluating representations in relation to task filters. Although task demands require a propositional representation, the glance–look model evolving here has the basic elements that enable affect, goals, body-state, and meaning to be addressed.

The glance–look model's simulation of blink attenuation with distraction does prompt an intriguing prediction. The model explains such attenuation in terms of an over emphasis on propositional level processing and, in that sense, fits with over investment theories of the AB (Taatgen et al., 2007). Importantly, this explanation is highly stimulus and task type dependent. That is, we are proposing that, in the context of the experimental laboratory, the cognitive system applies an extensive propositional analysis when it is, in some cases, not strictly necessary. This level of analysis is particularly redundant in the context of highly over learnt stimulus sets that are easily classified on the basis of surface features at the implicational system. However, this, at least partial, redundancy of propositional processing, would not obtain so significantly when semantic salience judgments are being made. Such salience would particularly obtain in the semantic key-distractor AB tasks considered in Section "Experiment 1" of this paper. Thus, we predict that addition of distraction manipulations, such as, background starfield (Arend et al., 2006), inducing positive affect (Olivers and Nieuwenhuis, 2005, 2006), and a peripheral task (Taatgen et al., 2007), would not attenuate the semantic key-distractor blink without, at the least, a cost to baseline target report performance. In other words, in semantic key-distractor tasks, emphasis on propositional processing is necessary and cannot be subverted without performance cost.

In addition to testing this pivotal prediction, it would clearly be beneficial to broaden the application of the glance–look model beyond the AB domain. In particular, it will be important to test the model in the context of Stroop and emotional Stroop experiments, particularly those focused on strategic, typically conflict-based, patterns of behavior (Botvinick et al., 2001; Wyble et al., 2008).

It is also important to note that the glance–look model is formulated within a broader architectural framework: the ICS architecture (Barnard, 1985). Such broader theories are not now very common in cognitive neuroscience theory, which has become focused on rather small scale neural network models of particular cognitive phenomena. Integration within architectural frameworks, though, enables higher level macro theoretic constraints to be brought to bear, such as the macroscopic information flows between component subsystems (Barnard, 2004). Such broader perspectives should enable the undoubtedly extensive and diverse constraints that impinge on central executive function, and the role affect and meaning play in that system, to be coherently brought to bear.

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APPENDIX

LATENT SEMANTIC ANALYSIS OF KEY-DISTRACTOR WORDS

Human reference words: human people mankind womankind someone mortal fellow sentient folk soul.

Occupation reference words: occupation profession job trade employment work business career livelihood vocation.

Payment reference words: payment fee remuneration recompense bribe salary honorarium income earnings wages.

Household reference words: ornament device utensil gadget tool possession decoration fitting fixture furnishing.

Nature reference words: archipelago backwoods beach biosphere brook channel cliff cloud cloudburst coastline crevasse crevice cyclone desert diamond drought sediment earthquake eruption estuary everglades fissure fjord floodplain frost geyser gorge grassland habitat hailstone headwind hillside hoarfrost iceberg icecap inlet island landscape lightning limestone meadow monsoon moonlight moraine mudflats outback outcrop pampas plains plateau puddle quartz rainbow raindrop rapids reef riverbank riverbed salt marsh sandstorm savannah seashore shoreline skyline snowflake straits stream sunshine swamp tempest tornado breeze tributary causeway waterfall wetlands whirlpool woodland.

Taboo reference words: vulgar offensive feces sex slang slur disgusting taboo blasphemous insulting.

FITTING THE PARAMETERS AND MODEL REFINEMENT

Black-box (Extensionalist) model: fitting the behavior curves with closed form equations

In the first step of the refinement trajectory, we regard the system as a black-box. That is, no assumptions are made about the internal structure of the system and there is no decomposition, at all, of the black-box into its constituent components. Thus, the point of reference for the modeler is the externally visible behavior, i.e., the semantic blink curves (Barnard et al., 2004). Such models are *extensionalist* in nature, and they simply characterize the data. A critical benefit of black-box cognitive modeling is that there are less degrees of freedom and fewer hidden assumptions, making data fitting and parameter setting both well-founded and, typically, computationally tractable. For example, if the system can be described in closed form, key parameters can be determined by solving a set of equations, if not, computational search methods can be applied.

An extensionalist model simply provides a systematic characterization of how data in a domain varies. This technique has been widely used in modeling response time distributions (Van Zandt, 2000; Cousineau et al., 2004) and, more recently, in modeling serial position curves of basic attentional blink tasks (Cousineau et al., 2006). In our context of exploring the key-distractor attentional blink task, the behavior curves have sharp blink onsets and shallow recoveries as shown in **Figure 5**. This shape matches an inverted Gamma distribution. Hence, we use the following equation to model blink curves.

$$y(x) = a - b \cdot G(x)$$

$$G(x) = \frac{\left(\frac{x-\mu}{\beta}\right)^{\alpha-1} e^{\left(-\frac{x-\mu}{\beta}\right)}}{\beta \cdot \int_0^\infty t^{\alpha-1} e^{-t}\, dt} \quad x \geq \mu;\ \ \alpha, \beta > 0$$

where G is a Gamma distribution, x is the serial position, a sets the baseline performance, and b describes the difference between the deepest point of the blink and the baseline. If b is set to 0, the function models the complete absence of the blink and baseline performance at all lags. So, we call b the depth parameter or the *capture constant*. In particular, b is related to key-distractor salience and thus characterizes the attentional capture by salience effect we are interested in. After fitting y to the human blink curves show in **Figure 5**, a is set to 0.67 for both high and low salient cases, but b is 1.8 for high salient key-distractors and 0.8 for low salient ones. It will become clear later that the capture constant b is related to implicational salience assignment threshold. Other parameters in the model ($\alpha = 2.2$, $\beta = 1.6$, $\mu = 0$) were fixed during data fitting, so, they do not affect the depth of the blink.

Gray-box model: adding assumptions of internal structure

In the black-box model, we used a Gamma distribution to describe the shape of blink curves. However, it does not describe the underlying mechanism of the cognitive system and what mental processes are likely to produce the AB phenomenon. In addition, the black-box model does not set all parameters in the glance–look model. We have introduced an intermediate step between black- and white-box models in order to incrementally add complexity. In particular, we set as many parameters as we can, at this stage, without defining the complex semantic space, which will be left to the final refinement. Hence, the intermediate gray-box model refines the black-box model, and reflects the three assumptions about internal structure explained in Section "Theory of the Glance-look Model" (i.e., sequential processing, two stages, and serial allocation of attention).

Salience assignment. In the gray-box model, the salience assignment threshold is indirectly modeled using a parameter called the *intrinsic probability of identification* (denoted P), which refers to the probability that an item will be seen if it is presented as a single target in an RSVP stream. Note, $P(\mathrm{X}, \mathrm{Y})$ is not the probability that both items X and Y are seen in an AB setting, but rather the probability that both would individually be seen in two separate single target events.

The intrinsic probability of detecting a target $P(\mathrm{T}) = 0.67$ is set by the baseline performance of humans (Barnard et al., 2004). The intrinsic probability of a background word being implicationally salient is assumed to be zero, since this sort of error is so rare as to be effectively zero. The intrinsic probability of detecting a high and low salient key-distractor is $P(\mathrm{HS})$ and $P(\mathrm{LS})$ respectively. According to the gray-box model, if the key-distractor is implicational salient and the buffer shifts to Prop, the lag-3 item (i.e., the deepest point in AB) will always be missed. So, the deepest point in the blink curve reflects the joint probability of missing the key-distractor and detecting the target, i.e., $P(\neg\mathrm{HS}, \mathrm{T})$ and $P(\neg\mathrm{LS}, \mathrm{T})$ in the high and low salient conditions respectively. According to the behavior curves (Barnard et al., 2004), we set $P(\neg\mathrm{HS}, \mathrm{T}) = 0.34$ and $P(\neg\mathrm{LS}, \mathrm{T}) = 0.54$, cf. **Figure A1**. Assuming Implic assesses targets and key-distractors independently, we have the following.

$$P(\mathrm{HS}) = 1 - P(\neg\mathrm{HS}) = 1 - P(\neg\mathrm{HS},\mathrm{T}) \,/\, P(\mathrm{T}) = 0.49$$

$$P(\mathrm{LS}) = 1 - P(\neg\mathrm{LS}) = 1 - P(\neg\mathrm{LS},\mathrm{T}) \,/\, P(\mathrm{T}) = 0.19$$

Latent semantic analysis cosines for high salient key-distractors of Barnard et al. (2004).

	Human	Occupation	Payment	Household	Nature	*m* Value
Heretic	0.12	0.01	0.02	0.05	0.02	0.087
Raconteur	0	0.02	0.01	0.06	0.06	0.031
Volunteer	0.2	0.34	0.16	0.1	0.04	0.438
Opponent	0.13	0.08	0.02	0.09	0.05	0.078
Patron	0.11	0.08	0.03	0.16	0.08	0.11
Coward	0.26	0.07	0.02	0.06	0.14	0.11
Pragmatist	0	0.17	0.03	0.02	0.02	0.102
Heathen	0.18	0.07	0	0.14	0.13	0.081
Scoundrel	0.17	0.06	0.02	0.14	0.09	0.11
Visitor	0.36	0.21	0.02	0.19	0.21	0.226
Grandson	0.21	0.14	0.03	0.02	0.18	0.094
Informant	0.03	0.17	0.03	0	0.07	0.087
Disciple	0.19	0.07	0.03	0.07	0.05	0.14
Witness	0.37	0.09	0.07	0.19	0.11	0.277
Voter	0.03	0.04	0.02	0.02	0.02	0.059
Widow	0.22	0.12	0.09	0.1	0.09	0.211
Vegetarian	0.16	0.03	0	0.08	0.06	0.085
Adversary	0.16	0.16	0.09	0.15	0.03	0.255
Thinker	0.25	0.13	0	0.07	0.04	0.177
Extrovert	0.14	0.1	0.01	0	0	0.118
Stranger	0.4	0.08	0.04	0.13	0.2	0.181
Visionary	0.21	0.07	0.02	0.11	0.1	0.122
Neighbor	0.17	0.08	0.02	0.11	0.07	0.127
Kinsman	0.17	0.07	0.06	0.12	0.11	0.127
Hunchback	0.11	0.04	0	0.04	0.01	0.081
Enthusiast	0.16	0.05	0	0.07	0.13	0.051
Accomplice	0.17	0.06	0.01	0.09	0.04	0.122
Sweetheart	0.11	0.08	0.05	0.05	0.05	0.115
Cousin	0.25	0.13	0.04	0.11	0.16	0.147
Egghead	0.08	0.04	0	0	0.03	0.059
Admirer	0.24	0.11	0	0.08	0.06	0.153
Spectator	0.16	0.12	0.01	0.12	0.07	0.134
Refugee	0.18	0.08	0.03	0.05	0.04	0.14
Hooligan	0.05	0.01	0	0.03	0.07	0.027
Shopper	0.08	0.06	0.08	0.09	0.03	0.13
Savior	0.18	0.04	0.01	0.09	0.07	0.099
Auntie	0.08	0	0	0.05	0.08	0.033
Pedestrian	0.14	0.03	0	0.03	0.03	0.081
Tourist	0.18	0.21	0.08	0.1	0.24	0.13
Husband	0.22	0.16	0.11	0.11	0.08	0.261

Hence, the intrinsic probability of identification sets the likelihood of an item passing the salient assignment threshold at Implic. Although humans perceive information in a noisy environment, so salient items may be missed, in the current model, to limit degrees of freedom and obtain a model that is as simple as possible, we assume that Prop is perfectly accurate in classifying targets from non-targets.

Buffer movement delay. In the glance–look model, the buffer can move in two directions, i.e., from Implic to Prop and vice versa. So, there are two buffer movement parameters *D1* and *D2*, which denote the delay of buffer movement from Implic to Prop and vice versa respectively. We also assume that salience assignment only takes place at the first three constituents in a subsystem's delay-line. When fitting the delay parameters, lag-1 sparing sets the lower bound of *D1*. That is, Implic determines that the buffer needs to move if the first three constituent representations is implicationally salient, as shown in **Figure A2A**. In order to report targets that immediately follow the key-distractor (i.e., the lag-1 case), Implic should process at least three constituent representations of the lag-1 item, as shown in **Figure A2B**. Hence, *D1* should be no less than 120 ms.

Furthermore, the onset of the blink sets the upper bound of *D1*. In order to miss lag-2 targets, *D1* must be larger than 220 ms.

Latent semantic analysis cosines for low salient key-distractors of Barnard et al. (2004).

	Human	Occupation	Payment	Household	Nature	*m* Value
Barometer	0.01	0.03	0	0.12	0.06	0.042
Button	0.19	0.07	0.04	0.3	0.08	0.186
Cabinet	0.09	0.12	0.02	0.15	0.06	0.122
Cellophane	0.08	0.04	0	0.14	0.07	0.065
Chandelier	0.1	0.03	0.02	0.07	0.09	0.059
Cosmetic	0.07	0.09	0	0.12	0	0.11
Cupboard	0.16	0.05	0.02	0.12	0.07	0.11
Curtain	0.21	0.06	0.03	0.09	0.14	0.099
Deodorant	0.04	0.05	0	0.17	0.03	0.078
Detergent	0.07	0.02	0.01	0.24	0.07	0.078
Dictionary	0.06	0.12	0	0.08	0.02	0.102
Freezer	0.04	0	0.02	0.07	0.05	0.046
Hammer	0.15	0.16	0	0.53	0.13	0.19
Handle	0.21	0.34	0.13	0.57	0.12	0.489
Ladder	0.19	0.29	0.06	0.09	0.18	0.202
Ladle	0.09	0.04	0.01	0.22	0.04	0.102
Lantern	0.14	0.1	0	0.08	0.17	0.056
Notepaper	0.09	0	0	0	0.05	0.037
Oven	0.07	0.05	0.01	0.14	0.06	0.074
Percolator	0.01	0.01	0.01	0	0.1	0.018
Picture	0.16	0.09	0.03	0.13	0.18	0.078
Pillow	0.14	0.02	0.01	0.09	0.09	0.068
Porcelain	0.09	0.09	0.02	0.16	0.1	0.087
Projector	0.02	0.02	0	0.17	0.07	0.046
Radiator	0.04	0.01	0	0.12	0.09	0.035
Settee	0.1	0	0.01	0.07	0.05	0.056
Souvenir	0.13	0.16	0.02	0.12	0.07	0.147
Spatula	0.02	0.04	0	0.17	0.09	0.033
Spotlight	0.17	0.15	0.05	0.11	0.07	0.181
Staircase	0.13	0.03	0.03	0.13	0.11	0.074
Tablecloth	0.11	0.05	0.03	0.1	0.06	0.094
Tankard	0.04	0	0.02	0.03	0.02	0.049
Television	0.19	0.13	0.03	0.11	0.06	0.177
Toothpaste	0.13	0.05	0	0.11	0.03	0.102
Trolley	0.08	0.06	0.01	0.04	0.05	0.068
Wireless	0.07	0.06	0.01	0.09	0.05	0.074

The threshold for m value (i.e., the activation level of the output unit) is 0.115.

This is the time when the first two constituent representations of the lag-2 item have just entered Implic, as shown in **Figure A3**. The phenomenon of fringe awareness indicates that lag-2 targets can be processed to some extent. So, some of the lag-2 constituent representations are likely to be implicationally processed before the buffer moves away. As a result of these constraints, *D1* is sampled from a narrow Gamma distribution peaks at 200 ms and bounded between 120 and 220 ms. The AB curves generally have a sharp onset and slow recovery, so *D2*, which is related to blink recovery, is more variable than *D1*, and is sampled from a wider Gamma distribution.

Delay-line length. Each subsystem has a local memory, which holds its representations before they are sent to other subsystems. We denote the length of the implicational and propositional delay-lines by *L1* and *L2* respectively, which are measured by the number of constituent representations they hold. We argue that the lower bound of *L1* is set by the fact that the buffer must move to Prop in time to process the item. Items cannot enter Prop immediately after being processed at Implic. (If this were the case, the buffer would have to move immediately to Prop in order to process it, but this would rule out lag-1 sparing as previously explained.) Rather, constituent representations progress along an intermediate portion of delay-line that functionally sits between the point of implicational salience assessment and the point of exit from Implic, and buffers (using the standard computer science meaning here) Implic to Prop communication. In other words, targets that are presented alone in an RSVP stream

Latent semantic analysis cosines to taboo reference words for key-distractors of Arnell et al. (2007).

Neutral	LSA	Positive	LSA	Negative	LSA	Taboo	LSA
Aisle	0.02	Beauty	0.08	Broken	0.07	Aids	0.03
Binder	0.08	Birthday	0.06	Decay	0.04	Ass	0.08
Blimp	0.03	Bouquet	0.05	Decline	0.11	Bastard	0.08
Butter	0	Champ	0.05	Dismay	0.09	Bitch	0.09
Card	0.02	Cheer	0.06	Dull	0.09	Clitoris	0.65
Chat	0.04	Flower	0.05	Faded	0.03	Cock	0.05
Chew	0.03	Friendly	0.13	Fail	0.15	Dildo	0.27
Dazzle	0.01	Fun	0.06	Feeble	0.1	Erotic	0.74
Desk	0.02	Glad	0.08	Guilt	0.32	Fire	0.02
Fish	0	Gold	0.04	Negative	0.12	Fuck	0.26
Gel	0	Happy	0.11	Poorly	0.23	Gun	0.02
Glove	0.03	Holiday	0.02	Punish	0.13	Incest	0.51
Guzzle	0.038	Joyful	0.04	Sad	0.02	Lesbians	0.37
Haggle	0.038	Leisure	0.09	Slave	0.03	Murder	0.07
Jacket	0.07	Prize	0.04	Slob	0.16	Naked	0.17
Justify	0.16	Sky	0.02	Suffer	0.22	Naughty	0.08
Loop	0.04	Smart	0.09	Tedious	0.1	Nipples	0.08
Planet	0.01	Smile	0.1	Thief	0.05	Orgasm	0.78
Ruffled	0.07	Sunny	0.01	Tired	0.03	Orgy	0
Spare	0.08	Sweet	0.06	Unhappy	0.14	Penis	0.76
Staple	0	Tender	0.08	Useless	0.06	Piss	0.03
Vote	0.08	Treasure	0.04	Weary	0.05	Rape	0.52
Wire	0.03	Vacation	0.06	Weep	0.04	Sexual	0.87
Zipper	0.01	Winner	0.04	Broken	0.07	Shit	0.09

All negative LSA values are replaced by zeros, and items that do not have LSA entries are replaced by group means. Threshold for taboo relatedness is 0.35.

can be potentially processed by both Implic and Prop, given the buffer moves with a delay. This is ensured by the following inequation:

$$D1 \leq (L1 + L - 3) \times 20\,\text{ms}$$

where $L = 6$ denotes the number of constituent representations in an RSVP item. The right hand side of the inequation is the delay between an item being detected as implicational salient and all its constituents entering Prop, as shown in **Figure A4**. (Note, the figure and its caption explain the above inequation in detail.) Given the values of *D1* calculated previously, we found the lower bound of *L1* is around 7.

The recovery of the blink sets the upper bound of *L1*. That is, Implic can only process the beginning of the lag-2 item as shown in **Figure A5A,B**. The decision is made for the buffer to move back from Prop to Implic, when Prop has detected three implicationally unprocessed constituent representations, which is the back end of the lag-2 item, as shown in **Figure A5C**. In general, the blink recovers after lag-5. Thus, the buffer should potentially return to Implic when the lag-5 item enters Implic or soon after that point in time, as shown in **Figure A5D**. (Note, the figure and its caption explain the above inequation in detail.) Hence, *L1* is constrained by the following inequation.

$$D2 \leq (4 \times L - L1 - 4) \times 20\,\text{ms}$$

Given the distribution of *D2*, we set the length of the Implic delay-line to 10, which is around the mean of the *L1* distribution. The length of the Prop delay-line *L2* is unconstrained in this model because it does not affect the shape of the blink. Thus, for simplicity, we assume that delay-line lengths are the same for all subsystems.

Relating to the (white-box) glance–look model

The glance–look model is an intensionalist account (i.e., it is structurally detailed). Importantly, it uses the delay-line length and buffer movement delay distributions inferred for the gray-box model. However, in the white-box model, the salience assignment threshold is explicitly modeled from word meaning represented in LSA space. The choice of the threshold for the response unit (as previously introduced) was directly constrained by the two higher level models, ensuring analogous parameter manipulations in all models. In particularly, the threshold value makes 52.5% of high salient and 22.2% of low salient key-distractors implicationally salient. In the glance–look model, the ratio between high and low salient key-distractors based on the response unit activation is $52.5/22.2 = 2.36$. In the gray-box model, the ratio between high and low salient key-distractors in the intrinsic probability of identification is $0.49/0.19 = 2.58$. In the extensionalist model, the ratio between high and low salient key-distractors in the capture constant is $1.8/0.8 = 2.25$. This similarity suggests that the activation of the response unit, the intrinsic probability of identification and the

capture constant model the same underlying cognitive mechanism consistently.

Discussion

In the general domain of theory development in cognitive psychology, there has always been something of a tension between theorists who operate at the level of box-and-arrow models and those that rely on complete, fully specified, simulations. Here we have provided evidence that classic box-and-arrow models can be implemented at a level appropriate to the constraints built into the model, and reproduce a dataset in a manner consistent with a purely extensionalist account of the data. We then showed how the addition of a more detailed account of a key component, the processing of word meanings, could be added to refine the model, again maintaining consistency in model parameters.

Computer science, which has often been used as a metaphor in the cognitive modeling domain, gives a clear precedent for analyzing and thinking about modeling a single system in terms of multiple views. Cognitive science has, of course, developed similar and parallel conceptualizations to those of computer science; indeed, Marr famously elaborated a version of this position in his three levels of cognitive description (Marr, 2000). However, despite Marr's observations, concrete modeling endeavors in cognitive science typically seek to model data accurately and to compete for adequacy. Rarely, if ever, are multiple abstraction levels explicitly modeled for the same data while maintaining formal relationships between models at different levels.

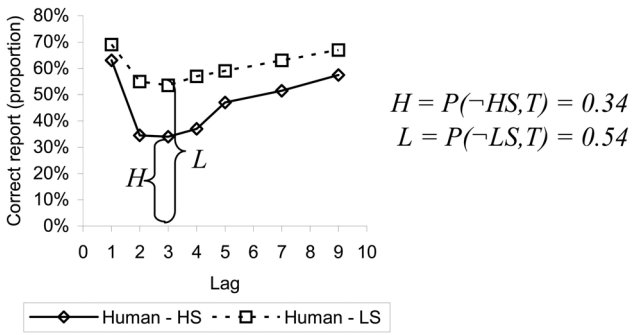

FIGURE A1 | Target report accuracy by lag in humans for high and low salient key-distractors with intrinsic identifications.

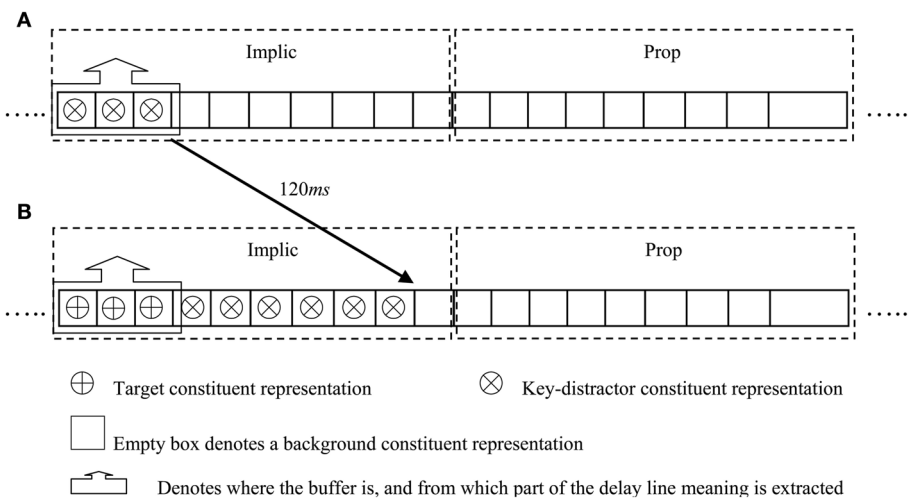

FIGURE A2 | Snapshots of the delay-line in the lag-1 case when (A) Implic decides to move the buffer to Prop after it has processed the first three constituent representations of the key-distractor; (B) the buffer actually moves just after Implic has processed the first three constituent representations of the target.

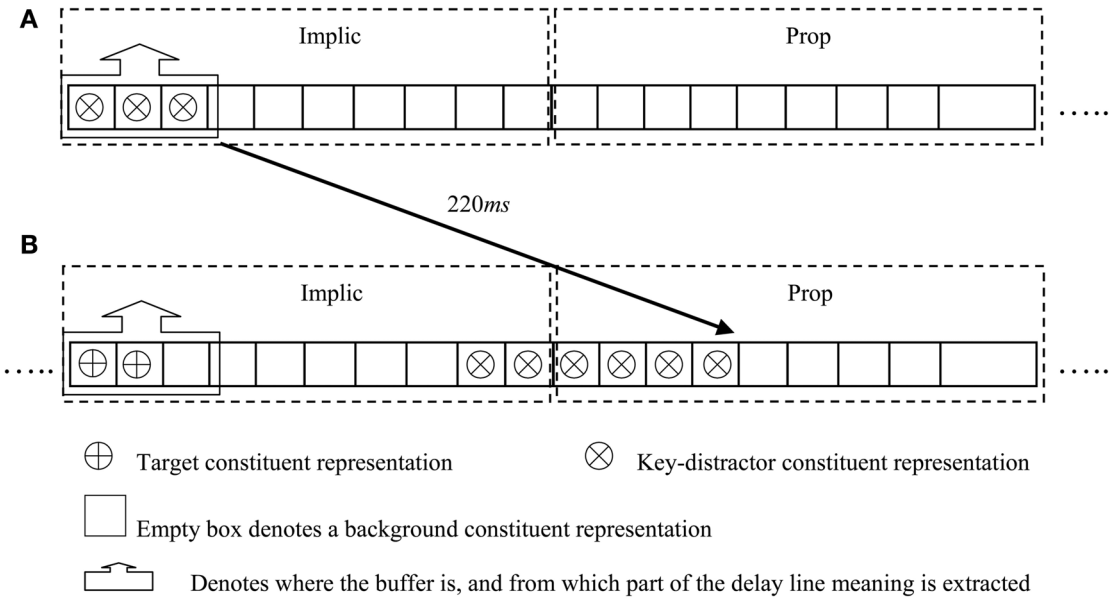

FIGURE A3 | Snapshots of the delay-line when (A) Implic decides to move the buffer to Prop after it has processed the first three constituent representations of the key-distractor; (B) the first two constituent representations of the lag-2 item (target) have entered Implic.

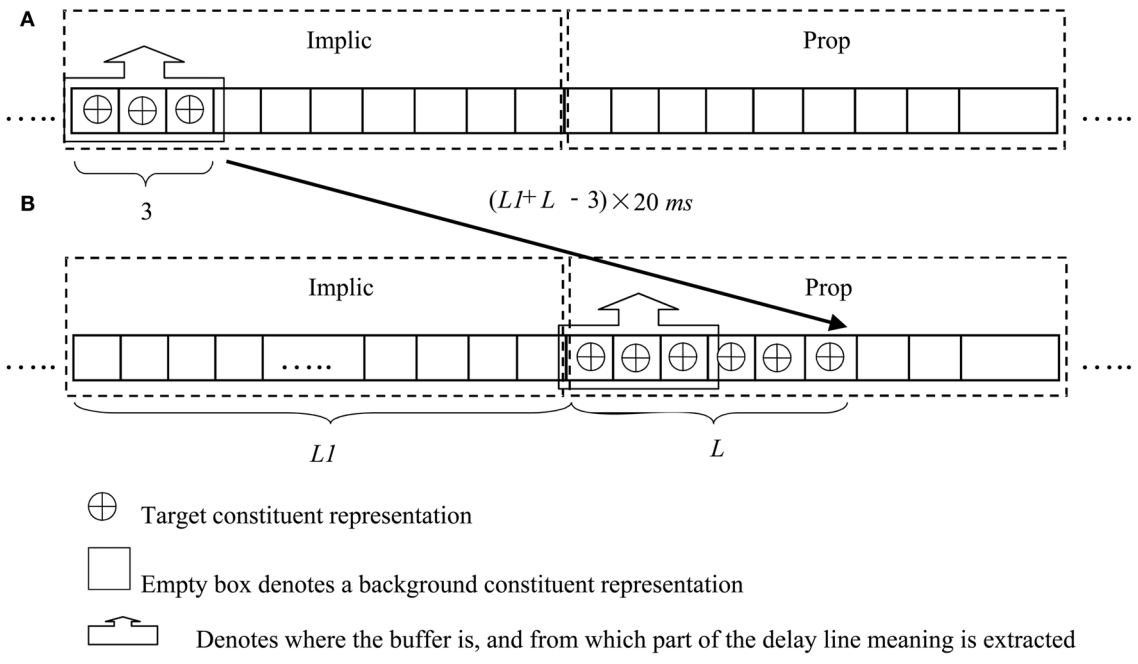

FIGURE A4 | Snapshots of the delay-line when (A) Implic decides to move the buffer to Prop after it has processed the first three constituent representations of the target; (B) the buffer actually moves just after the last three constituent representations of the target have entered Prop.

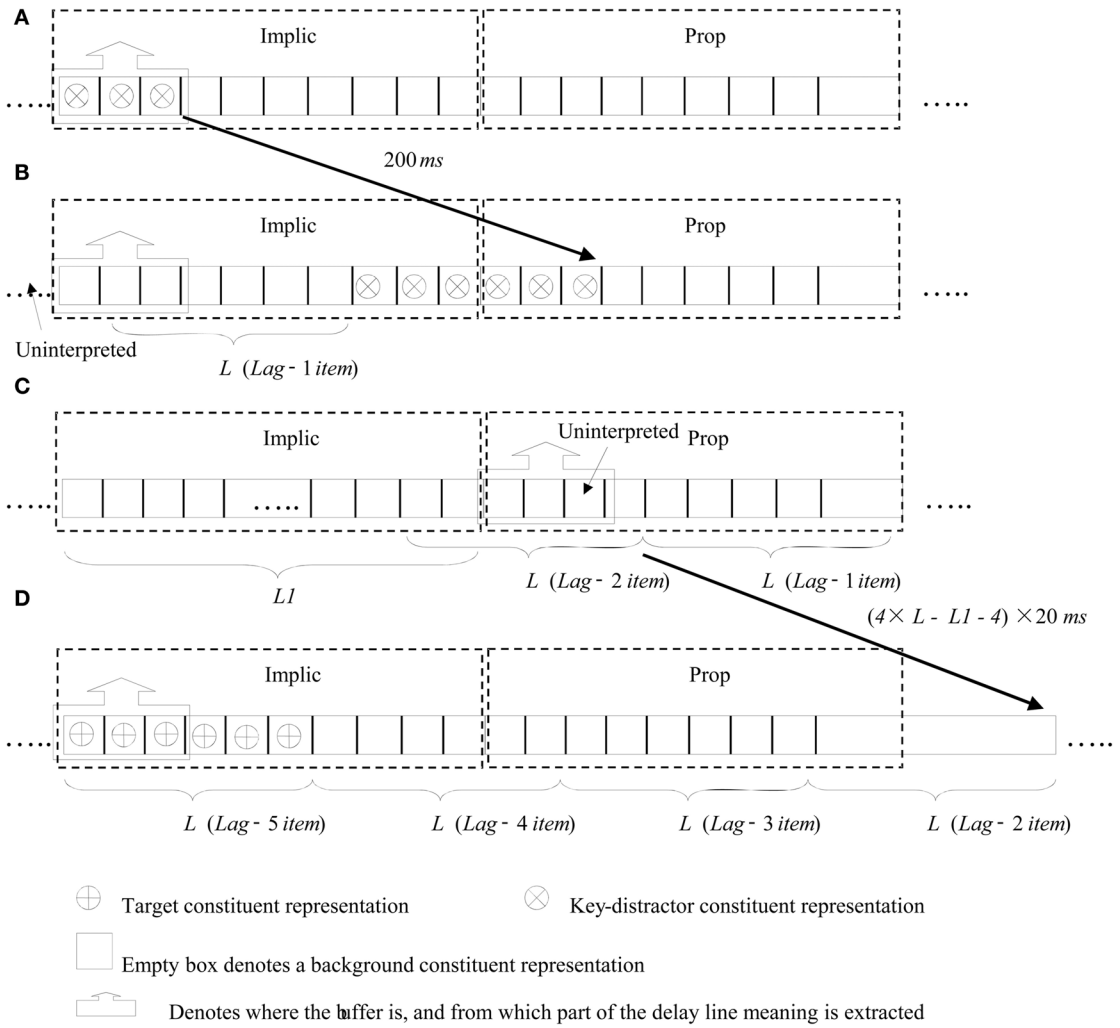

FIGURE A5 | Snapshots of the delay-line when (A) Implic decides to move the buffer to Prop after it has processed the first three constituent representations of the key-distractor; (B) buffer actually moves after a delay of 200 ms; (C) Prop decides to move the buffer back to Implic after it has seen three implicationally un-interpreted constituent representations; (D) the buffer actually arrives at Implic when the last three constituent representations of the lag-5 item (target) have entered Implic.

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A potential role of the inferior frontal gyrus and anterior insula in cognitive control, brain rhythms, and event-related potentials

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In the present paper, we review evidence for of a model in which the inferior frontal gyrus/anterior insula (IFG/AI) area is involved in elaborate attentional and working memory processing and we present the hypothesis that this processing may take different forms and may have different effects, depending on the task at hand: (1) it may facilitate fast and accurate responding, or (2) it may cause slow responding when prolonged elaborate processing is required to increase accuracy of responding, or (3) it may interfere with accuracy and speed of next-trial (for instance, post-error) performance when prolonged elaborate processing interferes with processing of the next stimulus. We present our viewpoint that ventrolateral corticolimbic control pathways, including the IFG/AI, and mediodorsal corticolimbic control pathways, including dorsal anterior cingulate cortex areas, play partly separable, but interacting roles in adaptive behavior in environmental conditions that differ in the level of predictability: compared to dorsal feed-forward control, the ventral corticolimbic control pathways implement control over actions through higher responsiveness to momentary environmental stimuli. This latter control mode is associated with an attentional focus on stimuli that are urgent or close in time and space, while the former control mode is associated with a broader, more global focus in time and space. Both control pathways have developed extensively through evolution, and both developed their own "cognitive controls," such that neither one can be properly described as purely "cognitive" or "emotional." We discuss literature that suggests that the role of IFG/AI in top-down control is reflected in cortical rhythms and event-related potentials. Together, the literature suggests that the IFG/AI is an important node in brain networks that control cognitive and emotional processing and behavior.

Keywords: post-error slowing, post-conflict slowing, speed–accuracy trade-off, cognitive control, inferior frontal gyrus, anterior insula, event-related potentials, cortical rhythms

INTRODUCTION

Performance adjustments following conflict or erroneous responses, as well as changes in speed–accuracy trade-off, are regarded as examples of cognitive control and are most often ascribed to brain areas such as medial frontal cortex/anterior cingulate cortex (ACC) and dorsolateral prefrontal cortex. Although these performance adjustments have also been associated with activity in a ventrolateral prefrontal cortical area (that includes several sub-areas including the inferior frontal junction) that we will broadly refer to as inferior frontal gyrus/anterior insula (IFG/AI), such activity is often discounted as being related to motor inhibition or orienting responses, and not functionally meaningful or "cognitive." In the present opinion paper, we will present a model, and discuss evidence, suggesting that the IFG/AI is involved in elaborate attentional and working memory processing and present the hypothesis that this processing may take different forms and may have different effects, depending on the task at hand: (1) it may facilitate fast and accurate responding, or (2) it may cause slow responding when prolonged elaborate processing is required to increase accuracy of responding, or (3) it may interfere with accuracy and speed of next-trial (for instance, post-error) performance when prolonged elaborate processing interferes with processing of the next stimulus. Through this function, IFG/AI appears involved in minimizing interference effects in flanker and Stroop tasks, in slowing effects such as post-error slowing, post-conflict slowing, and in speed–accuracy trade-off.

Although we think there is converging support for this function of IFG/AI, we do not argue for a focus on the IFG/AI at the expense of investigation of the ACC, of interactions between areas including ACC and IFG/AI, or at the expense of more detailed characterization of areas within IFG/AI and ACC and their participation in brain networks. Rather, because functions of areas

including dorsal ACC have more often been described as cognitive, while functions of IFG/AI, and the ventral corticolimbic control pathways it is part of (including also the amygdala), have often been described as emotional, in this paper we want to distil from the literature an emerging picture of IFG/AI contributions to cognition and focus on characterizing the cognitive control functions the IFG/AI might have. In doing so, we are guided by our viewpoint that ventral corticolimbic control pathways that include the IFG/AI, and dorsal corticolimbic control pathways that include dorsal ACC areas, are interacting but partly separable by their respective adaptations to environmental conditions that differ in the level of predictability (Tops et al., 2010). We will discuss that, in our opinion, both control pathways continued to develop during evolution, and both developed their own "cognitive controls," such that neither one can be properly described as "cognitive" nor "emotional" (Tops et al., 2010). More general and balanced discussions of theories and research of cognitive control and post-error adjustments can be found in other contributions to this Research Topic (e.g., Danielmeier and Ullsperger, 2011).

Increasing attention to the role of IFG/AI could have important implications and could facilitate future research in several directions. For instance, cognition and ACC function are increasingly being investigated in the context of psychopathology, mental health, and stress. However, the IFG/AI is among the areas that most consistently show increased activity related to anxiety and stress (see for a meta-analysis Etkin and Wager, 2007; for discussion Tops and Boksem, 2011).

In Section "Cognitive Control in the Brain," we will argue that IFG/AI has a relatively neglected role in cognitive control that is different from ACC. In Section "Cognition and Emotion in Dorsal and Ventral Corticolimbic Control Pathways" we will present our general model of ventral and dorsal control pathways and their role in cognition. In Section "The Role of IFG/AI in Cognitive Control" we focus on cognitive control functions of IFG/AI in the ventral control pathways, and how this function may be implicated in minimizing interference effects in flanker and Stroop tasks, in slowing effects such as post-error slowing, post-conflict slowing, and in speed–accuracy trade-off. In Section "Support for a Role of IFG/AI in Cognitive Control" we discuss evidence for involvement of IFG/AI in these interference and slowing effects. In Section "Long-Term Goals" we suggest that both ventral and dorsal control pathways can direct behavior toward long- and short-time goals, but in different ways. Finally, we discuss evidence suggesting that the role of IFG/AI in top-down control is reflected in cortical rhythms and event-related potentials.

COGNITIVE CONTROL IN THE BRAIN

Functional neuroimaging studies suggest that dorsal ACC plays an important role in cognitive control. This brain area is reliably activated when tasks require the ongoing adjustment of the allocation of attention. The ACC has come to occupy a central role in theories of attention and cognitive control, which hold that the ACC either monitors response conflict, signaling the need for adjustments in cognitive processes, or directly mediates such adjustments (e.g., Botvinick et al., 2001). However, it has been shown that subjects with damage to the dorsal ACC show normal adjustments in performance following manipulations in response conflict in both Stroop and go–no-go tasks. Furthermore, damage to the ACC did not impair performance on anterior attention tasks, post-error slowing, nor the ability to adjust performance in response to explicit speed or accuracy instructions, arguing against a necessary role for the ACC in these processes (Fellows and Farah, 2005; Baird et al., 2006).

A study in which the ACC was lesioned in monkeys, showed that the ACC is not involved in detecting or correcting errors, but in guiding voluntary behavior based on the history of actions and outcomes (Kennerley et al., 2006). ACC lesions did not impair the performance of the monkeys immediately after errors, but made them unable to integrate rewards and punishments over multiple trials to guide the continuation of behavior. But if the ACC is not involved in next-trial response–adjustments, this means that this kind of computations and behavioral control is performed elsewhere, and that the outcomes of such computations are forwarded to the ACC, in order to be integrated over multiple trials, guiding the choice of a general behavioral set and level of engagement. Interestingly, in a recent fMRI study dorsal ACC activity was not sensitive to Stroop congruency, error likelihood, or response conflict after controlling for increased brain activity with time-on-trial, suggesting that the greater ACC activity on incompatible trials may stem from longer reaction times rather than response conflict; however, left IFG activity was correlated with increased Stroop congruency effects (Grinband et al., 2011). There is discussion about the study of Grinband et al. (Yeung et al., 2011), and there appears to be evidence for involvement of rostral ACC in next-trial cognitive control (di Pellegrino et al., 2007). Our intention is not to argue against ACC involvement in next-trial cognitive control, but to highlight potential IFG/AI contributions.

There is growing support for, and attention to, the role of IFG/AI pathways in executive functions such as post-error slowing that have so far largely been ascribed to the ACC–prefrontal cortical circuit, and recognition of the involvement of orienting responses in such functions (Tucker et al., 2003; Tops, 2004; Brass et al., 2005; Tops et al., 2006, 2010; Boksem et al., 2008; Eckert et al., 2009; Notebaert et al., 2009; Tops and Boksem, 2010, 2011; Ullsperger et al., 2010; Ide and Li, 2011). Where less then 10 years ago strong activation of the IFG/AI was regarded of no importance, and in an astonishing number of cases was reported without comment, an extraordinary convergence of evidence has since prompted authors to describe this area as the integral hub and convergence zone between networks that control behavior in low-predictable environments (Bossaerts, 2010; Craig, 2010; Menon and Uddin, 2010; Nelson et al., 2010; Tops et al., 2010; Higo et al., 2011). Likewise, while classically considered a limbic region, recent evidence from network analysis suggests a critical role for the IFG/AI in high-level cognitive control and attentional processes (Craig, 2010).

COGNITION AND EMOTION IN DORSAL AND VENTRAL CORTICOLIMBIC CONTROL PATHWAYS

Research investigating the evolution and ontogeny of the prefrontal cortex suggests that the lateral prefrontal cortex initially emerged from ventrolateral prefrontal regions, followed by dorsolateral and then anterolateral cortices (Flechsig, 1901, 1920; Fuster et al., 1997). We previously argued that evolution did not lead to the

development of separate brain systems for emotion vs. cognition, but did lead to the development of partially separate ventrolateral and mediodorsal control pathways sustaining behavioral programs adapted to different environments (Tops et al., 2010). We previously described how the distinction between ventrolateral and mediodorsal control pathways can be applied to literatures about temperament, personality, emotion, and psychopathology (Tops et al., 2010). However, the theory of Tucker and Luu from which it was developed has also been applied to cognitive control (e.g., in the empirical work of Luu and Tucker) and the modulation by emotion of the width of attention (reviewed by Friedman and Förster, 2010). In **Figure 1** we present a model of our hypothesis regarding how the ventral and dorsal control pathways are implicated in cognitive control, with a relative focus on the often neglected ventral controls.

In short, the revised model of Tucker and colleagues (Tops et al., 2010) proposes that two types of brain systems developed during evolution. One type was adapted to control cognition and behavior in high-predictable environments. These systems control behavior guided by context models; models that are formed in long-term memory by the predictability of the environment/context. The other type of system was adapted to control cognition and behavior in low-predictable environments. In low-predictable environments, effective context models can not be formed nor used to control behavior in adaptive ways. Instead, behavior is guided *reactively* by momentary feedback control by environmental stimuli. This *reactive* guidance by momentary environmental stimuli is associated with attentional focus on stimuli that are urgent and close in time and space. Those stimuli can be positive ("I have to catch that reward that is in my reach before it gets away") or negative ("I have to get away from that danger before it gets me, because I'm in its reach"). The reactive systems are involved in, and relate stimuli to, the experienced self in the here and now. In contrast, there is less urgency and focus on the moment (i.e., broader, more global focus in time and space) when behavior is guided *proactively* (in feed-forward fashion) by context models. Playful exploration of the environment may be stimulated by this type of control, to support the construction and updating of context models.

Friedman and Förster (2010) reviewed literature showing that positive emotional states and implicit affective cues expand (global focus), and that negative emotional states and implicit affective cues constrict (local focus), the scope of attention on both the perceptual and conceptual level. They concluded that a large and growing body of research supports the model and assumptions that originated from Tucker's work. Starting with Tucker's neuropsychological theory (e.g., Tyler and Tucker, 1982; Tucker and Williamson, 1984; Derryberry and Tucker, 1994; Luu et al., 1998), early studies were collectively inspired by a set of converging empirical and theoretical contributions (e.g., Schwarz, 1990; Tucker et al., 1995; Fredrickson, 1998). Friedman and Förster also discussed recent findings and ideas by Harmon-Jones and Gable, which, as an exception, did not appear to fit the theoretical framework. These authors reported several studies in which reactive positive (appetitive, e.g., hunger) reward motivation facilitated a

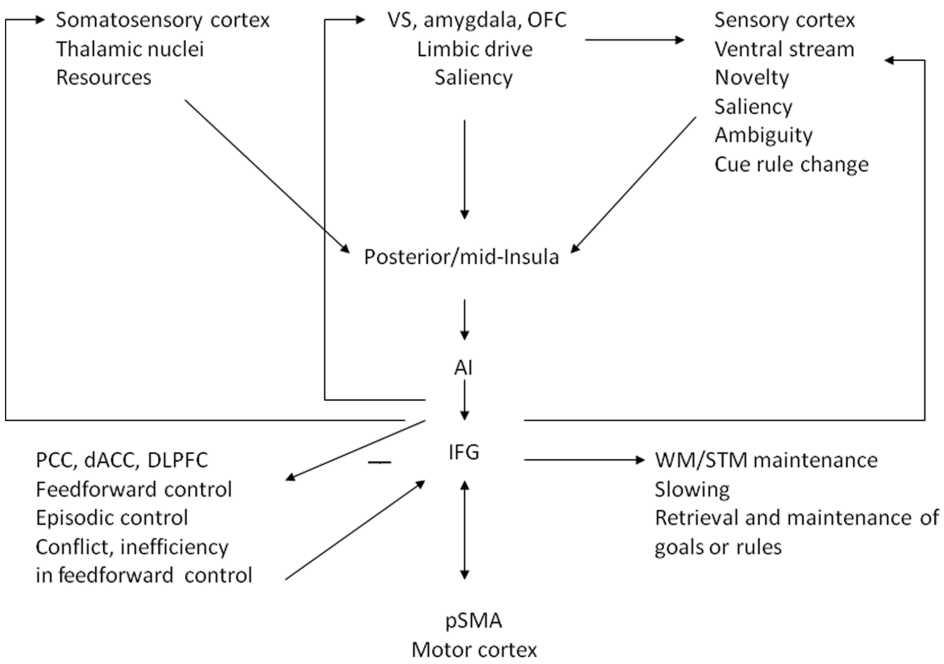

FIGURE 1 | Schematic depiction of the roles of ventral and dorsal corticolimbic control pathways in cognitive control, focusing on ventral control. At the level of the IFG/AI interoceptive, limbic emotion–motivational, and sensory inputs are integrated. IFG/AI connects back to somatosensory, limbic, and sensory/attentional orienting areas to regulating the level of activation of representations that are relevant for response selection. IFG/AI may also keep the representations and goals active as part of maintenance working memory function. IFG, inferior frontal gyrus; AI, anterior insula; VS, ventral striatum; OFC, orbitofrontal cortex; PCC, posterior cingulate cortex; dACC, dorsal anterior cingulate cortex; dlpfc, dorsolateral prefrontal cortex; pSMA, presupplementary motor area; WM, working memory; STM, short-term memory.

local focus (Gable and Harmon-Jones, 2008; Harmon-Jones and Gable, 2009). Although the attempt by Friedman and Förster to reconcile the ideas of Gable and Harmon-Jones with their own ideas and the general framework figured prominently in their original review article, Harmon-Jones et al. (2011b) published a comment in Psychological Bulletin proposing an alternative model to explain the findings. However, in their reply, Friedman and Förster (2011) argue that the alternative model fails to establish a compelling alternative explanation for the multitude of specific findings they reviewed. Interestingly and importantly, the revision and update of Tucker's model we published last year actually prominently discussed the findings of Gable and Harmon-Jones to explain how small revisions to the original model increase the explaining power and fit to the literature (Tops et al., 2010).

The revised model retains the hypothesis that the systems associated with the context models are biased toward positive emotion, optimism, self-efficacy, and confidence, because the context models are based on previous predictive success and positive outcomes. However, one adjustment is the addition of a reactive system with a narrow focus in space and time on obtaining rewards, in addition to the reactive system with a narrow focus in space and time on avoiding punishment or harm. This additional appetitive reactive reward-oriented system incorporates the findings (local focus when reactive reward motivation is stimulated) and ideas of Harmon-Jones et al. (2011b) within the broader framework that was built from Tucker and colleagues's original work. Moreover, it also seems to incorporate findings with other outcome measures in the research by Förster and colleagues (e.g., Förster, 2009; Liberman and Förster, 2009). They found that a global attentional focus was associated with larger psychological distances in time and space, "promotion focus," high power and a focus on similarities (which is compatible with the formation of context models), while a local attentional focus was associated with small psychological distances in time and space, "prevention focus," low power and a focus on differences.

According to the self-regulation theory of Higgins (1997), a promotion focus guides behavior according to ideals and aspirations, while a prevention focus guides behavior according to "oughts" such as safety and responsibilities. Interestingly, a recent theory of reasoning and decision making proposes that dorsolateral prefrontal cortex represents behavior-guiding principles for evaluating the permissibility or fairness of observed behavior while social norms for necessary (obligatory or prohibited) courses of action are represented by ventrolateral prefrontal cortex (Barbey et al., 2009). Promotion relative to prevention focus has been associated with perceptions of power and predictability (e.g., Langens, 2007). Behavior control by social norms for necessary courses of action in ventrolateral prefrontal cortex may have developed from its attentional focus on stimuli that are urgent and close in time and space, while reflection on ideals, permissibility, and fairness may be allowed for when there is less urgency and focus on the moment and may involve activation of context models. Barley et al. argue that, from an evolutionary perspective, the emergence of lateral prefrontal cortex subregions reflects their relative priority for the formation of organized social groups. Furthermore, consistent with its evolutionary development, the ontogeny of the lateral prefrontal cortex reflects the importance of first representing social norms for necessary behavior (i.e., fundamental rules the child must obey), followed by an understanding of permissible courses of action (e.g., guided by judgments of equity and fairness), and finally high-order inferences involving both forms of representation (Barbey et al., 2009). The work by Barbey et al. (2009) may extend our model to the realm of higher order processing such as social reasoning and decision making, and inductive and deductive inference making.

The present model seems related to the well-known hypothesized distinction between ventral and dorsal posterior visual processing streams specialized respectively in processing of "what" and "how" information (Goodale and Milner, 1992). According to Goodale and Milner, the dorsal pathway extracts visual signals relevant for driving automatic or feed-forward motor behavior (perception for action), whereas the ventral pathway extracts information relevant for identification and other forms of semantic knowledge. It has been suggested that these processing streams are extended toward frontal ventral and dorsal areas that feed back to implement cognitive control (Sakagami et al., 2006; O'Reilly, 2010). Ventrolateral prefrontal cortex (IFG/AI) mediates active maintenance of stimulus information, and this produces a top-down biasing effect to drive selection and retrieval dynamics in posterior cortex. On the other hand, one should expect to see dorsolateral prefrontal cortex activation whenever the dorsal parietal cortex requires extra cognitive control (such as working memory and top-down biasing) to carry out the processing of sensory information to guide action outputs (O'Reilly, 2010; cf. Sakagami et al., 2006). Although similar, compared to this account our model seems more integrative, associating ventral and dorsal systems with behavioral programs that include motivation, emotion, viscerosensation, and memory (Tops et al., 2010).

Figure 1 shows that at the level of the IFG/AI interoceptive, limbic emotion–motivational, and sensory inputs are integrated (Craig, 2008, 2009). Based on reviews of these and other findings, Craig reasoned that in the IFG/AI an integrated representation is formed of the global emotional moment, i.e., that awareness of the immediate moment is formed. Also in our model, the IFG/AI is an integration and control node in ventral networks that implement "immediate" reactivity to sensory stimuli, and "immediate" connection between sensory processing and action control. Adapted to low-predictable environments, ventral control applies a narrow spatial (target-focused) and temporal (immediate moment) focus to processing, action control, and awareness (cf. Craig, 2009). In contrast, adapted to predictable environments, dorsal control applies a wider spatial and temporal focus, which allows for recruitment of context models, and sustained control over behavioral episodes according to information conveyed by temporally remote events and history of actions and outcomes, and implementation of feed-forward control of behavioral patterns and integrated action sequences (Heidbreder and Groenewegen, 2003; Kennerley et al., 2006; Kouneiher et al., 2009). The wider temporal focus produces awareness that includes a sense of past and future. Notice that quite differently from reactive ventral control, feed-forward implementation, and execution of action sequences may be facilitated by suppressing ongoing processing of sensory input that might disrupt motor output (Jacobs and Fornal, 1995).

Classical work showed that affective arousal states carry resource information (physiological resources such as glucose levels and the condition of muscles, as well as social resources) and are associated with implicit perceptions of coping abilities (Thayer, 1989). Only in low-predictable environments it is necessary to have a continuous readout of the level of available resources to constrain immediate action. In the IFG/AI information about the level of resources is combined with emotional or "drive" information that biases the direction of action either toward (approach, e.g., craving, hunger, love, trust) or away from (avoidance, e.g., disgust, pain, distrust) a target object (Tops and de Jong, 2006; Tops et al., 2010). This directional drive property may have been derived from functions of the gustatory cortex that is situated in the insula. The directional drive bias and resource information is furthermore combined with relevant target information and, depending on circumstances, priming or preparation of responses and matching autonomic responses (Heidbreder and Groenewegen, 2003). Together, the continuous readout of drive direction bias, resource level, and orienting toward potential targets enable fast, opportunistic action at the spur of the moment. In other words, it may enable us to catch a reward that is in our reach before it gets away and to get away from a danger before it gets us, when we are in its reach. In contrast, in predictable environments dorsal control enables fast and efficient action by means of feed-forward action control and guidance by context models.

In many situations and for many tasks, dorsal, and ventral systems will collaborate and interact in the control of behavior. For instance, support has been found for interactions between IFG and dorsal frontal areas, where IFG implements reactive (momentary) immediate action according to the information conveyed by concomitant contextual signals, while dorsal frontal areas implement "proactive" episodic motivation control: sustained control over behavioral episodes according to information conveyed by temporally remote events (or context models) and history of actions and outcomes, and implementation of feed-forward control of behavioral patterns and integrated action sequences (Heidbreder and Groenewegen, 2003; Kennerley et al., 2006; Kouneiher et al., 2009). However, temporary or relatively stable biases toward reactive control may result from temperament, unpredictable dangerous or urgent situations, perceptions of unpredictability after trauma or inconsistent parenting, and interactions between those. In contrast, bias toward context model-guided control may relate to temperament or follow consistent parenting and predictable, secure early environments that favored exploration and the development of context models (i.e., internal working models, in terms of Bowlby's (1988) attachment theory).

The input and feedback to IFG/AI and back to somatosensory, limbic and sensory/attentional orienting areas as depicted by arrows in **Figure 1**, fit the consistent implication of IFG/AI activity in somatosensory working memory (e.g., Auksztulewicz et al., 2011), anxiety, and somatic complaints (Paulus and Stein, 2006; Etkin and Wager, 2007; Tops and Boksem, 2011). We suggest that during evolution cognitive control areas have developed that are basically control areas involved in temperament, emotion regulation, and stress responding (Cromwell and Panksepp, 2011), but now also implicated in cognitive task performance. Hence, we think the model in **Figure 1** can be applied to temperament, emotion regulation, stress responding, as well as to cognitive control. However, the next sections of this paper will focus on the involvement of the connections between IFG/AI and the ventral processing stream in cognitive control.

THE ROLE OF IFG/AI IN COGNITIVE CONTROL

In **Figure 1**, the feedback connection between IFG/AI and ventral sensory cortex is meant to illustrate the role of IFG/AI in regulating the level of activation of representations in posterior brain areas that are relevant or irrelevant, respectively, for response selection (Higo et al., 2011). In other words, those connections have a role in the coordination of focal elaborate processing of target stimuli; focal in time and space. Depending on the requirements of the task at hand, these connections together with output to motor cortex allow for tight feedback control of action. This means that IFG/AI is involved both in monitoring target or cue events and in updating the corresponding action plan. One example that supports such a combination of functions is a study that combined the causal methodology of reversible cortical interference (transcranial magnetic stimulation) with an experimental task that measured different types of updating. This study found that the right inferior frontal cortex can be functionally segregated into two subregions: an inferior frontal junction region that seems critical for visual detection of changes in the environment, and a more ventral IFG region, which updates the corresponding action plan (Verbruggen et al., 2010).

However, if the target stimulus is removed before (elaborate) processing is finished, processing may proceed on a short-term memory representation of the target or sensory input. Much has been learned over the last two decades on where in the brain working memory functions are carried out. Much less is known on how the brain accomplishes short-term maintenance and goal-directed manipulation of information. One view proposed a functional distinction, arguing that ventrolateral areas are mostly involved in pure maintenance of information and keeping representations active without external input, whereas dorsolateral areas are more involved in tasks requiring some processing of the memorized material (Owen, 2000). The IFG appears to project back to the temporal lobe to keep target/object representations active (Assadollahi and Rockstroh, 2008) and may do so through mechanisms of synchronization of neuronal firing (cf. Hommel et al., 2006). We suggest that similar feedback connections may be implicated both in regulating the level of activation of representations in posterior brain areas that are relevant to prevent interference from irrelevant simultaneous representations, and in regulating the level of activation of representations in posterior brain areas that are relevant to prevent decay or interference from subsequent representations. However, as we are not aware of much evidence for this suggested mechanism, we leave the exact mechanism of short-term memory or maintenance working memory in the IFG/AI open.

Both prolonged focus on, and processing of, potentially ambiguous or relevant target representations that are still updated in sensory cortex, and prolonged processing of targets that have been removed from input channels, constitute what has been called a "redundancy bias" to processing in ventral systems (Tucker et al., 1995). Importantly, although both operations exclude the continuous stream of sensory input largely or completely from

momentary awareness, momentary awareness itself is continued with focus on the target. That is, if processing is performed on a short-term memory representation, the "emotional moment" (Craig, 2009) constitutes of outcomes of this processing in IFG/AI and simultaneous inputs such as limbic drive and somatosensory inputs.

Prolonged processing of targets that have been removed from input channels (or replaced or updated) places that target in momentary awareness, and excludes subsequent stimuli and targets from momentary awareness for the duration of this prolonged processing. This may cause interference with IFG/AI mediated processing and momentary awareness of stimuli following targets in rapid serial visual presentation tasks and following stimulus events that would trigger prolonged elaborate processing in speeded reaction time tasks, such as performance errors, incongruent flankers, novelty, saliency, ambiguity, or cues of rule change (**Figure 1**). We think that this interference with the processing of subsequent stimuli is involved in post-error slowing and post-conflict slowing.

We leave open whether control from the IFG/AI involves any active inhibition of processing or action, or only activation of representations (input or behavior) that compete with other representations, which has been suggested to be a more parsimonious explanation of IFG/AI functions (Hampshire et al., 2010). In terms of visual processing, inhibition of one object when attention is focused on another can be explained as a secondary effect, i.e., an emergent property of local competition when one competing item is subjected to top-down potentiating signals which have their source in the IFG/AI and may reflect willful focusing of attention (Hampshire et al., 2010). However, this leaves the possibility that motor programs of immobility or behavioral inhibition are part of innate stimulus – response programs or are often used and therefore primed in certain individuals (Tops and Boksem, 2011).

Similarly, we will not discuss extensive evidence that IFG/AI may inhibit processing of emotional stimuli in sensory cortex, memory, and limbic areas. Although this literature is too extensive and complicated to discuss within the scope of this paper, we will mention a few examples, because we think emotion control by the IFG/AI may involve mechanisms that are similar to, and may overlap with, those implementing cognitive control. In one study, anticipatory bilateral IFG/AI activation before picture presentation was inversely correlated with superior temporal gyrus (STG) activation during presentation of scary pictures in anxiety prone individuals, suggesting that IFG/AI activation suppressed the sensory representation in STG (Simmons et al., 2006). According to another study, emotional memories are initially suppressed by the right IFG over regions supporting sensory components of the memory representation (visual cortex, thalamus; Depue et al., 2007). One explanation for these findings is that the right IFG is engaged in a coping strategy – for example retrieving an alternative thought, image, or memory in order to swamp limited capacity processing resources (Hampshire et al., 2010). Wager et al. (2008) identified a right IFG/AI region whose activity correlated with reduced negative emotional experience during cognitive reappraisal of aversive images. They then applied a pathway-mapping analysis on subcortical regions to locate mediators of the association between IFG/AI activity and reappraisal success (i.e., reductions in reported emotion). They identified two separable pathways that together explained approximately 50% of the reported variance in self-reported emotion: (1) a path through nucleus accumbens that predicted greater reappraisal success, and (2) a path through ventral amygdala that predicted reduced reappraisal success (i.e., more negative emotion). These results provide direct evidence that IFG/AI is involved in both the generation and regulation of emotion through different subcortical pathways. Maybe this result means that the right IFG is involved in the passive coping strategy of emotion-focused coping by amplifying positive vs. negative emotions.

SUPPORT FOR A ROLE OF IFG/AI IN COGNITIVE CONTROL

FLANKER INCONGRUENCY EFFECTS AND SPEED–ACCURACY TRADE-OFF

The right IFG/AI may have an alarm/orienting function as part of its critical role in the switching between internally and externally oriented control modes in response to salient stimuli (Sridharan et al., 2008). Furthermore, the IFG/AI may coordinate and participate in further processing of salient and/or ambiguous stimuli. Leitman et al. (2010) showed that increased saliency of emotion-specific acoustic cues was associated with increased activation in key components of the ventral emotional/attentional system including STG, insula, and amygdala, whereas decreased saliency of acoustic cues was associated with increased IFG activity and IFG–STG connectivity. These results suggest that sensory-integrative processing that is central in emotional intensity and attentional absorption is facilitated when the stimulus is rich in affective information, yielding increased activation in temporal cortex and amygdala. Conversely, when the stimulus is ambiguous, greater evaluative processes are recruited, increasing activation in IFG, and IFG–STG connectivity (Leitman et al., 2010).

Incongruency in a flanker task activates the IFG/AI and underlying striatum area, and although the direction of the relationship is inconsistent over studies, activity in this area was in some studies correlated with the flanker incongruency effect on reaction times (Bunge et al., 2002; Wager et al., 2005); a relationship was also found with the incongruency effect on reaction times during a Stroop task (Melcher and Gruber, 2008). The IFG/AI–ACC network is thought to be involved in incongruency detection or resolving, and/or in inhibitory processes that dampen the tendency to make an inappropriate response (e.g., Aron et al., 2004; Wager et al., 2005). The IFG/AI is active across tasks involving incongruency, inhibition or working memory (including the flanker, go/no-go, stop signal, stimulus–response compatibility, Simon, Stroop, and spatial- and verbal-working memory tasks; Nee et al., 2007; McNab et al., 2008) including tasks involving semantic or emotional incongruency that elicit the N400 evoked potential (Maess et al., 2006; Van Petten and Luka, 2006), consistent with a role of this area in a bias to working memory or attention that increases processing of ambiguous stimuli. The IFG/AI also consistently shows error-related activity (Wittfoth et al., 2008) which may reflect increased processing after erroneous responses. Moreover, a meta-analysis suggested that there is an asymmetry in the activation of the IFG/AI, left IFG/AI showing larger activation by flanker incongruency and right IFG/AI by errors (Ullsperger et al., 2010).

Flanker incongruency effects depend on speed–accuracy strategy, the effects being larger when speed is stressed (Wylie et al., 2009). The speed and accuracy of decision making have a well-known trading relationship: hasty decisions are more prone to errors while careful, accurate judgments take more time. Using functional magnetic resonance imaging (fMRI), Ivanoff et al. (2008) showed that emphasizing the speed of a perceptual decision at the expense of its accuracy lowers the amount of evidence-related activity in the IFG/AI that is gathered before responding. Moreover, this speed–accuracy difference in activity correlated with a behavioral measure of speed–accuracy difference in decision criterion. Thus, the IFG seems involved in elaborate attentional and working memory processing that may facilitate fast and accurate responding or slow responding in the case of prolonged elaborate processing to increase accuracy of responding.

POST-ERROR SLOWING

It has been reported that post-error slowing was larger when instruction stressed accuracy rather than speed (Jentzsch and Leuthold, 2006; Danielmeier and Ullsperger, 2011). In a study by Dudschig and Jentzsch (2009), post-error slowing was found to be increased and performance more error-prone with a decreasing response–stimulus interval, providing evidence for the idea that error evaluation can produce substantial interference with subsequent trial processing, particularly when there is insufficient time between the error and the subsequent event. Whereas response–stimulus-intervals were varied block-wise in the study by Dudschig and Jentzsch, their result was replicated in a study that varied response–stimulus-intervals trial-to-trial (Danielmeier and Ullsperger, 2011). EEG alpha power during task performance was recently shown to display error-related and incongruency-related changes (Carp and Compton, 2009; Compton et al., 2011). While alpha suggested temporary disengagement after correct responses, after errors there appeared to be a failure to disengage which predicted post-error slowing. Indeed, depression is associated with a decrease in accuracy, increased slowing and inability to disengage after errors (Tucker et al., 2003; Compton et al., 2008) and error-related negativity (ERN) ERP amplitude predicted post-error slowing only among depressed participants in an emotional Stroop task condition involving negative words (Compton et al., 2008), suggesting a relationship in depression between slowing and failure to disengage. Interestingly, color-naming reaction time interference effects by threat stimuli in the emotional Stroop, which has been associated with anxiety in numerous studies, also appear to reflect slow disengagement from the previous trial, similar to what seems to be happening in post-error slowing (Phaf and Kan, 2007). See for a discussion of the relation between post-error slowing and anxiety, arousal, and orienting responses, Notebaert et al. (2009) and Tops and Boksem (2011).

Unpublished results of a recent study (Tops and Boksem, 2010) in which subjects performed the Eriksen flanker task for 2.5 h, support that post-error processing may interfere with accurate next-trial performance when prolonged elaborate processing interferes with processing of the next stimulus. Over the whole group of subjects, post-error slowing was only significant in the first interval of the experiment (first 20 min), and parallel to increasing reaction times, post-error accuracy in the first intervals switched to post-error *inaccuracy* in the last interval (i.e., the last 20 min). Moreover, in the first interval, the only interval that showed both significant post-error slowing and post-error accuracy, post-error accuracy was correlated with shorter reaction times. After controlling for reaction times, post-error slowing predicted post-error accuracy. This suggests that only when reaction times were short the response–stimulus interval was long enough to benefit from the post-error processing, while when post-error processing added up to longer overall reaction times, there was interference of post-error continued engagement with the last trial with processing and performance in the subsequent trial.

In contrast, in a task in which an error on a difficult "lure" trial predicted that the same lure would be repeated between two and seven trials later, such that effects of post-error failure to disengage were unlikely to interfere with performance on the next lure trial, post-error slowing was related to increased accuracy on the next lure trial; the slowing and the increased accuracy were predicted by activity in right IFG/AI, middle frontal gyrus, and ACC (Hester et al., 2007). Right IFG activation is related to post-error slowing after errors (Marco-Pallarés et al., 2008; King et al., 2010) and after failures to inhibit responding (Li et al., 2008) and lesions of the right inferior frontal sulcus reduced post-error slowing (Molenberghs et al., 2009). A correlation has been found between individual differences in post-error slowing and white matter integrity beneath dorsal ACC regions that are connected to the right IFG (Danielmeier et al., 2011).

In conclusion, post-error processing may be associated with increased post-error accuracy, if conditions are such that post-error processing does not interfere with performance in the post-error trial. There are indications that prolonged processing in the IFG/AI is involved in post-error slowing and either post-error accuracy or post-error interference, depending on response–stimulus timing.

POST-CONFLICT SLOWING

Verguts et al. (2011) recently reviewed and "introduced" the phenomenon of post-conflict slowing. Previous studies suggested that following incongruent trials subjects slow responses on the subsequent trials when task conditions trigger the need for the allocation of attentional control, such as in the case of high conflict, perceptual ambiguity, or difficult tasks (Ullsperger et al., 2005; Bugg, 2008; Verguts et al., 2011). This post-conflict slowing has been interpreted as reflecting a speed–accuracy trade-off (Ullsperger et al., 2005). Indeed, in our study mentioned in the previous section (Tops and Boksem, 2010) more post-conflict slowing correlated with a speed–accuracy bias toward accuracy, as well as with less flanker congruency effect on accuracy (unpublished results).

Because post-conflict slowing is a less-described phenomenon (Verguts et al., 2011), we will present an analysis of this effect from our previous study (Tops and Boksem, 2010). We performed a General Linear Model analysis of reaction times with as within-subject factors flanker congruency, post-incongruency (i.e., previous trial was a congruent vs. incongruent trial). There was a main effect of post-incongruency [$F(1,23) = 42.83$, $p < 0.0001$] showing that reaction times were longer when the previous trial was incongruent ($M = 488$, SD = 64) than when the previous trial was congruent ($M = 478$, SD = 60). A similar analysis

of proportion correct responses found a main effect of post-incongruency [$F(1,23) = 6.01$, $p < 0.05$] showing that proportion correct responses was higher when the previous trial was incongruent ($M = 0.924$, SD = 0.034) than when the previous trial was congruent ($M = 0.911$, SD = 0.044). Post-incongruency increase in accuracy correlated negatively with reaction times ($r = -0.62$, $p < 0.001$)[1].

LONG-TERM GOALS

In proactive systems context models can assist in directing behavior toward long- and short-time goals. In reactive systems goals and motivational stimuli can be held active by redundant attentional and working memory processing and actually lead to perseveration or obsessional behavior and rumination (Tucker et al., 1995; Tops et al., 2010). We adhere to the view that mediodorsal areas implement sustained episodic motivation control over behavioral episodes (Kouneiher et al., 2009), guiding voluntary behavior based on the history of actions and outcomes (Kennerley et al., 2006), and context models. When action outcomes are unfavorable and/or context models suggest it is better to stop the particular endeavor, and do what "experience has taught you is best for you," the endeavor will be abandoned in favor of flexible and adaptive switching to alternative endeavors or exploration. However, adapted to low-predictable environments, the ventrolateral prefrontal cortical controls of reactive systems may persevere on a potential opportunity to exploit (Tops et al., 2010). Maintenance of drive, and retrieval and/or maintenance of goals in working memory may keep goals active over time and in the face of resistance and help to implement effortful control of behavior in the service of long-range goals.

We sometimes use the label "proactive" to refer to the feed-forward and context model-guided action control by the dorsal systems. We think that the labels "proactive" and "reactive" may help us connecting to an audience of social and clinical psychologists. However, the functions of the systems cannot be derived from the labels, and the labels can even cause confusion. Elaborate processing of stimuli in working memory at the expense of subsequent stimuli may not seem "reactive" in some sense. Keeping goals active in working memory seems to be part of what other researchers called "proactive" or goal-directed, and contrasted with reactive, stimulus or cue-driven control (Dosenbach et al., 2007; Braver et al., 2009; Aron, 2011). It is convenient and necessary to sometimes use short labels to express which systems or parts of the model are referred to, but no labels can capture the complexities of the systems, and it is important not to derive system function simply from the labels.

Elsewhere (Tops et al., 2010) we discussed how the combination of a reactive approach system, a reactive avoidance system, and a proactive or context model-guided system produces a model that is very similar to a model that has recently been proposed to integrate literatures on temperament and self-regulation systems, neuromodulatory function of serotonin, psychopathology, and neuroimaging studies of dorsal vs. ventral cortical function (Carver et al., 2008, 2009). The model of Carver et al. was inspired by the work of Mary Rothbart and Michael Posner on the development of attention systems. We discussed the advantages of our approach, and the need to distinguish between the dorsal system on the one hand, and the controls that developed during evolution in each of the ventral and dorsal systems on the other hand. Related to this, we argued against the meaningfulness of an emotion–cognition distinction: although dorsal context model-guidance systems may on first intuition appear more cognitive compared to ventral reactive systems, each of them involves motivation and emotion and controls that continued to develop during evolution. And both dorsal and ventral control areas are implicated in aspects of sustained performance control and task sets (Dosenbach et al., 2007).

Additionally, the ventral cortical systems may have specialized slightly differently in each hemisphere (Tops and Boksem, 2010). We speculate that, at least relatively, in the left hemisphere the ventrolateral pathways involved in cognitive control elaborated to specialize in keeping goals active (reflecting in constraint, persistence, determination; Schiff et al., 1998; Bunge et al., 2003; Gusnard et al., 2003; Whittle et al., 2006; Stuss and Alexander, 2007; Li et al., 2008; Harmon-Jones et al., 2011a; cf. Bernal and Altman, 2009; O'Reilly, 2010), while in the ventrolateral pathways in the right hemisphere cognitive control elaboration specialized in intense attentional absorption in the moment and keeping targets active (Hampshire et al., 2009; Nelson et al., 2010; Higo et al., 2011). This may be why in the left hemisphere the IFG/AI displays stronger connections with dorsolateral prefrontal cortex and bilateral supplementary motor area while in the right hemisphere the IFG/AI displays stronger connections with the rostral ACC, STG, and occipital cortex (Cauda et al., 2011).

ORIENTING AND IFG/AI TOP-DOWN CONTROL REFLECTED IN fMRI, EEG, AND ERPs

IFG/AI TOP-DOWN CONTROL REFLECTED IN fMRI

The right IFG/AI may have an alarm/orienting function as part of its critical role in the switching between internally and externally oriented control modes in response to salient stimuli (Sridharan et al., 2008). Moreover, the IFG/AI appears to exert top-down control over sensory areas including STG or sulcus (STS) to coordinate focal elaborate processing of target stimuli (Frye et al., 2010; Leitman et al., 2010; Zanto et al., 2010; Chadick and Gazzaley, 2011; Higo et al., 2011), functional correlations being positive or negative, depending on which stimulus is to be attended, and which ignored (Higo et al., 2011). For example, the results of Leitman et al. (2010) suggested that sensory-integrative processing is facilitated when the stimulus is rich in affective information, yielding increased activation in STS and amygdala. Conversely, when the stimulus is ambiguous, greater evaluative processes are recruited, increasing activation in IFG and IFG–STS connectivity. As another example, functional connectivity analysis of human fMRI data revealed that visual cortical areas (fusiform face area and parahippocampal place area) that selectively process task goal relevant information are functionally connected with the ventral attentional system areas including bilateral inferior frontal junction, whereas those that process irrelevant context information are

[1]Notice that, because reaction time flanker incongruency effects decrease with increasing reaction times (in our study: $r = -0.44$, $p < 0.05$), post-conflict slowing may cause a spurious conflict-adaptation-like effect (i.e., a decreased incongruency effect following incongruent trials).

simultaneously but dissociatably coupled with the "default mode network" part of the dorsal system, which is activated by prospective/retrospective memory. This indicates that sensory cortical regions are differentially and dynamically coupled with distinct networks on the basis of task goals (Chadick and Gazzaley, 2011).

IFG/AI TOP-DOWN CONTROL REFLECTED IN EEG AND ERPs

The central role of IFG/AI in switching between networks and exertion of top-down control (cf. Menon and Uddin, 2010) by coupling with areas such as STG appears to go together with synchronization of brain rhythms and generation of ERPs.

Midfrontal theta increase has been shown to predict post-error slowing (Cavanagh et al., 2009). Subjects with stronger medial frontal error-related theta also showed stronger white matter connectivity between the medial frontal theta source and the IFG and ventral striatum (Cohen, 2011). A correlation has been found between individual differences in post-error slowing and white matter integrity beneath dorsal ACC regions that are connected to the right IFG (Danielmeier et al., 2011). Similarly, inhibition-related increases in beta band power have been shown to relate to increased post-error slowing (Marco-Pallarés et al., 2008) and to right IFG activity through intracranial EEG recording (Swann et al., 2009). Top-down control from IFG/AI that increases processing in STS or visual cortices appears mediated by coherence in low-beta (12–14 Hz; Frye et al., 2010) and alpha ranges (8–12 Hz; Zanto et al., 2010). An fMRI/EEG study found that theta-constrained fMRI activation was strongest in the insula, temporal pole, STG, and hippocampus (Sammer et al., 2007). Also alpha activity has been related to activity in the insula in studies combining EEG and fMRI measurements (Goldman et al., 2002; Martinez-Montes et al., 2004). Alpha activity related to activity in the insula, thalamus, and parieto-occipital cortex. The source reconstruction from the EEG spatial signature showed only the parieto-occipital sources, suggesting that the insula participates in the control of brain rhythms that it does not generate itself (Martinez-Montes et al., 2004). It can similarly be hypothesized that the IFG/AI participates in computations leading to ERP components, although it does not, or only to a small extent, participate in the actual generation of such components.

Even when other sources have been found also, the IFG/AI and STG have been linked to several ERPs. Different measuring techniques converge on the IFG and STS being involved in sensitivity of the N400 potential to semantic and emotional incongruency (Maess et al., 2006; Van Petten and Luka, 2006). During a switch task including a go-delay response condition that was performed in an fMRI and an ERP study, neither frontal N2 nor right IFG/AI activity were associated with either task set switching or response delaying *per se*. Instead, both were seen specifically for switching to a mode of response delaying (Swainson et al., 2003). The signal to switch from immediate to delayed responding may be similar to the signaling function of a perceived error during speeded response time tasks to delay the next response until processing of the erroneous trial is finished. Tomographical analyses of the N2 difference observed in conditions of equal go and no-go trial frequency localized N2 to the right IFG/AI and dorsolateral prefrontal cortex (Lavric et al., 2004). Error positivity (Pe) activity after about 300 ms that may relate to awareness of errors has been hypothesized to reflect activity in IFG/AI (Ullsperger et al., 2010). A recent MEG study found the late positive potential, which is sensitive to stimulus saliency, and Pe, to share similar STS sources (Helenius et al., 2010). The ventral cortical attentional saliency network involving IFG/AI, STG, temporo-parietal junction, and inferior parietal lobe, is sensitive to stimulus novelty and is the neural basis of the P3 potential response to novelty (e.g., Horn et al., 2003; Mulert et al., 2004; Gómez et al., 2008; Li et al., 2009), and fMRI regions that correlate with the amplitude of the P3 are insula, thalamus and right medial frontal gyrus (Horovitz et al., 2002). The stimulus-preceding anticipatory relatively right-lateralized negative potential that precedes aversive stimuli and feedback and seems to depend on its affective–motivational properties, was localized into the IFG/AI (Böcker et al., 1994; Lavric et al., 2008; Kotani et al., 2009; see also Brunia et al., 2000; Stern and Mangels, 2006). Interactions between the IFG/AI and STG have been implicated in the generation of the mismatch negativity (Opitz et al., 2002; Doeller et al., 2003). Occasional tone omissions elicited a significant increase in right STG activity 140 ms after the omitted stimulus, followed 60 ms later by right IFG activity (Tse et al., 2006). The functional relationship of STS and IFG is consistent with both the contrast enhancement and response inhibition accounts of IFG activity in passive deviance detection (Tse and Penney, 2008). Finally, face stimuli trigger a vertex positive potential (VPP, P150)/N170 component of the ERP. There is strong evidence that the fronto-central VPP and occipito-parieto-temporal right-hemisphere-dominant N170 components can be accounted for by the same dipolar configuration, and the components show identical functional properties (Joyce and Rossion, 2005). The component has been source localized to the STS, with relative right lateralization (Itier and Taylor, 2004; Conty et al., 2007) as well as correlated with activity in the STS in a fMRI study (Horovitz et al., 2004).

IFG/AI TOP-DOWN CONTROL REFLECTED IN ERROR-RELATED ERPs

ERP studies have revealed a neural response to errors that has been termed the ERN or error negativity (Ne; Falkenstein et al., 1990; Gehring et al., 1990). The ERN/Ne is a negative ERP with a fronto-central scalp distribution, peaking 60–110 ms after an error response and is thought to be generated by the ACC.

The IFG/AI may also be involved in the occurrence of the ERN/Ne and Pe, ERP components that have been hypothesized to reflect partial phase-locking of intermittent theta-band EEG activity (Luu et al., 2004). The degree of right AI activation has been related to trait anxiety and the probability of selecting a "safe" response following a punished response (Paulus et al., 2003). A study that allowed subjects to reject trials to avoid errors, found error-specific responses only in bilateral AI (Magno et al., 2006). It is thought that the ERN/Ne is evoked by phasic reductions in DA striatal input in response to non-reward, i.e., "reward overprediction error" or "temporal difference prediction error." An fMRI study found that reward overprediction error produced decreased ventral striatal activation, consistent with prediction error theory; in addition, increased activation was found in the right IFG/AI and right ACC (Abler et al., 2005). Left IFG/AI were active during expectation of reward. The right AI has been shown to encode aversive cue-related prediction errors during Pavlovian learning

of physical punishment (Seymour et al., 2004). Similarly, a recent pharmacological fMRI study showed that, during instrumental learning, reward prediction error was positively related to activity in the ventral striatum and posterior putamen, whereas during loss trials an aversive prediction error-related to activity in right AI (Pessiglione et al., 2006). Pharmacologically enhanced dopaminergic activity improved choice performance toward monetary gains but not avoidance of monetary losses, suggesting that the AI is involved in a non-dopaminergically modulated mechanism of aversive stimulus value processing during avoidance learning. A similar dissociation has been found between anticipatory ventral striatum activity related to subsequent financial risk taking and gain-seeking mistakes, vs. anticipatory AI activity related to subsequent risk avoidance and loss-aversion mistakes (Kuhnen and Knutson, 2005).

Indeed, the AI, together with the ACC, is the only prefrontal cortical area that densely projects to the striosomes in the striatum (Eblen and Graybiel, 1995) that are thought to be involved in reward prediction error calculations and the generation of the ERN/Ne in the ACC (Holroyd and Coles, 2002). Thus, neural activity in the IFG/AI could drive the activity of the mesolimbic dopamine system, which would then be reflected back to the ACC and other areas. Given the association of the ERN/Ne with punishment sensitivity, anxiety, and worry (see Boksem et al., 2006; Tops and Boksem, 2011), the focus in the literature on possible relationships between the ERN/Ne and reward prediction error but neglect of possible relationships with punishment prediction error is surprising. There appear to be physiological differences between selecting actions to achieve rewards and selecting actions to avoid losses (Pessiglione et al., 2006). Lesions involving the AI decrease or abolish ERN/Ne amplitude, and when involving the peri-insular white matter, disrupting connections to motor ACC, and the striatum, severely impair error corrections (Ullsperger and von Cramon, 2006).

To summarize, fMRI, EEG, and ERP studies converge on a role of IFG/AI in top-down control over motor as well as sensory areas.

CONCLUSION

We reviewed evidence for a model in which the IFG/AI is involved in elaborate attentional and working memory processing and we presented the hypothesis that this processing may take different forms and may have different effects, depending on the task at hand: (1) it may facilitate fast and accurate responding, or (2) it may cause slow responding when prolonged elaborate processing is required to increase accuracy of responding, or (3) it may interfere with accuracy and speed of next-trial (for instance, post-error) performance when prolonged elaborate processing interferes with processing of the next stimulus. We presented our viewpoint that ventrolateral corticolimbic control pathways that include the IFG/AI, and mediodorsal corticolimbic control pathways that include dorsal ACC areas, are interacting but partly separable by their respective adaptations to environmental conditions that differ in the level of predictability (Tops et al., 2010). In the brain ventral corticolimbic control pathways have specialized in reactive behavioral control that incorporates fast associative learning that is adaptive in low-predictable environments. This contrasts to dorsal control pathways that specialized in guiding behavior proactively by context models in long-term memory that are formed and kept stable by slow learning, which is adaptive in high-predictable environments. The reactive systems produce a momentary, immediate sense of awareness, an experience of emotional stimuli as being close in time and space. In our opinion, both control pathways continued to develop during evolution, and both developed their own "cognitive controls," such that neither one can be properly described as purely "cognitive" nor "emotional." We speculate that IFG/AI may exert top-down control simultaneously over motor and sensory cortices to facilitate tight sensory guided feedback control of fine motoric ongoing actions in the context of a goal that is held active simultaneously (high-constraint control that may involve left hemisphere specialization; Tucker et al., 1995). Additionally, IFG/AI may facilitate sensory processing while preparing target-triggered responses, a type of control that may involve right hemisphere specialization. We discussed literature that suggests that IFG/AI function in top-down control modulates cortical rhythms and ERPs. Together, the literature suggests that the IFG/AI is an important node in brain networks that control cognitive and emotional processing and behavior.

Increasing attention to the role of IFG/AI in cognition and emotion could have important implications for research on mental health and psychopathology. For instance, the IFG/AI is among the areas that most consistently show increased activity related to anxiety and stress (see for a meta-analysis Etkin and Wager, 2007; for discussion Tops and Boksem, 2011). Also, antidepressant effects of medication and sleep deprivation were correlated with an activity shift from IFG/AI toward dorsolateral prefrontal cortex (Wu et al., 2008). A more global shift in activity from ventral toward dorsal cortical systems related to antidepressant effects has been discussed elsewhere, and may reflect difficulty to disengage from problems and rumination which goes at the expense of positive prospective and retrospective memory in depression (Tucker and Luu, 2007; Carver et al., 2008; Tops et al., 2010).

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"What's that?" "What went wrong?" Positive and negative surprise and the rostral–ventral to caudal–dorsal functional gradient in the brain

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Medial prefrontal cortical (mPFC) functions may be aspects of ventral or dorsal control pathways, depending on the position along a rostral–ventral to caudal–dorsal gradient within medial cortex that may mirror the pattern of interconnections between cortex and striatum. Rostral–ventral mPFC is connected to ventral striatum and posterior cingulate cortex/precuneus are connected with dorsal striatum. Reentrant ventral (limbic), central (associative), and dorsal (motor) corticostriatal loops pass information from ventral-to-dorsal striatum, shifting hedonic processing toward habitual action. Splitting up unexpected occurrences (positive surprise) from non-occurrences (negative surprise) instead of splitting according to valence mirrors the importance of negative surprise in dorsal habitual control which is insensitive to the valence of outcomes. The importance of positive surprise and valence increases toward the rostral–ventral end of the gradient in mPFC and ventrolateral prefrontal cortex. We discuss paradigms that may help to disentangle positive from negative surprise. Moreover, we think that the framework of the functional gradient may help giving various functions in mPFC their place in a larger scheme.

Keywords: cognitive control, predictability, anterior cingulate cortex, inferior frontal gyrus, prediction error

In a recent issue of Nature Neuroscience, Alexander and Brown (2011) presented a computational model that suggests that seemingly diverse medial prefrontal cortex (mPFC) or dorsal anterior cingulate cortex responses may be explained by a single construct, "negative surprise," which occurs when actions do not produce the expected outcome. The simulation results demonstrated that a single term, not reflecting the valence of the outcome, but reflecting the surprise related to the non-occurrence of a predicted event, can capture a broad range of cognitive control, and performance monitoring effects from various research methodologies. The negative surprise signals consist of rich and context-specific predictions and evaluations.

The model of Alexander and Brown seems compatible with a theory we recently proposed (Tops et al., 2010; Tops and Boksem, 2011). In short, we proposed that two control pathways developed during evolution. The dorsal pathway, including mPFC, dorsolateral prefrontal cortex, posterior cingulate cortex, and precuneus, was adapted to control learning and behavior in high-predictable and stable environments. These systems control behavior in a prospective, *feedforward* fashion, guided by context models; models that are formed in long term memory by the predictability of the environment/context and kept stable by slow learning. The ventral pathway, including ventrolateral prefrontal cortex [inferior frontal gyrus (IFG) and anterior insula], was adapted to learning and behavior in low-predictable environments. In low-predictable environments, effective context models can not be formed nor used to control behavior in adaptive ways. Instead, behavior is guided *reactively* by momentary feedback control by environmental stimuli. Ventral corticolimbic control pathways incorporate fast associative learning that is adaptive in low-predictable environments.

Our theory seems related to the well-known hypothesized distinction between ventral and dorsal posterior visual processing streams specialized respectively in processing of "what" and "how" information (Goodale and Milner, 1992). According to Goodale and Milner, the dorsal pathway extracts visual signals relevant for driving automatic or feedforward motor behavior (perception for action), whereas the ventral pathway extracts information relevant for identification and other forms of semantic knowledge. It has been suggested that these processing streams are extended toward frontal ventral and dorsal areas that feed back to implement cognitive control (Sakagami et al., 2006; O'Reilly, 2010). Ventrolateral prefrontal cortex (IFG) mediates active maintenance of stimulus information, and this produces a top-down biasing effect to drive selection and retrieval dynamics in posterior cortex. On the other hand, one should expect to see dorsolateral prefrontal cortex activation whenever the dorsal parietal cortex requires extra cognitive control (such as working memory and top-down biasing) to carry out the processing of sensory information to guide action outputs (O'Reilly, 2010; cf. Sakagami et al.,

2006). Although similar, compared to this account our model seems more integrative, associating ventral and dorsal systems with behavioral programs that include motivation, emotion, viscerosensation, and memory (Tops et al., 2010; Tops and Boksem, 2011).

In many situations and for many tasks, dorsal and ventral systems will collaborate and interact in the control of behavior. Alexander and Brown (2011) suggest that negative surprise signals may provide an important reactive control signal to other brain regions to drive a change in strategy when the current behavioral strategy is no longer appropriate. Interestingly, in the same issue of Nature Neuroscience, Thiebaut de Schotten et al. (2011) reported evidence for connections between the parietal component of the ventral attentional network and the prefrontal component of the dorsal network, especially in the right hemisphere. This and other findings (Sridharan et al., 2008) suggest that inputs from right IFG may modulate dorsal networks, redirecting goal-directed, or internally oriented attention mediated by dorsal networks to events identified as salient by the ventral network. Additionally, support has been found for interactions between IFG and dorsal frontal areas, where IFG implements reactive immediate action according to the information conveyed by concomitant input signals, while dorsal frontal areas implement episodic motivation control: sustained control over behavioral episodes according to information conveyed by temporally remote contextual events (Kouneiher et al., 2009).

Functions of mPFC areas may be aspects of ventral or dorsal control, depending on the position along a rostral–ventral to caudal–dorsal gradient within medial cortex. A rostral–ventral to caudal–dorsal gradient was recently found in mPFC, displaying a functional shift from responding to events (errors) of a kind that may trigger the need for "manual" momentary feedback guided control and learning, via feedforward control learning to action selection aspects of more automated action control (Nee et al., 2011). This gradient may mirror the pattern of interconnections between cortex and striatum, as IFG and rostral–ventral mPFC are connected to ventral striatum and posterior cingulate cortex/precuneus are connected with dorsal striatum. Reentrant loops through the ventral striatum terminate in regions of prefrontal cortex that are more dorsal than where they begin, forming ventral (limbic), central (associative), and dorsal (motor) corticostriatal loops through which information can pass from ventral striatum forward into dorsal striatum, and this shift from ventral-to-dorsal striatum is associated with a shift from hedonic processing toward automated, non-hedonic habitual action control (see **Figure 1**; Alcaro and Panksepp, 2011). High density electroencephalographic source modeling suggests that this shift is also seen in the slow wave during sleep, which propagates from IFG through mPFC to posterior cingulate cortex and precuneus (Murphy et al., 2009).

Information about the integration between striatal regions is obtained from examination of results from multiple retrograde and anterograde tracing experiments, which demonstrate an interface between ventromedial (limbic), central (associative), and dorsolateral (motor) striatal regions via the midbrain dopamine cells (ventral tegmental area and substantia nigra) as well as via cortical areas, that forms an ascending spiral between regions (Haber et al., 2000; cf. Joel and Weiner, 2000). The ventromedial striatum influences the central striatum, and the central striatum influences the dorsolateral striatum. This anatomical arrangement creates a hierarchy of information flow and provides an anatomical basis for the limbic/cognitive/motor interface via the midbrain and cortex (Haber et al., 2000; Joel and Weiner, 2000). Functional connectivity between the cortex and the striatum in a

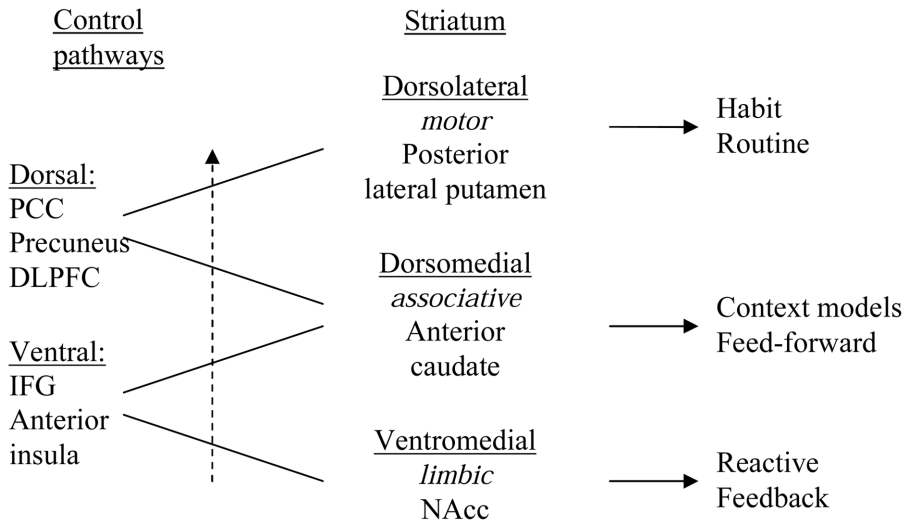

FIGURE 1 | Schematic representation of an interface between ventromedial (limbic), central/dorsomedial (associative), and dorsolateral (motor) striatal regions, via ventrolateral corticolimbic and dorsomedial corticostriatal control pathways and via the midbrain dopamine (DA) cells (ventral tegmental area and substantia nigra, not shown), that forms an ascending spiral between regions. The ventromedial striatum influences the dorsomedial striatum and the dorsomedial striatum influences the dorsolateral striatum. IFG, inferior frontal gyrus (ventrolateral prefrontal cortex); PCC, posterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; NAcc, nucleus accumbens/ventral striatum.

meta-analysis of 126 published functional neuroimaging studies in humans confirmed this pattern (Postuma and Dagher, 2006), as did recent studies (Di Martino et al., 2008; Zhang and Li, 2012).

A similar functional subdivision has previously been proposed between emotional rostral–ventral and cognitive caudal–dorsal (midcingulate) mPFC (Devinsky et al., 1995; Bush et al., 2000). Overviewing the whole cingulate cortex and its striking dichotomy in structure and function, it has been suggested to participate in two distinct parts of the limbic system. A rostral–ventral part engaged in executive functions including those associated with affect and a caudal part involved in visuospatial and memory functions. In this context the rostral limbic system includes the anterior cingulate cortex and ventral striatum, and further the amygdale and septum, anterior insula, IFG, and orbitofrontal cortex. The caudal limbic system includes the posterior cingulate cortex and dorsal striatum, and the hippocampus, and posterior parietal, posterior parahippocampal cortices. Midcingulate cortex would be a transitional region in this conceptualization of two limbic systems (Devinsky et al., 1995).

Functionally, depending on the nature of the events within the learning task, the ventral-to-dorsal shift is seen when learning proceeds from manual, environmental feedback control to more feedforward control and eventually habitual control. Ventral controls "assume" unpredictability (Tanaka et al., 2006) and do not predict temporally distant outcomes. Dorsal controls "assume" predictability and initiate outcome prediction learning. At the same time, monitoring of outcome prediction learning enables estimation of the predictability of the outcome (instrumental contingency), and this calculation of instrumental contingency may be the basis of the subjective experience of control and of selection of more or less automatization for optimal control (Liljeholm et al., 2011). Relatedly, individuals with the greater effect of volatility of predictor–outcome relationships in the mPFC had a higher mean learning rate, and therefore gave more weight to the most recent piece of information (Behrens et al., 2007). The central position of mPFC between reactive ventral control and automated or context model-guided dorsal control suggests a pivotal role in control of learning and behavior, as an interface between the ventral and dorsal systems. Similarly, Luu et al. (2011) suggest that framing the role of the dorsal anterior cingulate cortex as early context-formation to temporarily guide actions, permitting current context to guide learning of new responses in similar situations while supporting the gradual context-updating process that must occur to support skilled performance, may serve as a generic theoretical model that subsumes more specific contemporary theories of mPFC function.

Our theory may explain why, according to the model of Alexander and Brown (Egner, 2011), surprise signals are split up as a function of whether they correspond to unexpected occurrences (positive surprise) or non-occurrences (negative surprise) of action outcomes. Alexander and Brown (2011) concede that positive surprise signals may also be seen in mPFC, but it does not follow from their model why population activity in this region should nevertheless be dominated by negative surprise signals (Egner, 2011). Our theory suggests an explanation. In unpredictable environments, potential threats and rewards are detected through unexpected salient, positive surprise stimuli ("What's that?"). In contrast, in predictable environments, cognitive control of feedforward, habitual, and/or context model-guided action involves detecting when actions do not produce the predicted outcome ("What went wrong?"). Feedforward action control may further be facilitated by context-specific "predictions and evaluations of multiple likely outcomes that provide a basis for evaluating candidate actions and decisions before execution" (Alexander and Brown, 2011).

The present analysis predicts that Alexander and Brown's (2011) suggestion of special importance of negative surprise may be true for dorsal control areas where negative surprise reflects the monitoring of habitual action control. Indeed, splitting up of action outcomes according to whether they are unexpected occurrences (positive surprise) or non-occurrences (negative surprise) instead of according to valence, may reflect the importance of negative surprise in dorsal habitual control, as this control is insensitive to the valence of the outcome (Alcaro and Panksepp, 2011). The importance and occurrence of positive surprise as well as valence signals may increase toward the rostral–ventral end of the gradient in mPFC function and toward the IFG. However, negative surprise and positive surprise are correlated – if a strongly predicted event fails to occur (high negative surprise), it follows that the event that *did* occur will also be very surprising (high positive surprise). We will discuss a few paradigms, such as contingency and instrumental contingency learning (Liljeholm et al., 2011), that may help in disentangling positive from negative surprise.

Our theory suggests that different brain areas should control behavior in future- vs. present moment-focused ways depending on the stability and predictability of the environment. There is some support from human fMRI studies. A study of reward prediction at different time scales showed graded maps of time scale within the right IFG–insula and the striatum: ventroanterior regions were involved in predicting immediate rewards and dorsoposterior striatal regions (and dorsolateral prefrontal cortex, posterior cingulate cortex) were involved in predicting future rewards (Tanaka et al., 2004). A follow-up study showed that the different learning systems in corticostriatal loops are sensitive to the predictability of the environment: the IFG–ventral striatum loop is involved in action learning based on the present state, while the dorsolateral prefrontal cortex–dorsal striatum loop is involved in action learning based on predictable future states (Tanaka et al., 2006).

Another study in monkeys suggests dissociation between prefrontal cortical areas, in which orbitofrontal neurons dynamically evaluate current choices relative to recent choice values, whereas mPFC neurons encode choice predictions and prediction errors using a common valuation currency reflecting the integration of multiple decision parameters (Kennerley et al., 2011). Notice that the evaluation of current choices relative to recent choice values facilitates the detection of stimuli that are motivationally important when action is controlled by a system using only recent information, i.e., proximate to the present moment. The deviation from recent values can be regarded a positive surprise stimulus.

The subjective experience of control or predictability varies with the level of instrumental contingency (Liljeholm et al., 2011), which is the number of contingent outcomes (expected minus negative surprise outcomes) minus the number of non-contingent

(positive surprise) outcomes. Liljeholm et al. (2011) found that positive surprise was associated with right IFG and dorsomedial striatum activation. In contrast, contingent outcomes were associated with mPFC and dorsomedial striatum activation. The composite measure of instrumental contingency appeared to be associated with mPFC, middle frontal gyrus, and inferior parietal lobe activation. Notice that in this study the inverse of negative surprise (the proportion of action-following outcomes that were contingent) was actually associated with mPFC activation.

We describe the posterior cingulate cortex and precuneus, which is connected to the dorsolateral striatum and dorsolateral prefrontal cortex, as the dorsal endpoint of the rostral–ventral to caudal–dorsal gradient within medial cortex, and mediators of dorsal context model-guided control. This may seem at odds with proposals that these areas are important in self-reflection and central parts of the default mode network that is active at rest. However, it may be important to realize that self-reflection may be possible only at rest, and when performing habitual actions, as in both states attention is not involved in other processes. Self-reflection may involve activation of memories of the self in contexts (context models). Although during self-reflection retrieval and action control may partly dissociate, true context model-guided control may involve activation of currently relevant context models that facilitate behavioral control guided and informed by previous experiences, involving automated and habitual behaviors. The default mode network has been proposed to support an ability to perform internal mentation by providing a platform for putting together dynamic mental models and scenarios that are largely detached from the specific or current external world (Buckner and Carroll, 2007). Typically, these scenarios would contain elements of auto-biographical episodic memory and self-related prospective thoughts. Further, it has been suggested that the purpose for a continuously on-going internal mentation process is to act as a simulator and predictor of future events that builds upon previous experiences.

Negative surprise is a different concept than negative prediction error in the classical reinforcement learning approach that has previously been applied to understand mPFC function. In our approach as well as in the model of Alexander and Brown (2011), positive and negative surprise are unrelated to valence and can be positively correlated. In contrast, positive and negative reward prediction error in the classical reinforcement learning approach are negatively correlated and form a dimension of valence (Matsumoto et al., 2007; Kennerley et al., 2011). Recently, a valence-based model has been proposed that contains units coding for the value of cues (stimuli or actions) and units coding for the differences between such values and the actual reward (prediction errors; Silvetti et al., 2011). The model reproduced the mPFC behavior of previous single-unit, EEG, and fMRI studies on reward processing, error processing, conflict monitoring, error-likelihood estimation, and volatility estimation, unifying the interpretations of the role performed by the mPFC in some aspects of cognition. It will have to be determined whether this model relates to our theory and how it compares to the model of Alexander and Brown. Moreover, we think that the framework of dorsal and ventral controls and the functional gradient associated with it, may help in giving various functions in mPFC their place in a larger scheme.

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Threat but not arousal narrows attention: evidence from pupil dilation and saccade control

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It has been shown that negative affect causes attentional narrowing. According to Easterbrook's (1959) influential hypothesis this effect is driven by the withdrawal motivation inherent to negative emotions and might be related to increases in arousal. We investigated whether valence-unspecific increases in physiological arousal, as measured by pupil dilation, could account for attentional narrowing effects in a cognitive control task. Following the presentation of a negative, positive, or neutral picture, participants performed a saccade task with a pro-saccade versus an anti-saccade instruction. The reaction time difference between pro- and anti-saccades was used to index attentional selectivity, and while pupil diameter was used as an index of physiological arousal. Pupil dilation was observed for both negative and positive pictures, which indicates increased physiological arousal. However, increased attentional selectivity was only observed following negative pictures. Our data show that motivational intensity effects on attentional narrowing can occur independently of physiological arousal effects.

Keywords: arousal, focused attention, valence, pupil dilation, anti-saccade task

INTRODUCTION

In order to cope with threatening events, organisms often recruit extra resources. Regarding cognitive resources, there is evidence that affectively negative stimuli immediately prioritize the perceptual processing (Öhman et al., 2001) and recall (Christianson, 1992) of related information at the cost of other processes (Bocanegra and Zeelenberg, 2009; Pessoa, 2009), and it has been argued that these effects are mediated by the organism's current state of arousal (Schimmack, 2005). According to Easterbrook's (1959) influential hypothesis, increased arousal may lead to the narrowing and focusing of attention, thus facilitating appropriate subsequent responding and coping behavior.

Although threatening events and stress have been demonstrated to narrow attention (Cohen, 1980; Chajut and Algom, 2003; Gable and Harmon-Jones, 2010a; for a discussion of opposite effects in trait anxiety, see Pacheco-Unguetti et al., 2010), it is not clear whether these observations are due to non-specific arousal or the activation of affect-specific emotional/motivational systems (Bradley, 2000). Even though Easterbrook's original hypothesis relates to unpleasant situations only, several authors have suggested that any increase of arousal – e.g., whether induced by caffeine ingestion or impulsivity traits – may modulate attentional selectivity (e.g., Anderson, 1990). Along similar lines, increasing motivational intensity has been reported to increase focused attention irrespective of the motivational system (approach versus avoidance) involved (Gable and Harmon-Jones, 2008, 2010a,b; Harmon-Jones and Gable, 2009). However, whether arousal can be conceived of as a unitary construct has been questioned (Lacey, 1967; Neiss, 1988, 1990) and it is not entirely clear how motivational intensity and arousal are conceptually related (cf. Gable and Harmon-Jones, 2010b). Accordingly, it remains to be shown whether the emotional modulation of the selective attention reflects non-specific arousal that can vary orthogonally to the valence of the present affective state or whether it is specific to negative, potentially threatening events.

To index a possible narrowing of the attentional focus we used a visual anti-saccade task (for a review, see Hutton and Ettinger, 2006). This task is typically used as a measure of cognitive control, which is closely related to attentional selectivity. That is, the amount of information that is entering the focus of attention may be limited by cognitive control exerted at a perceptual or more central processing level (Desimone and Duncan, 1995; Miller and Cohen, 2001; Pessoa et al., 2003). Moreover, given that the neural mechanisms underlying the anti-saccade task are well known and can easily be studied in monkeys as well (cf. Munoz and Everling, 2004), we considered this task to be particularly well suited for studying the neuro-cognitive effects of emotion on attentional selectivity. As a first step, our study aimed at disentangling the relative contributions of arousal and affective valence on attentional control using physiological and behavioral measures of arousal and focused attention, respectively.

In the anti-saccade task, participants are presented with a peripheral, emotionally neutral target stimulus that appears with an abrupt onset on the left or right of the central fixation. Depending on the instruction, they are to move their eyes either to this target (pro-saccade condition) or to the opposite side of the display (anti-saccade condition). The common finding is that saccades are initiated more slowly and less reliably in the anti-saccade than in the pro-saccade condition. This cost is commonly attributed to the automatic tendency to look at novel events, which requires active inhibition in the anti-saccade condition (Olk and Kingstone, 2003). Because improved attentional control decreases automatic

capture by the target stimulus (Yantis and Jonides, 1990), reflexive saccades toward the stimulus become suppressed. Thus, we expected that manipulations improving focused attention reduce the size of the latency costs, with anti-saccades becoming faster and pro-saccades becoming slower (cf., Kristjansson, 2007).

Affective states were induced prior to each saccade-task trial using positive, negative, or neutral pictures from the International Affective Pictures System (IAPS). Valence and arousal ratings of these pictures show a quadratic relationship, such that positive and negative stimuli are typically highly arousing and neutral stimuli low arousing (Lang et al., 2008). To ascertain that the pictures induced a physiological response we used pupillometry. Recent work by Bradley et al. (2008) has validated this approach. In that study, both negative and positive IAPS pictures were shown to produce pupil dilation, a response reflecting emotional arousal which is associated with increased sympathetic nervous activation. By means of this setup we were able to contrast two competing hypotheses. If more attentional selectivity in affectively laden circumstances would be driven by non-specific arousal, the difference in saccadic reaction time (RT) between anti- and pro-saccades should be reduced following negative as well as positive arousing pictures as compared to the non-arousing neutral pictures. Alternatively, if more attentional selectivity is specific to threatening situations, this latency cost should be reduced following negative stimuli but comparable for positive and neutral stimuli.

MATERIALS AND METHODS

PARTICIPANTS

Eleven students from Leiden University (18–22 years old; two males; one left-handed) participated for either payment (5 Euros) or course credits.

MATERIALS

Thirty-two highly arousing negative, 32 neutral, and 32 highly arousing positive pictures were selected from the IAPS set (Lang et al., 2008[1]). The stimulus set was almost identical to the one used by Bradley et al. (2008). Like that study, negative and positive stimuli could be differentiated on the basis of valence IAPS ratings, whereas they were matched for arousal IAPS ratings (Lang et al., 2008, see **Table 1**). Neutral pictures had low arousal ratings and intermediate valence ratings. In order to avoid light reflex confounds we used gray-scaled pictures (cf. Bradley et al., 2008); brightness and contrast were adjusted to ensure identical mean luminosity values for all pictures.

TASK

Each trial started as soon as participants had successfully looked at the central fixation cross for at least 1 s. Then an IAPS stimulus appeared for 500 ms, which was replaced by the fixation cross for a jittered interval ranging from 1500 to 2500 ms. Following a 200-ms blank gap (cf. Everling and Fischer, 1998), the target stimulus (also a cross) appeared for 500 ms 8° to the left or right to the screen center. Then the central fixation cross appeared for another interval (ranging from 1000 to 2500 ms) before the next trial started. At the beginning of each block an 8-s verbal cue (approximately 5.7° × 1.4°; width × height) indicated whether a pro- (target position) or an anti-saccade (mirror position of the target) was to be made to the next target stimulus. The picture (16° × 12°) and the black fixation cross (0.8° × 0.8°) were shown on a gray background with luminosity equal to the mean of the pictures. In rare cases (0.2% of the time), eye tracker recording problems delayed the trial presentation (inter-trial intervals > 9 s). Because the interruption of the ongoing presentation sequence by either delayed presentation or by the onset of a new block is likely to compromise the experimental manipulation of the arousal and valence, the first five trials after such events were excluded.

Table 1 | Emotion and performance measures as a function of picture content (table shows means and SE between brackets).

	Picture content					
	Negative		Neutral		Positive	
SELF REPORT						
Valence rating	2.4	(0.11)	5.0	(0.11)	7.0	(0.11)
Arousal rating	5.9	(0.16)	3.6	(0.16)	5.5	(0.16)
PHYSIOLOGY						
Pupil diameter (mm)	4.25	(0.040)	4.19	(0.026)	4.23	(0.033)
BEHAVIOR						
Pro-saccadic RT (ms)	196	(8.3)	185	(7.5)	189	(7.4)
Anti-saccadic RT (ms)	257	(12.7)	260	(10.8)	265	(9.8)

PROCEDURE

Participants were instructed to attend to the emotional pictures and to make pro- and anti-saccades to the target as fast and accurate as possible. They were also asked to avoid eye blinks during picture and target presentation. After informed consent and eye tracker calibration, subjects practiced with six pro-saccade and six anti-saccade trials preceded by neutral IAPS pictures and followed by accuracy feedback for 1 s. Calibration and/or practice were repeated in case of eye tracking problems or when the subject did not follow the instructions. The task consisted of six alternating pro-saccade and anti-saccade blocks (counterbalanced order), with two self-paced breaks in between. Each block consisted of 48 trials, and every chosen IAPS picture appeared three times in randomly chosen trials. Participants were debriefed after the experiment.

DATA ACQUISITION AND ANALYSIS

Saccadic behavior and pupil diameter were recorded at 120 Hz using a Tobii T120 eye tracker, which was integrated into a 17-inch TFT monitor. Participants were seated at a distance of approximately 60 cm from the monitor while their head was stabilized by using a chin rest. Artifacts and blinks as detected by the eye

[1]The library numbers for the IAPS stimuli used in the present study are: Negative: 2120, 2205, 2520, 2590, 2691, 2730, 2750, 2800, 3015, 3030, 3053, 3100, 3170, 3180, 3181, 3400, 3500, 3530, 3550, 6210, 6211, 6212, 6821, 6834, 6838, 9041, 9250, 9300, 9341, 9405, 9800, 9921. Neutral: 2020, 2190, 2200, 2210, 2214, 2215, 2220, 2221, 2235, 2240, 2270, 2272, 2278, 2383, 2393, 2410, 2441, 2491, 2493, 2514, 2579, 2620, 2749, 2752, 2810, 2850, 2870, 2890, 3210, 5455, 7550, 9210. Positive: 2208, 2250, 2260, 2501, 2560, 2650, 4611, 4617, 4640, 4650, 4653, 4658, 4659, 4689, 5621, 8041, 8080, 8090, 8116, 8120, 8161, 8180, 8200, 8280, 8300, 8320, 8330, 8370, 8380, 8400, 8420, 8465.

tracker were corrected by using a linear interpolation algorithm. A saccade was considered to begin as soon as the horizontal angle exceeded 2° and speed passed a 30°/s threshold. For all analyses, we excluded the following trials: trials including and following recording-related delays (see above), trials following performance errors, trials with saccadic RT outliers (<80 or >500 ms), and trials where no saccades could be detected. Repeated-measures ANOVAs with the factors picture content (negative, neutral, positive) and task (pro versus anti) were run on pupil dilation and saccadic behavior measures. Paired *t*-tests were used for *post hoc* tests.

RESULTS

PUPIL DILATION

Following Bradley et al. (2008), pupil dilation to the picture content was measured after the initial light reflex. Dilation was defined as the mean pupil diameter in a window from 2 to 2.5 s after picture onset, using a 200 ms pre-picture baseline. As **Table 1** shows, both negative and positive pictures caused dilation in comparison to neutral pictures. Analyses revealed a main effect of picture content [$F(2,20) = 4.74$, $p < 0.05$, MSE = 0.005], independent of task [$F(2,20) = 1.02$ n.s., MSE = 0.003]. Replicating Bradley et al. (2008), planned *t*-tests confirmed that arousing pictures (pooling the positive and negative condition) increased pupil diameter [$t(10) = 2.49$, $p < 0.05$]. As in that study, there was also a trend for negative pictures to induce more dilation than positive pictures [$t(10) = 1.822$, $p = 0.09$]. Using neutral pictures as comparison, separate *t*-tests indicated a significant dilation for negative pictures [$t(10) = 2.487$, $p = 0.032$] and a marginal significant dilation for positive pictures [$t(10) = 1.822$, $p_{\text{one-sided}} = 0.049$].

SACCADIC BEHAVIOR

See **Table 1** for details. As usually found, correct saccadic RTs were slower during anti blocks than during pro blocks [$F(1,10) = 77.08$, $p < 0.001$, MSE = 1073.76]. More importantly, this task effect interacted with picture content [$F(2,20 = 3.82$, $p < 0.05$, MSE = 112.48]. Planned *t*-test showed that the latency cost (anti-RT minus pro-RT) was not reduced for arousing pictures (pooling the positive and negative condition) versus neutral pictures [$t(10) = 1.50$, $p = 0.163$]. Instead, the latency cost was reduced following negative pictures only [$t(10) = 2.84$, $p < 0.02$] in comparison to neutral pictures, but not for positive pictures [$t(10) = 0.21$, n.s.]. As **Figure 1** illustrates, relative to the neutral baseline, negative pictures slowed down pro-saccadic RT [11 ms; $t(10) = 3.34$, $p < 0.01$] but did not significantly speed up anti-saccadic RTs [3 ms; $t(10) = 0.71$, n.s.], whereas positive pictures did not make any reliable difference (4 and 5 ms, respectively, all n.s.).

Task also affected the error rates [$F(1,10) = 17.90$, $p < 0.01$, MSE = 0.025]: subjects committed 18% erroneous saccades in anti-saccade blocks but only 1.5% in pro-saccade blocks. This effect did not interact with picture content [$F(2,20) = 0.97$, n.s., $MSE = 0.006$].

To further test whether arousal might mediate any of these negative emotion effects we re-ran the analyses of correct saccadic RTs with strong versus weak pupil dilation as an additional factor. For this purpose, we categorized the trials following emotional pictures by means of a median split of the corresponding dilation measures. However, even though we replicated the task effect and its interaction with picture content, the dilation factor was not involved in any main effect or interaction (Fs < 1).

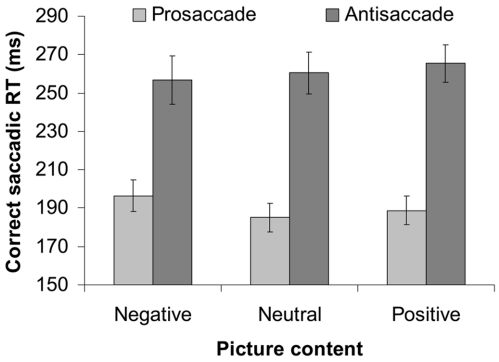

FIGURE 1 | Correct saccadic reaction times as a function of picture content and task context.

DISCUSSION

The aim of our study was to test whether attentional narrowing is due to general arousal or is selectively triggered by negative affective events. Although pupil dilation data confirmed that both negative and positive pictures increased the arousal level – a finding replicating Bradley et al. (2008) – attentional narrowing was observed following negative pictures only. This indicates that attentional narrowing is not caused by emotional arousal *per se*, at least as it can be measured by pupil dilation following the presentation of high-arousing pictures. In other words, increased emotional arousal may be a necessary condition, but it is not a sufficient condition for increased attentional selectivity. The same conclusion is suggested by the lack of impact of pupil dilation in the combined analysis. Hence, our observations do not provide any evidence for a role of arousal in driving attentional narrowing. Instead, the attentional focus seems to narrow whenever individuals are encountering events of negative affective valence.

How may negative affect regulate attentional narrowing? According to one account, dangerous situations may mobilize executive functions that protect against interference from disruption by irrelevant, distracting information (Norman and Shallice, 1986). Neuroimaging studies have suggested that these adjustments in cognitive control are implemented in the prefrontal cortex (Miller and Cohen, 2001), probably via signaling from the anterior cingulate cortex, a brain region involved in the detection of demanding and aversive situations (Botvinick et al., 2001; Shackman et al., 2011). Frontal cortex modulation, in turn, may modulate saccadic eye movements via the basal ganglia (Munoz and Everling, 2004). Thus, the reduced latency costs triggered by the negative pictures may originate from affect-driven modulation of cognitive control. This interpretation also fits earlier work that has used the anti-saccade task to assess inhibitory control (cf. Munoz and Everling, 2004). A similar explanation may also apply to earlier published studies such as effects on Stroop tasks usually attributed to attentional narrowing (e.g., Callaway, 1959; Agnew and Agnew, 1963; cf. Wachtel, 1967).

However, it is important to emphasize that the reduced latency cost with negative pictures was driven by a slowing of RT during the pro-saccade block rather than a speeding of RT during the anti-saccade blocks. This indicates that processes other than improved control may also play a role in the affective modulation of behavior. For example, although it is likely that negative emotions increased control and attentional selectivity, which inhibits the visuo-motor grasp reflex resulting in delayed pro-saccadic RTs (Kristjansson, 2007), this effect may have become attenuated during the anti-saccade blocks. Because a state of high cognitive control is known to attenuate the effects of negative emotions (Ochsner and Gross, 2005), it might be that the effects of emotions on cognitive control were less pronounced in situations of higher task demands. Alternatively, it could be that the possible speeding of anti-saccades is masked by an overall slowing effect induced by the negative pictures. Indeed, several studies suggest that the processing of negative events may compete for perceptual and/or executive resources, which may slow down performance on a subsequent task (e.g., Gehring et al., 1993; Notebaert et al., 2009; Pessoa, 2009; Cohen et al., 2011). It remains an important aim for future studies to disentangle the role of these bidirectional interactions between emotions, perception, and executive function (cf. Vuilleumier et al., 2003).

Our study demonstrates for the first time that increased emotional arousal is not a sufficient condition to produce focused attention: pro-saccadic slowing presumably reflecting attentional narrowing was observed for negative affect, but not for positive affect. Consistent with this finding, and in contrast to a common misinterpretation, Easterbrook's (1959) original hypothesis attributed attentional narrowing not to general arousal but to a drive or motivation to withdraw. Given that positive emotions with approach–motivation have been shown to increase attentional focus (e.g., Gable and Harmon-Jones, 2008; for a review, see Gable and Harmon-Jones, 2010b), it is an important challenge for future research to determine which affective dimension, other than arousal accurately predicts attentional narrowing induced by positive emotions. In line with very recent discussions (cf. Friedman and Forster, 2011; Harmon-Jones et al., 2011), our results imply that it is now time to start research programs that search for emotional dimensions beyond valence and arousal that are responsible for tuning one's attentional scope.

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