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When mothers go wrong: likely neural undercurrents related to poor parenting

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The mammalian model of survival begins with puzzling-out a simple but stark truth: Life must learn to care for life. We have described the experiences involved in changing from nulliparous female to mother, from unresponsive to committed. The transition taking place in the nervous system that underpins the shift from largely self-centered organism to other-focused caregiver is accompanied by an assortment of effects ranging from basic gene expression changes, to modifications of neuronal complexity and activity, to wholesale shifts in the size of specific brain structures. In total, the female changes in ways both subtle and striking, and for one simple reason: reducing the cost:benefit ratio required for successful rearing of young. The system, however, is imperfect. Here we attempt to reconcile the ultimate goals of life and evolution, the manifest reshaping of the female mammal's brain in service to her young, with its incomplete or faulty development. That is, things go wrong; mothers can be apathetic, abusive, or worse. How might the inherent adaptation that, on average, directs the formation of the maternal brain, and which in turn, governs the set of behaviors required for successful reproduction and gene passage, fail to adequately express itself?

Susan Smith drives her car and two young boys into a lake in South Carolina, killing the children. Andrea Yates drowns five children, one after the other, in her family's bathtub. These cold and heinous acts are doubly shocking because of the perpetrators' relationship to the victims: the children's mothers. How, we ask, can that most hard-wired of mammalian behaviors – in fact, the very word "mammal" derives from the act of caring – go awry? Why and in what ways do mothers vary in their degree of maternal motivation/care?

The simple answer is, nothing is perfect. Things go wrong, sometimes terribly so. From the standpoint of the brain's regulation of maternal behavior, however, which of the factors that normally order maternal responsiveness might malfunction? The *fosB* gene is a good place to start.

Brown et al. (1996) established a regulatory role for the *fosB* gene in the display of mouse maternal behavior. A knockout of *fosB* gene activity, including preoptic area (POA) expression, led to a significant reduction in what the authors refer to as "nurturing," wherein a lack of maternal responsiveness affected offspring survival, in the absence of observed deficits in basic hypothalamic activity, pregnancy, cognition, or olfaction. Reduced medial preoptic area (mPOA) neural activity is associated with poor maternal behavior (Numan and Insel, 2003), much as reductions in frontal lobe volume recently reported in human mothers may be related to fewer positive thoughts toward young (Kim et al., 2010). Both may depend, in part, on responding appropriately to offspring-related sensory stimuli. It appears, then, that some more intimate aspect of maternal–offspring interaction may be lacking, a defect which resembles an apparent inability to accurately attend to the offspring's sensory load. Cues that normally elicit maternal behavior from the mother failed to do so in the *fosB* knockouts and, perhaps, failed to cascade onto the otherwise receptive brain. The possibility exists, then, that deficiencies in other maternally related genes (Contino et al., 2007; Ferguson et al., 2008; Kinsley et al., 2008; Mann and Lee, 2010), as well as *fosB* and its human analog – which plays a role in stress responsiveness (Vialou et al., 2010) and which could be related to reactions to young – may be associated with a diminution in maternal responsiveness or interest. Two major research questions arise: Do such genetic effects mean a greater likelihood of neglect or abuse? And is the system similar in humans?

A spate of recent papers describes some of the alterations that define the normal maternal brain. Kim et al. (2010), using magnetic resonance imaging (MRI), reported significant structural changes in several major brain regions of human mothers over the first few postpartum months, during which the intimate relationship between mother and infant develops and deepens. The images of mothers' brains at 2–4 weeks postpartum, and 3–4 months postpartum, showed increased gray matter volumes in prefrontal cortex, parietal lobe, and midbrain areas. Further, increased gray matter volume in the hypothalamus, substantia nigra, and amygdala was correlated with maternal positive perception of the baby (more positive, more gray matter). These results suggest that the first months of motherhood in humans are accompanied by structural changes in brain regions implicated in maternal motivation and behaviors (Kinsley and Meyer, 2010).

Other work is suggestive, too, as it parallels human experiences. Lippmann et al. (2007) reported that chronic maternal separation during the postnatal period induces long-term behavioral and neural modifications in the adult. Such individuals exhibited significant reductions in the level of the protein, brain-derived neurotrophic factor (BDNF) and marked maternal behavioral deficiencies. Francis et al. (1999) and others have shown that rat pups that experience their mothers licking and grooming, are likely to act the same way toward their own offspring, non-genomically passing-on behaviors to subsequent generations. Korosi and Baram (2009) suggest that early childhood deprivation of "maternal love" may lead to variable neuroendocrine stress responses and differential coping. It is of interest, therefore, to hear accounts of childhood stress and abuse in the sad cases of Yates and Smith above.

Such experiences likely change the brain in manifest ways, possibly influencing other cognitive and emotional processes. Keyser-Marcus et al. (2001) have reported that neurons in the mPOA demonstrate a significant increase in volume the closer an animal

gets to parturition, as if the region were readying itself for the requisite maternal responsiveness to follow. Numan and Insel (2003) have discussed at length the role of the mPOA in maternal behavior, and the Kim et al. (2010) data implicate anterior hypothalamic structures such as the mPOA in their "good thoughts-good mother" data mentioned above. A model for likely neural changes that do ***not*** occur in so-called bad mothers would present a valuable research tool to the translational field.

In summary, the data suggest that adequate maternal motivation, far from an intrinsic or instinctual state, is, rather, a consequence of active processes "building" a responsive neural substrate. Therefore, if improperly assembled, an incomplete, or defective maternal brain may fail in its task of caring adequately for young – or worse – making the faulty maternal brain a valuable object for additional study.

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