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Markus Rüther, Bettina Schoene-Seifert, Marco Stier and Sebastian Muders

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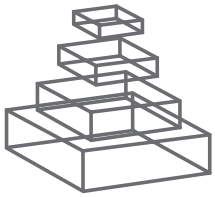

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THE PHILOSOPHY OF PSYCHIATRY AND BIOLOGISM

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Table of Contents

Part III: The Concept of Disease in Psychiatry

Part IV: Phenomenological Considerations

Part V: From Concepts to Consequences

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The philosophy of psychiatry and biologism

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In the philosophy of psychiatry, there has been an ongoing dispute about the capabilities and limits of the bio-natural sciences as a source of methods and knowledge for quite some time now. Still, many problems remain unsolved. This is at least in part due to the regrettable fact that the opposing parties are far too rarely prepared to swap ideas and to try to increase their mutual understanding. On the one hand there are those—psychiatrists as well as philosophers—who maintain a more mentalistic and/or phenomenalistic view of the psyche and its disturbances. On the other hand there are researchers who follow biologically inspired strategies: Since the human mind is something through and through biological, mental diseases, too, can and should be explained and treated biologically. Even though there are examples of fruitful collaboration, in general the split prevails. One often gets the impression that both sides remain in their "trenches", busy with confirming each other's opinions and developing their positions in isolation. Even though there are also examples of fruitful collaboration, the split leads to several shortcomings:

(1) Good arguments and insights from both sides of the debate get less attention than they deserve.
(2) The further improvement of each position becomes harder without criticism, genuinely motivated by the opposing standpoint.
(3) The debate is not going to stop, at least not in the way it would finish after a suggested solution finds broad support.
(4) Related to this, insisting on the ultimate aptness of one side is just plainly wrong in almost every case, since undeniably, most philosophical positions usually have a grain of truth hidden in them.

In sum, many controversies persist with regard to the appropriate methodological, epistemological, and even ontological level for psychiatric explanation and therapies. In a conference which took place in December 2011 in Muenster, Germany, we tried to contribute to a better understanding about what really is at issue in the philosophy of psychiatry. We asked for a possible common basis for several positions, for points of divergence, and for the practical impact of different solutions on everyday work in psychiatry.

The present Frontiers research topic is a fruit of that conference. Since psychiatry is a subject too wide to be covered *in toto*, this research topic collects six target articles, each focusing a particular aspect. They are accompanied by a number of commentaries providing both critical and supportive arguments.

First, Henrik Walter sets the stage by presenting what he calls "the third wave of biological psychiatry" (Walter, 2013). The first two waves were primarily driven by the ambition to uncover the relation between mind and brain and by the integration of genetic insights as well as the upcoming of psychopharmacology. While these where—in a sense—one-sided, the third wave, starting only in the last two decades of the previous century, conceptualizes mental disorders "as brain disorders of a special kind." As Walter explains, they require "a multilevel approach ranging from genes to psychosocial mechanisms." This broader account might be an indication that the alleged reductionism today's biological psychiatry is often accused of is unjustified. Markus Pawelzik doubts in his commentary that the "third wave," as conceived by Walter, has in fact the potential to overcome psychiatry's biologistic thought (Pawelzik, 2013). Michael Noll-Hussong picks up on Walters idea of "waves," arguing that the "sinks" between them have to be taken into account, too, for an adequate understanding of psychiatry's momentum (Noll-Hussong, 2014). He predicts the upcoming of a fourth wave that will arise from the background of information integration theory, using computer-simulations of the mind to increase our understanding of the psyche. In a third commentary Gerhard C. Bukow cautions against using externalist approaches of mental disorders too uncritically (Bukow, 2013). What externalist accounts need, but hardly can provide, are criteria where to stop adding further and further external constituents to the notion of psychiatric disorder.

In the second target article Marco Stier argues against the reducibility of the concept of mental disorder (Stier, 2013). Even if the mental may in principal be reducible to brain functions, mental disorders are not, Stier holds. The reason is that we can only call behavior disordered by comparing it to non-disordered behavior, i.e., by using norms which, in turn, are not reducible to anything physical. Stier's claim has provoked a number of critical replies. Markus Rüther finds several argumentative shortcomings in Stier's account (Rüther, 2014). According to Rüther, Stier's constructivist thesis and the associated anti-reductionism suffer from a lack of argumentative force. Similarly, Sebastian Muders in his comment examines the relation between normativity on the one hand, and non-naturalness, non-objectivity, and relativity, on the other (Muders, 2014). He argues that the normative character of mental disorders does not mean that they are non-natural, non-objective, or relative. Anneli Jefferson

principally agrees with Stier, but adds two aspects regarding normativity and non-reducibility: she holds firstly that the importance of the mental level can be explained independently of value judgments, secondly she points out the need to investigate normativity in any kind of disease or disorder ascriptions, not just in the mental area (Jefferson, 2014). Bettina Schoene-Seifert stresses—in accordance with Jefferson—that values don't come into play only in regard of mental disorder (Schoene-Seifert, 2014). Above that, she warns against mistaking Stier's argument as being one in favor of methodological antireductionism in psychiatry. Peter Hucklenbroich's commentary is on both Stier and Walter, bringing central and well established principles of modern medical pathology to mind (Hucklenbroich, 2014). In particular, he opposes the normativity claim, arguing that the criteria of pathologicity are rooted in nature and not relative to social norms and values.

The third target article by Thomas Schramme is concerned with the autonomy of the concept of disease in psychiatry (Schramme, 2013). On the background of classical ideas from the philosophical debate on the mind-body problem, he argues that denying substance dualism does not force us to adopt a purely materialistic account of the mental. Especially, some psychiatrists' belief that this denial necessarily leads to a neurophysiological account of mental disorder is wrong in his eyes. Even in the absence of any form of substance dualism we still need an irreducible psychological level of explanation. Marcella Rietschel argues in her commentary, contra Stier and Schramme, that mental disorders in actual fact are somatic disorders, as actual scientific insights show (Rietschel, 2014). According to Jan-Hendrik Heinrichs the terms "psychiatric disorder" and "neural defect" belong to different types of analysis and cannot be identified with or reduced to each other (Heinrichs, 2014). While Schramme argues on a more general level, Michael Jungert takes up his view and exemplifies the irreducibility of the mental on the basis of posttraumatic stress disorder (Jungert, 2013). Since an analysis of the internal perspective of a patient is indispensable, neither neuroscience nor any biological psychiatry is able to approach mental disorders appropriately. The final comment in this section is again on Stier and Schramme together. Gerald Ulrich stresses that the currently prevailing "either-or interrogations" are utterly ill-posed (Ulrich, 2014). In the context of aspect dualism regarding mind and body Ulrich recommends to realize that what is at stake is an "as-well-as" issue.

One of the central criticisms against biological accounts in psychiatry is that they disregard the phenomenal perspective of the suffering person. The target article by Kerrin A. Jacobs focuses on this aspect by analyzing "the depressive situation" (Jacobs, 2013). Her approach is a phenomenological one, but one that is informed by empirical research. In depression, she explains, the pre-reflective self-evaluative dynamics of the depressed is significantly altered, leading to impairments of agency. While Jacobs stresses the *pre*-reflective dimension of depression Lara Rzesnitzek in her commentary reminds of the importance the notion of a "self-feeling" had in early theories of depression (Rzesnitzek, 2014). Although her commentary is a "historical note" it nevertheless points to one of the most paramount problems in debates on current biological psychiatry.

The article of Hanfried Helmchen, an experienced practicing psychiatrist, cautions against any dogmatism in psychiatry (Helmchen, 2013b). As history shows, an exclusively biological account of mental disorder is as disadvantageous for the patient as an exclusively social or psychological one. What is needed instead is a biopsychosocial model of mental disorder. In his commentary Marco Stier admits that the integrative account favored by Helmchen is indeed important as a warning sign against misapprehensions of the mental (Stier, 2014). But he assumes nevertheless that it will in effect either lead to explanatory arbitrariness, or end up as a theory that is ultimately biological.

Last not least, the target article by Lara Rzesnitzek shows that some issues debated in today's psychiatry have already an astonishing long history (Rzesnitzek, 2013). An example of this is the "psychosis risk syndrome"—one of the contentious points in the preparation of the fifth revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). Even though attempts of identifying symptoms of a looming schizophrenia are much older, in 1938 "early psychosis" entered the stage as a possible independent diagnosis. Hanfried Helmchen comments on ethical implications of "early psychosis," and adds some remarks to Rzesnitzek's historical description (Helmchen, 2013a). Finally, Nicolas Henckes suggests complementing this historical picture with a sociological view (Henckes, 2014). As he points out, psychiatric diagnoses always have a life of their own outside medicine. This is especially true for politics and the people affected.

Our approach to the research topic "philosophy of psychiatry and biologism" is not intended to be exhaustive. Rather, our aim was to bring together experts of different fields in order to work with—and not against—each other. In this sense, the articles and commentaries of the present volume may serve as a stepping-stone for future cooperation.

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The third wave of biological psychiatry

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In this article I will argue that we are witnessing at this moment the third wave of biological psychiatry. This framework conceptualizes mental disorders as brain disorders of a special kind that requires a multilevel approach ranging from genes to psychosocial mechanisms. In contrast to earlier biological psychiatry approaches, the mental plays a more prominent role in the third wave. This will become apparent by discussing the recent controversy evolving around the recently published DSM-5 and the competing transdiagnostic Research Domain Criteria approach of the National Institute of Mental Health that is build on concepts of cognitive neuroscience. A look at current conceptualizations in biological psychiatry as well as at some discussions in current philosophy of mind on situated cognition, reveals that the thesis, that mental brain disorders are brain disorders has to be qualified with respect to how mental states are constituted and with respect to multilevel explanations of which factors contribute to stable patterns of psychopathological signs and symptoms.

Keywords: mental disorder, cognitive neuroscience, neuroimaging, genetics, philosophy of mind, philosophy of psychiatry, RDOC, DSM-5

WHAT IS BIOLOGICAL PSYCHIATRY?

As a first approximation we can say that it ties psychiatry closely to the biology of the brain. Under such a broad characterization today nearly everyone would qualify as a biological psychiatrist, as only very few would deny such a connection. However, there are stronger and more controversial claims, for example the ontological claim that psychiatric disorders are disorders of the brain, or, on the therapeutic level, that the best therapies are biological ones like medication or deep brain stimulation. However, many biological psychiatrists would not share these stronger claims, so this characterization seems too narrow.

To better understand the characteristics of the third wave, it will be helpful to take a short look at the first and second wave in the history of psychiatry (Shorter, 1998). The first wave in the second half of the nineteenth century can be best understood as a new research agenda. It was not so much characterized by the idea that the mental and the nervous system are closely linked – this was already believed by ancient philosophers – but rather by the ambition to uncover the relation between mind and brain by doing systematic research linking neuropathology and mental disorder and by using the experimental method in animals and humans. Wilhelm Griesinger (1817–1868), one of the most important figures of this first wave, famously declared: mental disorders are disorders of the brain. Note, that this was not primarily intended as a reductionist claim, but rather as a statement intended to delineate his ideas against the two prevailing approaches of that time: the moral approach on the one hand, and the somatic approach, linking mental disorder to body processes in the lung, liver or other organs, on the other hand. Nevertheless, Griesingers claim was not at all uncontroversial as theorists felt that such a brain approach would not do justice to the intricate psychopathological phenomena psychiatrists dealt with. For example, Karl Jaspers, the philosopher-psychiatrist, called 1913 the localationist models of two main protagonists of the first wave, Theodor Meynert and Carl Wernicke, "brain mythologies."

In the early twentieth century, there was a decline in the biological approaches through various developments. Emil Kraeplin, one of the most influential psychiatrists at his time, started as an opponent to biological psychiatry, and developed his diagnostic system on systematic observations of symptoms and course of mental disorders, laying the groundwork for the later DSM. Also, psychological models, inspired by psychoanalysis and behaviorism became increasingly fashionable and had a large impact on therapy.

The second wave of biological psychiatry started only in the second half of the twentieth century and was, according to Shorter, driven by two new discoveries. The first was genetics, which could show that severe mental disorders, in particular schizophrenia, have a strong genetic component. The second was the discovery of efficient medication for various mental disorders (1949 lithium, 1952 chlorpromazin, 1957 imipramin, 1958 haloperidol, 1963 diazepam). They quickly became a major pillar of psychiatric treatment and contributed strongly to the opening and later disappearance of the large mental asylums in the second half of the last century. Soon, the concept of a neurochemical imbalance of neurotransmitters became the favored explanatory model for psychiatric disorders. Interestingly, at the same time as psychiatry for the first time used effective medications, the movement of antipsychiatry emerged. It was part of a more general political protest against tradition starting in the 1960s and declared "mental illness as a myth" (Szasz, 1961). It also was quite effective in discrediting one of the most effective treatments for severe depression, electroconvulsive therapy, supported among other things by the impressive movie "One flew over the cockoo's nest" (1975) by Milos Forman. So although the second wave was in effect quite successful there was always some opposition against it on one

hand, but on the other hand those insights and practices that were helpful for patients are now integrated into daily practice.

So what is the third wave of biological psychiatry? I want to suggest that this wave has started in the last two decades of the twentieth century and is now in full progress. Again, it has been driven by methodological and technological progress. Since the declaration of the last decade of the twentieth century as the decade of the brain by the president of the United States, neuroscience has developed into one of the largest research programs worldwide. According to my view, there were two developments particularly relevant in the transition of the second wave into the third wave. The first is the progress in the molecular neurosciences. The journal *Molecular Psychiatry*, founded in 1997, is now one of the fields most prestigious and most cited journals. It became increasingly clear that the effects of psychiatric drugs are not primarily exerted via the level of neurotransmitters in the synaptic cleft, but that there is up- and down-regulation of receptors, effects on intracellular cascades, and even regrowth of neurons in the hippocampus. The picture of the neurobiological changes underlying psychiatric disorders and treatment thus became much more complex and differentiated and it became apparent that different levels of brain organization are important which interact in a complex way. The second development was the birth of cognitive neuroscience and neuroimaging. This field studies information processing in the brain by combining the methods of experimental psychology with tools to record brain activity or to stimulate the brain. In fact, neuroimaging, in particular functional magnetic resonance imaging (fMRI) has contributed much to public "brain awareness," by (although wrongly) suggesting that we can literally watch "the brain at work." With the first human study published in 1991, fMRI has today become a major research tool in psychology as well as in psychiatry. This development could not have taken place without a large increase in computational power. In fact, computational neuroscience which tries to develop mathematical models of brain function, has become an important tool in explaining neurocognitive processes and recently the program of computational psychiatry has begun to evolve (Montague et al., 2012). Further methods and technologies have become available to investigate the interplay of genetics, experience and environment in the etiology and neural explanation of psychiatric disorders like imaging genetics, epigenetics, optogenetics, or deep brain stimulation. Also big science, combining large – omic datasets like the (epi)genom, metabolom, proteome, or connectom with clinical data is becoming more important in psychiatric research and allows for new ways of discovery. The underlying model is that of systems medicine, understood as an interdisciplinary field of study that looks at the dynamic systems of the human body as part of an integrated whole, incorporating biochemical, physiological, and environmental interactions that sustain organismic life. In brain science, the paradigm of localationist thinking is substituted increasingly by thinking in functional systems and brain connectivity patterns (Buckholtz and Meyer-Lindenberg, 2012).

At this moment, we are at a critical stage of the third wave. In fact, progress in the first decade of this century has been so impressive that researchers as well as media have been overenthusiastic with regard to the power of the new methods. In particular neuroimaging results, probably due to their seemingly simple and straightforward presentation, have ignited the imagination of researchers, lay people and the media. Results are reported, similar to genetic results, in a oversimplified causal language ("love is in the ACC," "the God spot," "gene for schizophrenia discovered," etc.). Such oversimplified messages are well for drawing attention to headlines, but way over what really can be inferred from most studies. Consequently, neuroscience has recently been criticized for its overambitious claims, and the field of "critical neuroscience" has flourished in the last 5 years immensely with an increasing number of books, papers and blogs (for a respectable example compare Slaby and Choudhury, 2011). Actually, in neuroscience in general, as well as in cognitive neurosciences and neuroimaging in particular self-critical articles concerning methods have begun to be increasingly published (e.g., Kriegeskorte et al., 2009; Button et al., 2012) which is a healthy self-correcting development.

According to the third wave of biological psychiatry, mental disorders are relatively stable prototypical, dysfunctional patterns of experience and behavior that can be explained by dysfunctional neural systems at various levels. As with any understanding of disease in general the notion of a "dysfunction" inevitable involves normative judgments of what is regarded as normal, functional, healthy on the one hand, and as abnormal, dysfunctional, pathological on the other hand. Further below I will come back to normative issues. But before I do so, let's look at the concept of mental disorder within biological psychiatry.

WHAT ARE MENTAL DISORDERS?

Modern psychiatry has taken a lot of effort to make the description of psychopathology reliable by introducing standardized ways of exploring, describing and rating psychopathological patterns over time. In America, psychiatric disorders are diagnosed using the DSM-IV (published 1994), the Diagnostic Statistics and Manuals of Mental Disorders, the official handbook of the American Psychiatric Association (APA), sometimes referred to as the "bible" of psychiatry. According to DSM-IV mental disorders are diagnosed by carefully checking if subjects fulfill a certain number of psychopathological criteria for a certain amount of time. DSM-IV is agnostic on etiopathogenesis, i.e., the causal genesis of disorders, but rather has put emphasis of establishing a reliable, intersubjective schema for diagnoses on the psychopathological level. But what about validity, i.e., what is measured or rather intended to be measured with DSM-criteria? What kind of things are mental disorders? Kendler et al. (2011a) have distinguished four types of kinds that mental disorders could be. *Essentialist kinds* are based on an essence, e.g., an underlying cause, from which the defining features (the typical symptoms) do arise. Although this theory fits to some cases like progressive paralysis in syphilis or Mendelian defects in cholesterol metabolisms, it is now widely acknowledged that this model neither fits most chronic diseases as atherosclerosis, hypertension or autoimmune disease, nor psychiatric disorders. Rather, it is generally accepted that psychiatric disorders arise from a multitude of causes that are probabilistically related to signs and symptoms. Even in cases, where family and twin studies unambiguously have demonstrated that most of the variance is explained by genetics factors (e.g., up to 80% in schizophrenia) there is no single gene causing this disorder. Recently discovered risk variants explain only a tiny portion of

variance, usually less than 1%, although, using imaging genetics, they can be shown to have much stronger effects on the brain level (Walter et al., 2011). A second approach is to understand psychiatric disorders as *socially constructed kinds* which are brought about solely by the human activity of describing and classifying but not by an underlying structure independent of human constructs. Although this still is a popular thesis in the camps of cultural relativists and anti-psychiatry, this theory is rarely taken serious today. Instead, it is now widely acknowledged that cultural influences and social factors play important roles in the expression of symptoms, e.g., in the content of delusions. But it is also clear that for certain prototypical diseases (e.g., schizophrenia, bipolar disorder, depression, and some anxiety disorders) there are invariant patterns in experience and behavior despite eminent cultural differences. Many people think that what matters most is how we handle mental problems. So maybe psychiatric disorders are best understood as *practical kinds*. This approach holds that psychiatric disorders do not carve nature at its joints but just are those kinds which are most useful for certain purposes, ranging from medical ones (diagnoses and treatment) to sociological or even political ones (this is the point of departure of anti-psychiatry). This model is grounded in pragmatist philosophy and instrumentalism and has some appeal. In fact, the philosophy of DSM is very close to this approach with its agnostic and atheoretical framework. Although the practical kind of view avoids metaphysical discussion (like: What is schizophrenia really?), it gives us no advice as how classifications should be build and goes against many realistic intuitions that are the basis of successful applications not only in medicine. Instead, Kendler et al. (2011a) argue for a concept that is based on a model originating in the philosophy of biology, dealing with the problem how species are classified and on recent developments in the theory of neuroscience: the *concept of mechanistic property clusters (MPC)*. According to this view, the items to be classified rest on properties that need not to be shared by all members of a class, rather they should be understood as a cluster within an abstract space of features or properties in a multidimensional space. Some of those features may be more essence like, some more practical. Importantly, the MPC-view encourages the thought that there are robust explanatory structures to be discovered underlying psychiatric disorders. These explanatory multidimensional structures (genes, cell receptors, neural systems, psychological states, environmental inputs, social-cultural variables) are interacting in a complex and intertwined way, are sometimes fuzzy, but nevertheless stable. Importantly, it cannot simply be read from the superficial structure of items if they belong to the same kind. Rather, their membership is explained by the causal mechanisms that regularly ensure that their properties are instantiated together (a historical account). The interaction typically is inter-level, but can also be on the level on symptoms, thus mutually re-enforcing the pattern, e.g., in depression insomnia predisposes to tiredness and guilt predisposes to suicidal ideation. As MPC kinds are defined in part by the mechanisms that underlie and sustain them, the reductionist intuitions of old wave biological psychiatry are partially satisfied. However, the kind cannot be fully explained and thus understood if inter-level interactions, which are often hidden to the subject as well as to the external observer, are not taken into account. For example, it has been empirically shown that subjective explanations for depressive episodes by patients do not correlate with objective risk factors for depression (Kendler et al., 2011b) – a finding that makes it likely that explanations based on just a selection of levels (subjective experience and remembered behavioral events) do not explain depression well. The same can be said for simplified biological models of depression as a neurotransmitter deficit that ignores many of the other levels. Although the MPC-model does not tell us in advance what the relevant causal mechanisms are, it is consistent with the new biological wave in psychiatry which we will now characterize by describing a controversy around the introduction of DSM-5.

DSM-5 AND ITS CRITICS

On May 18th 2013 DSM-5 was launched at the meeting of the APA. When the APA started to work on DSM-5, it was hoped that it would be able to integrate new dimensional approaches (constellation of symptom dimensions, rather than categories of disorders) and more of the exploding neurobiological research results from the molecular and cognitive neurosciences. However, this hope was frustrated. Shortly before publication, the APA-DSM task force decided against these ideas, as it felt it would be too early and that research was not far enough to deliver sound evidence that could be integrated. Moreover, another feature of DSM-5 steered much controversies. Diagnostic criteria for some disorders were changed and new disorders were included. For example, the former exclusion criterion for the minimum duration of a depressive episode (normally 2 weeks, but after the death of a significant other at least 2 months) was skipped, which was criticized as the medicalization and pathologization of the normal human experience of grief. Diagnoses like binge eating disorder, mild cognitive disorder, and disruptive mood regulation disorder in childhood were introduced. These decisions were heavily criticized, most prominently by the psychiatrist who led the development of DMS-IV, Allen Frances. In fact, Frances had been arguing for years that DSM-5 was on the wrong track by introducing more and more disorders without taking into account that these will be overdiagnosed in practice and will create millions of new patients as well as justification for medication that is not indicated. In concert with the practices that advertisement for medication in the U.S. is allowed (not in most European states) this would lead to severe individual and societal side effects of overmedication, so the prediction of Frances and many other critics. Notably, he did not shy away to accuse himself of having performed similar mistakes by introducing three diagnoses in DSM-IV which he now regards as a mistake: attention deficit hyperactivity disorder (ADHD), child bipolar disorder and the Asperger-syndrome (a form of high-functioning autism). In his book "Saving Normal" (Frances, 2013) he argues that DSM-IV has been and DSM-5 will even be more leading to overdiagnoses, to pathologizing normal children and to the treatment of only slightly dysfunctional persons at the expense of taking care of the severely ill.

Here I will not discuss his arguments and the truth of his prognosis in detail, although it is highly likely that some of his predictions will become true, but rather point to an event surrounding the introduction of DSM-5 that makes the claims of the third wave of biological psychiatry clearer.

THOMAS INSEL'S ATTACK ON DSM-5

The date of the launch of DSM-5 at the APA meeting on May 18th was long known to everybody in the field. So it was probably not by pure chance that just 3 weeks earlier, on April 29th a blog was posted by Thomas Insel, a renowned neuroscientist himself (working in particular on oxytocin and vasopressin in animal research) and since 2002 director of the National Institute of Mental Health, the world's largest research institute investigating psychiatric disorders. He declared that "the weakness (of DSM-5) is its lack of validity. Unlike our definitions of ischemic heart disease, lymphoma, or AIDS, the DSM diagnoses are based on a consensus about clusters of clinical symptoms, not any objective laboratory measure. In the rest of medicine, this would be equivalent to creating diagnostic systems based on the nature of chest pain or the quality of fever. Indeed, symptom-based diagnosis, once common in other areas of medicine, has been largely replaced in the past half century as we have understood that symptoms alone rarely indicate the best choice of treatment" (Insel, 2013). This is a harsh judgment. And he also drew consequences: "That is why National Institute of Mental Health (NIMH) will be re-orienting its research away from DSM categories." This is quite a severe conclusion: just before the official diagnostic textbook of the APA is published after more than a decade of work, the largest research organization on mental health declares that it will orient its research away from DSM categories. Why? "(I)t is critical to realize that we cannot succeed if we use DSM categories as the "gold standard." The diagnostic system has to be based on the emerging research data, not on the current symptom-based categories. Imagine deciding that ECGs (=electrocardiograms, H.W.) were not useful because many patients with chest pain did not have ECG changes. That is what we have been doing for decades when we reject a biomarker because it does not detect a DSM category. We need to begin collecting the genetic, imaging, physiologic, and cognitive data to see how all the data – not just the symptoms – cluster and how these clusters relate to treatment response" (Insel, 2013).

So, in a nutshell: psychiatry has not been able to develop any objective laboratory test for clinical use because the current development of such tests is based on superficial criteria (symptoms), but not on the causal explanatory structures that underly them. If these structures exist he is right: it is difficult to make progress if you are measured by the fit with a descriptive, possibly faulty diagnostic system.

But there are further, homemade, problems within scientific psychiatry. Shitij Kapur, the Dean of the Institute of Psychiatry in London, and coauthors, among them Thomas Insel, gave three possible explanations for slow progress (Kapur et al., 2012). First, many studies in biological psychiatry are underpowered, i.e., they perform p-value chasing with small numbers of subjects (or animals). A good example is psychiatric genetics, but the same argument has been put forward for neuroscience in general (Button et al., 2012). Secondly, many studies are only approximately replicated, i.e., with different methods, different scanners, different paradigms, making it difficult to judge whether an effect is really stable. Thirdly: many stable effects, i.e., effects with large effect sizes are only found in extreme comparisons, i.e., by comparing patients with healthy controls. However, for clinical purposes it would be much more interesting to compare different patient populations. Kapur et al. (2012) also suggest methods to improve the situation, including to increase power, share data, and to report data more accurately. Most importantly, they argue for a stratified medicine (and psychiatry), i.e., for the identification of biomarkers or cognitive tests that stratify a broad-illness phenotype into a finite number of treatment-relevant subgroups. To put it into their metaphor of jacket producing: not to hope for a jacket with one-fits all (the usual approach) but also not hoping for a personally tailored jacket (like in the overambitious project of personalized medicine) but rather to go for a series of chest sizes of jackets for different groups.

RESEARCH DOMAIN CRITERIA: COGNITIVE SYSTEMS, NEURAL CIRCUITS, AND DIMENSIONS OF BEHAVIOR

A paradigmatic example of how the third wave of biological psychiatry is trying to get a grip on mental disorder and their underlying mechanisms is the initiative of research domain criteria (RDoC) developed by the NIMH which has been suggested as an alternative to investigate mental disorders and develop new classifications that are based on observable behavior and neurobiological measures. According to Morris and Cuthbert (2012) it developed out of two initiatives that targeted schizophrenia, in particular the MATRICS (measurement and treatment research to improve cognition in schizophrenia) and the CNTRICS (cognitive neuroscience treatment research to improve cognition in schizophrenia). RDoC can be regarded as a generalization of these initiatives being constructed for application to all mental disorders. It is based on three central assumptions: (1) mental disorders are presumed to be disorders of brain circuits. (2) Tools of neuroscience, including neuroimaging, electrophysiology and new methods for measuring neural connections can be used to identify dysfunctions of neural circuits. (3) Data from genetics research and clinical neuroscience will yield biosignatures that will augment clinical signs and symptoms for the purposes of clinical intervention and management. It also includes developmental processes and interaction with the environment as orthogonal dimensions that should inform hypotheses and conclusions derived from the RDoC organization structure. This structure is organized as a 2-dimensional schema. One dimension includes constructs that represent five core domains of mental functioning: Negative valence systems, positive valence systems, cognitive systems, systems for social processes and attention/arousal systems. Each of these domains includes subconstructs (around five). For example the negative valence systems include: active threat ("fear"), potential threat ("anxiety"), sustained threat, loss and frustrative non-reward. To take another example: the cognitive systems domain comprises attention, perception, working memory, declarative memory, language behavior, and cognitive (effortful) control. The second dimension consists of units of levels of organization on which the constructs can be measured. These levels are defined as follows: genes, molecules, cells, circuits, physiology, behavioral, self-reports, and paradigms. The "circuits" unit of analysis refers to measures that can index the activity of neural circuits, either through functional neuroimaging or through recordings previously validated as circuit indices (e.g., fear-potentiated startle). "Physiology" refers to well-established measures that have been validated by assessing various constructs, but that do not measure

brain circuit activity directly (e.g., heart rate, cortisol). "Behavior" may refer either to systematically observed behavior or to performance on a behavior task such a working memory. The advantage of this conceptualization in comparison to a purely symptom and course based system like DSM is that it is based on research on different levels, allows to characterize patients dimensionally, not categorically (diagnosis present or not) and that it is open to new evidence. Clearly, it cannot simply substitute DSM, which is based on long clinical experience, but it will inform classification based on multilevel science and might, in the long term, identify subgroups of patients that show characteristic constellations within this matrix that are helpful for categorization, treatment or management of patients. In the above mentioned blog Thomas Insel has announced that the NIMH will try to fund studies which follow such a transdiagnostic, systematic approach instead of studies that try to find neural correlates of categories that are simply based on the (superficial) clustering of signs and symptoms.

EVALUATING THE THIRD WAVE OF BIOLOGICAL PSYCHIATRY: A VIEW FROM INSIDE

By now the general approach or framework of the third wave of biological psychiatry should have become clear. It is focusing on a research-inspired, multi-level approach to understand what psychiatric disorders are, what mechanisms underly signs and symptoms and how an understanding of those mechanisms might help in classification, diagnosis, prognosis, and treatment. Note, that the approach does not entail the claim that biological approaches in a narrow sense are the best therapeutic approaches. It is as such neutral to the question what intervention will prove best to treat whatever there is. For example it may very well be that psychotherapeutic approaches will emerge as the best way to treat certain types of disorders. In fact, psychotherapists see no general problem in integrating their approach into such a framework as psychological mechanisms and principles that are effective in psychotherapy can be conceptualized as part of cognitive neuroscience itself (Walter et al., 2009; Disner et al., 2011). Also, the role of psychosocial and cultural factors can be integrated effortlessly as the MPC approach by Kendler et al. (2011a,b) makes clear: if social factors or societal and cultural mechanisms are part of the causal machinery that contributes to the instantiation of typical clusters of signs and symptoms that characterize psychiatric disorders they are part of the underlying explanatory structure.

However, probably many or at least some people will still view this approach skeptically. Indeed, there are several problems and limitations. To name just four of them: first, it could still be argued that the framework favors the neurobiological over other factors, as it entails the idea that psychiatric disorders are brain disorders. It will make no difference if you call psychiatric disorders "disorders of the brain" or "disorders of brain circuits" and thus do not justice to the mental within the concept of mental disorders. Second, the third wave does not include a solution to the normativity problem, namely the question when a constellation of psychological signs and symptoms is already a disorder or when it is still part of "normal experience," so it will still promote a medicalization of life problems. Third, even if we somehow could solve the first two problems, it might be argued that a focus on the brain will lead to inefficient resource allocation because the outcome for patients is not worth the effort be put in. History has shown that all general claims that we will in the near future know "the" causes of mental disorders have failed, and the continuous failure of neurobiology (with some exceptions) to sufficiently explain or predict mental disorders shows that it cannot account for such complex phenomena. Therefore, we should rather focus on the well-known psychosocial factors contributing to the development or sustainment of psychiatric disorders which are much more relevant in practice.

A recent critique of the thesis that "addiction is a brain disease" can be interpreted as a condensed combination of these worries. It argues that addiction would only be a brain disease if it has (i) neural correlates, (ii) these correlates are pathological and (iii) that pathology is sufficient for the person to have a disease, in almost any accessible environment Levy, 2013. As addicts are able to quit in certain environments, addiction would not qualify as a brain disease. This is a very clever argument as it uses one feature of the multilevel approach, namely the role of environmental factors, to argue against the "disorder of brain circuits thesis." Indeed, there is a grain of truth in this argument, but only insofar as it helps to distinguish "organic or neurological" from "mental or psychiatric" disorders. For example, neurodegenerative diseases like M. Huntington or Alzheimer will progress in almost any environment, whereas drinking might stop. However, there are two problems with this argument: first of all, it confuses behavior (drinking) with the disorder (alcohol addiction). It is well known that people suffering from alcohol addiction who manage to quit, still are addicted life-long and have a high propensity for relapse – exactly this might be explained by the brain disorder thesis. Secondly, the argument puts the stake much too high. Using the same kind of argument it could be argued that phenylketonuria, a genetically transmitted severe metabolic disorder is not *really* a metabolic disorder as it can be effectively treated by a diet, i.e., the pathology is not sufficient for a person to have a disease in almost any accessible environment.

Finally, some may argue, that also the third wave of biological psychiatry, like the preceding waves, will tend to devalue an approach to psychiatry that focues on the personal level. For example, the concept of MPC is based on the idea that regards minds as brains and brains as kind of machines that are causally effected by different levels. This approach, so the argument may go, ignores the personal level even if it may pay lip service to the subjective by for example including "subjective reports" in the RDoC grid.

There are several ways to response to these critiques from within, some of which I will mention here. First, admittedly, there is a common misunderstanding on the role of neurobiological findings in psychiatric disorders. Very often, it is either said, implicitly assumed, or implied that the mere fact that there is a neurobiological correlate of a mental dysfunction is already a proof that the "causes" of the respective disorder are biological in the same way as for neurological disorders. But this clearly is a misconception. Because every mental state has a correlate in the brain, we should be able to find at least in principle neurobiological correlates of any mental state, pathological or not. So the question is not, whether there is a neurocognitive correlate or mechanism, but whether it is pathological, how it came into being, whether it is persistent, whether and how it can be influenced, and so forth. In fact, the neurobiological misunderstanding even goes further in

many cases as often it is wrongly concluded that the existence of a "brain signature" (to use a more neutral term) would already imply that the disorder cannot be controlled or changed by psychological means, or even that it is inborn or genetically caused, implications which clearly are non-sequitures, but widely believed.

Second, the normative problem indeed has to be addressed – not only by biological psychiatry, but also by any other approach to psychiatry, and not only for psychiatric but also for all concepts of disorders – and consequently it has been discussed in medicine in general. As it is in no way specific for psychiatry, let alone biological psychiatry, I will not discuss it here in detail but just make some remarks. It is clear that the sheer discovery of neural correlates or mechanisms of a disorder cannot prove a state as pathologically. This can be done only by spelling out a concept of normal functioning. If a biological approach claims to be able to define mental disorders without reference to norms it must fail. Normativity in the context of mental disorders comes at least in three guises, "statistical," "biological design" or "value-preference laden" (Graham, 2013, p. 59). For example, most definitions of mental disorders include a criterion of suffering or of clinical relevance, that only can be spelled out with respect to a norm that cannot be read simply from biological facts. I will return to this issue later. Although it has to be admitted that the third wave of biological psychiatry does not take a specific stance to the normativity problem, it should be noted that this can be only used as a critique against variants of biological psychiatry that explicitly claim that normality can be inferred simply from biological measures.

Thirdly, why has neurobiology failed to deliver better results for explanation, diagnosis, prognosis or treatment? Some answers relating to methodological problems have been already discussed above (Kapur et al., 2012). However, a further explanation for only modest progress is often not mentioned. These are the ethical constraints under which biological psychiatric research has to operate which does make progress difficult. In contrast to other medical disciplines psychiatric research can access the "organ of the mind," the brain, only indirectly. There is no known ethically justifiable way to directly access brain tissue to investigate assumed molecular mechanisms. In contrast, the heart, the liver, the kidney and many other organs can be accessed directly in therapy and research by taking biopsies or measuring metabolites in the blood. There are only a few exceptions to this barrier, for example the possibility to measure certain molecules non-invasively with magnetic resonance spectroscopy, or with research windows related to invasive therapeutic procedures in epilepsy surgery or deep brain stimulation. Direct access to the brain in animal research also has its problems, because rodents and humans differ in many respects and animal experiments are confronted with ethical issues, too. So the "failure" of biological psychiatry is not necessarily related to its concepts or theoretical approach, but partly may be explained by important and relevant ethical barriers we have implemented in human research for good reasons.

Fourthly, does a biological psychiatry approach imply disrespect for persons? First note, that this critic in its most general and radical form is not confined to biological psychiatry but to any psychiatric approach that claims that there are mental disorders in the first place. This antipsychiatric argument claims that mental illness in general is a myth by confusing sickness with life difficulties and by stigmatizing people with mental problems as having a disorder and thus not giving them the credit and responsibility for what they do and chose to be. In a more specific and much less radical, but more frequent variant (not claiming the non-reality of mental disorders) a biological approach of psychiatry is accused of resulting in an overenthusiastic reliance on medication and an insufficient use of understanding the life stories and real-world problems of patients. Without doubt, overmedication is a problem in certain strands of psychiatry and admittedly this may be due to the fact of an oversimplified picture of mental disorders ("For depression you need to substitute serotonine like insuline in diabetes"). However, many of these implications are not inherent to the concepts of the third wave of biological psychiatry but rather are based on older conceptions that postulated a close connection between etiology and therapy, that has been abandoned today in current practice. For depression for example there was a distinction between endogenous depression (from within, medication, no talking cure), neurotic depression (originating in childhood, talking cure, no medication) and reactive depression (understandable reaction after a life event).

The aforementioned responses to a critique to biological psychiatry were given from within psychiatry and psychiatric research in itself. Many of these issues revolve about the "disorder" part of mental disorders. However, I think that a more comprehensive way of assessing the prospects of biological psychiatry can only be found when we turn to the "mental" part of a theory of mental disorders. In order to do so we can turn to a rich resource that has reflected on the concept of the mental for a long time: philosophy of mind.

RECONSIDERING BIOLOGICAL PSYCHIATRY: A PHILOSOPHY OF MIND PERSPECTIVE

If we want to understand what mental disorders are then we should take the question what "the mental" is more seriously. Traditionally, there has been a close link between philosophy in general and psychiatric theorizing. Here, I will restrict myself to recent philosophy of mind approaches, as they are targeting similar problems as biological psychiatry: what is the connection between mind and brain? The idea behind consulting philosophy is simple: if we better understand how mental states are related to brain states we might better understand how disordered mental states relate to disordered brain states. Take for example the thesis of identity theory that assumes that mental states are identical with brain states. If this is true, it seems to follow straightforwardly that disordered mental states simply are disordered brain states. Or take the problem of reductionism and mental causation: if we were really able to show that mental states can be reduced to brain states, this would leave us with only two possibilities: either we have to eliminate mental states, because they are nothing more than a convenient, folk psychological way to talk about hidden brain states or we have to conclude that mental states are epiphenomenal, i.e., have no causal powers. This seems like a conclusion only few people would like to embrace. Or take the idea of dualism. Do we have to assume a special substance that does all the work in explaining mentality that is in a separate ontological realm outside of physical reality?

However, if we dwell too deep into the heart of philosophy of mind, the danger is great, that we will end up with metaphysical

debates that might too easily be dismissed as theoretical talk with no direct relevance for psychiatry. Instead, I will refer here to two examples of the relevance of philosophy of mind for psychiatry: one specific approach of a theory of mental disorder by a philosopher (George Graham) and one family of problems discussed in contemporary philosophy of mind, namely if mental states extend beyond the brain in a relevant sense.

A comprehensive and accessible version of linking philosophy of mind and mental disorders has been given by Graham (2013). In his theory he explains what mental disorders are, according to which (normative) criteria we classify them as clinically relevant and how they differ as mental disorders from proper brain (=neurological) disorders. According to Graham a mental disorder is a disability, incapacity or impairment in one or more basic or fundamental mental faculties of psychological capacities of a person that has harmful or likely harmful consequences for its subject. It is a *disorder* because it is harmful and undesirable for the subject, whether the subject himself appreciates this or not. In more concrete terms this means that the person is worse off with than without the disorder, that the disorder has a non-voluntary and personally uncontrollable nature and that the disorder cannot be excised or extirpated by the mere addition of other psychological resources. For example, the delusion of a paranoid person will not be alleviated by giving more information about the content of his delusion and the sadness of a depressed person will not be cured by cheering him up. Mental disorders are *mental* disorders because they are brought about by a mix of mental forces and brute a-rational neural mechanisms, or at least Graham argues so. The crucial point here naturally is what Graham means by mental forces. He explicitly states that he is not a dualist. Rather, he tries to argue what the "mental" in mental disorders refers to. The mark of the mental is that states of mind are constituted by either or both of two elements, i.e., consciousness and intentionality. Only if the causal mechanisms bringing about or sustaining a mental disorder work through conscious and/or intentional states, so Graham claims, they should be categorized as mental disorders. Mental symptoms that arise from brute brain affections (like stroke, neurodegeneration, or infection) are neurological disorders even if they present with (secondary) mental symptoms. Also, the mental is decisive for the criteria when a mental state of mind should be regarded as a disorder and not as a variant of normal mind life: namely when they impact a person's reason-responsiveness or rationality considerably without totally destroying it.

In Graham's theory the mental plays a prominent role in several respects: first, because the mechanisms causing or sustaining mental disorders are supposed to work through those brain mechanisms that implement mental (intentional and/or conscious) states and thus through mental qua mental. Second, the normative criteria for clinical relevance (and thereby the criteria for separating normality from disorder) rely on the impairment of intentionality and rationality, i.e., marks of the mental. Thirdly, he argues that mental disorders (like panic attacks, schizophrenia, depression) should and can be distinguished from proper neurological brain disorders (like stroke, Parkinson, Alzheimer) by the fact that the latter are brought about by pure mechanical, brute, a-rational affections of the brain that moreover are not sensitive to psychological (mental) treatment. In contrast, the "mental" in mental disorders has a double role: first it is characterized by an impairment of intentionality and rationality and second, because these marks of the mental are not totally absent but within the symptoms there is still a sense of rationality and intentionality preserved.

A problem in Graham's theory is his explication of mental forces. Sometimes, he seems to imply that rationality or intentionality have a causal power of their own, although he denies that. But the worth of his approach for biological psychiatry seems for me that he insists on the relevance of the role of the mental in understanding, explaining and identifying mental disorders against pure brain disorders and non-pathological mind states on the other. In fact, many proponents of biological psychiatry now accept an interplay of neurobiological and psychological (mental) factors. However, if the mental is identical with the neural what does this claim of interaction amounts to? So let us turn then to the important question, if the mental can really be reduced to the neural.

In philosophy and in cognitive sciences there exist a number of proposals that doubt that cognitive processes (for our purpose: mental states) are best understood as only internal processes that happen within a cognitive system (in our case: the brain). Internal approaches, so the basic idea, ignore that cognitive processes are *situated*, i.e., that they essentially depend on (weak version), or even may be constituted by (strong version), their embodiment and the interaction with the natural, technological and social environment. There is not yet a consistent or complete theory of situatedness, rather there are several strands of research and theorizing that can be subsumed under the catchword "the 4Es": the embodied, extended, embedded and enacted mind (Lyre and Walter, 2013). The main idea is that in order to understand what cognition (the mental) is, it is necessary to take into account that cognitive capacities of a system may depend on the fact that those systems (our brains) are (i) *embodied*, i.e., coupled to our bodily constitution and that it therefore is necessary to regard the bodily realization of cognitive abilities as an integral part of the cognitive architecture; (ii) situationally *embedded*, i.e. are dependent in a specific way on their environment, i.e., cognitive systems exploit the specific circumstances of their environmental context in order to increase their performative abilities, (iii) *extended*, i.e., extend over the boundaries of our body into the technological or social environment and thus are constituted not only by internal factors but also by external, environmental factors and (iv) *enacted*, i.e., arise only by the active interaction of an autonomous systems with its environment (Walter, 2010).

The thesis of embodiment has a long tradition in phenomenological philosophy, e.g., in the writings of Merleau-Ponty. The thesis of the extended mind has more recently been introduced into the debate by a paper published in 1998 (Clark and Chalmers, 1998). They introduce an example of an external device for memory (a cognitive process) that has since then been discussed extensively in the literature. The example refers to the notebook of Otto, an Alzheimer patient with memory problems who uses his notebook instead of his normal physiological memory in order to remember certain things. The argument is that if the entries into the notebook play the same role in Ottos life and in the explanation of his behavior as neurally implemented memory contents

in healthy adults, it would be arbitrary or neural chauvinism if we would not regard them in the same way as genuine parts of the material substrates of his normal memories and beliefs. The general form of the argument inherent in this example is called the *parity principle*: if a part of the world functions in a way that, would it happen in our brain, we would have no hesitation in recognizing as part of a cognitive process, then that part of the world is part of the cognitive process. To make this part of the process more plausible it is easy to modify the example such that the notebook is constructed as a brain-computer-interface, e.g., as a digital device coupled more directly to the brain, for example in a technological advanced form of the actually existing google glasses.

Why could the 4E thesis be relevant to understand the nature of mental disorders? Because they regard processes external to the brain as constitutive for mental processes and thus also as constitutive for disordered, pathological mental processes. An example, where this might be relevant is ADHD. ADHD might be only correctly diagnosed as a mental disorder if the external world is such that adolescents grow up in an environment that favors attentional distraction and punishes hyperactivity. In a similar vein, anorexia nervosa, a severe and often deadly mental disorder in Western countries seems to be much less frequent or even non-existent in environments in which a slim figure and control of eating and weight is not promoted, like in poor countries in Africa. These facts seem to draw into doubt that every currently acknowledged mental disorder is best categorized as a pure brain disorder – which is not to deny that internal processes of the brain play an important role if specific circumstances hold.

The main point which I would like to make here is that biological psychiatry has to take into account theories about how the mental is constituted. The new wave of biological psychiatry might be able to incorporate these issues into its conceptualization of mental disorders – but only if it comes along with a consistent theory of the mental that should take into account arguments and insights of philosophy of mind.

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Commentary on Henrik Walter's "The third wave of biological psychiatry"

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There is good news: "Biological psychiatry is no longer biologistic!" According to Walter (2013), a seasoned German neuroscientist, psychiatrist and philosopher, a putative "third wave" of biological psychiatry has overcome many flaws that for a long time motivated our opposition to biological psychiatry: The third wave is no longer reductionist, localizations, or ignorant toward the normative, social, and cultural dimensions of mental problems. Rather, it analyses dynamic neural network activities, distinguishes multiple levels of description and takes every kind of context dependence you name into account. Furthermore, the third wave is aware of its methodological, theoretical and therapeutic limitations; and it self-criticizes all kinds of programmatic overstatements common in the field. In a nutshell: The third wave liberated biological psychiatry from its biologistic roots. It restricts itself to the legitimate search for the "biosignatures" of mental disorders.

From a down-to-earth point of view, this is hard to believe. Participate in any conference of biological psychiatrists these days and you can see that big money, pharmaceutical industry, reductionist ideas of man and a biologically biased psychiatric practice form a veritable, obviously flourishing coalition. But blaming theory for failed praxis would be unfair to Walter's informed and interesting paper. The question put forward by my commentary is, therefore, a theoretical one: *Are there any crucial remnants of biologism in Walter's apology of third wave biological psychiatry?* I think there are at least two points that justify further critical examination—points that Walter belabors extensively: the role of the mind and the role of the medical model of mental disorders in biological psychiatry.

What about the mind? Mentally ill people suffer. They consciously experience the burden of their condition. Psychiatry always intended to deal with the "mindedness" of mentally disordered people. But can modern neurosciences—the foundational basis of biological psychiatry—explain the phenomenon of mental life? The issue is not that mindedness depends on brain activity; it does. The controversial subject is that our scientific approaches to the study of the mental realm are misguided. Let me hint at three exemplary ways of misguidance:

(i) Due to the methodological restrictions of the behavioral sciences, biological psychiatry studies *"zombies"*—people with the same neuro-behavioral properties but without subjective consciousness. Zombie-psychology looks for strict correlations between operationalized behavioral paradigms and objectively measured neuronal activities; proceeding in this way excludes the mental realm by definition. As long as this is the case, all that biological psychiatry can ask for is the special status of an "applied clinical neuroscience" assisting general psychiatry.

(ii) Even worse is the *individualism* of biological psychiatry: The disturbed mental functions of psychiatric patients are regarded as individual, natural dispositions of the brain. Being a naturalist myself, I strongly disagree with this premise: Mental functions—our abilities to feel, to think, to act—are collectively defined, socio-cultural artifacts rather than purely natural, individual dispositions. To acculturate an individual, the "natural bottom up processes" of our species-design are developmentally coupled with socially mediated "non-natural top down processes" (Prinz, 2012). Conventional "mental instruments" are a result of the adaption to the cognitive niches of our culture (Sterelny, 2003); and they have to be continuously interpersonally re-calibrated to be effective (Pawelzik, 2013a). Walter acknowledges this point when he argues for a multi-level approach: The influence of interpersonally mediated cultural influences takes place on *supra-individual levels* of analysis: Activities on all levels—from gene expression to the cultural scaffolding of behavior—are relevant to understand a mind. But biological psychiatry does not study singular developments; it aims at the regularities of mental disorders; it therefore has to specify *which conditions on what levels generate the syndromatic pattern* that defines a kind of disorder. Looking into the brain for "biosignatures" will not inform you about the impact of the supra-individual level processes. In personal communication Walter would rebut: "All inputs, despite the level of origin, converge on processes in the brain; the brain is the eye of the needle of pathogentic influences." But what about the interpretation of the data you gather from the needle's eye? Will you be able to understand them if you disregard the nested senso-motoric slopes that "embody," "embed," and "extend" the "enactive" mind in a body, in a situation and a culture? Since Walter doesn't give us the slightest idea how this might succeed, I take his third wave as an individualistically limited enterprise.

(iii) What about the mind's *active role* in the etiology of pathological behavior? Walter mentions Levy's (2013) argument that addiction is no brain disorder like Alzheimer's since addicts can stop their addicted behavior. He counters this argument with the example of phenylketonuria: this metabolic disorder does not stop when you put your child on a phenylketon-free diet, since the pathogenetic mechanism is still left unchanged. Following this line of argument, one could say: all my thoughts, decisions and intentional actions—my way of life—will not change my brain, since the pathogenetic biological predispositions—from risk genes to temperament and maladaptive schemata—are still left unchanged. But this is simply not true. My actions can successively change my brain and its pathogenetic potentials. My strategies of *effortful control of attention*, e.g., that were entrained in early attachment-interactions and are actively developed to deal with all kinds of practical and social challenges in later life have an enormous influence on my behavior (Posner, 2012)—and therefore on my risk to develop a mental disorder (Pawelzik, 2013b). If the mind that supervenes on brain states can actively change brain states, thereby redirecting the brain's development depending on various environmental contingencies—than this "enactive mind" is obviously underspecified by the third wave concepts Walter offers. In order to overcome its traditional "mindlessness," biological psychiatry will have to undergo nothing less than a conceptual revolution.

Psychiatry is mainly about mental disorders, not about mindedness. "You are asking for too much," a sympathetic biological psychiatrist might respond. Scientific psychiatry would overstretch its chances if it tried to focus on the mindedness of the mentally ill. Scientific psychiatry's role is first and foremost to define and analyze mental disorders and to develop effective therapies. No wonder that Walter spilled most of his ink on the regulative idea of the field—the idea that mental disorders as nomological kinds.

"According to the third wave of biological psychiatry, mental disorders are relatively stable prototypical, dysfunctional patterns of experience and behavior," Walter declares. But is this really the case outside of university departments (where patients are strictly selected to fit scientific study designs)? Most of my patients show syndrome shifts, present symptoms that fit multiple diagnoses (on all axis of the DSM-taxonomy) and suffer from a wide range of nosologicaly ignored problems. These facts obviously limit the "prototypicality" of their illness. Furthermore, the contingencies of their learning histories, the influence of transdiagnostic, i.e., disease-unspecific developmental trajectories like attachment organization, their social situation and their individual "identity politics" call for an individualizing behavioral analysis that stretches over the whole spectrum of descriptive levels in order to plan effective therapy. No wonder that third wave biological psychiatry did not show up with the most convincing proof for the role of nomological pathogenesis—a therapy that fixes the pathogenetic mechanism.

Nevertheless, Walter might answer, that the phenotypical heterogeneity of mental illness might still depend on relatively homogenous biological regularities. Let's take genetic risk factors, e.g., Colleagues of Walter just demonstrated that *the same* genetic risk loci of two calcium channel signaling genes are involved in the development of five major mental disorders—autism, attention deficit-hyperactivity disorder, bipolar disorder, major depressive disorder and schizophrenia—that make up an astonishing broad spectrum of psychopathologies (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013). The pleiotropic effects of CACNA1C and CACNB2, this study highlights, might be due to the susceptibility for specific phenotypes depending on differential environmental influences. Well, we know that mental disorders are of multi-genetic origin and that gene-x-environment-interactions play an important role. But this truism doesn't specify the un-numberable interactive possibilities of epigenetics. To defend the idea of a quasi-nomologic etiology of mental disorder, one should at least be able to determine the interaction of a number of "risk-genes" that generate disease-specific "endophenotypes." In the case of Major Depressive Disorder, for instance, experts are discussing a rather long list of potential candidates ranging from anomalies of the HPA-axis to decreased subgenual PFC-activity (Hasler et al., 2004). What if vague syndromes like depression consist of individual mixtures of "sub-endophenotypes?" That might be the case; therefore we have to find out on which level we find the "mechanistic property clusters" that distinguish between supposed types of mental disorders, Walter might answer. If the nomological structure is not found on the levels of epigenetics or proteomics, it might still be found on the levels of the connectome and/or the activation patterns of definable neural networks. All we need is a biotype that robustly correlates with certain experiential and behavioral patterns. Without going into further details, my question is: What will happen if no connective or functional patterns fit our established nosology? Will we go for a better, strictly biological nosology, as Thomas Insel demands? Or will the regulative idea of psychiatry—mental disorders are nomological kinds—slowly degenerate? I don't know. But as a keen observer of the dynamic market of "biosignatures" I wouldn't put much money on this meta-hypothesis that Walter's third wave still entertains.

To sum up: Walter's description of third wave biological psychiatry is on the right track: We should embrace his purgation of a lot of biologistic thought. Still, as I tried to show, Walter left the main conceptual pillars of biological psychiatry—"mindlessness" and "medical model"—basically untouched.

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Of waves and troughs

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A commentary on

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In 1998, *Eric Kandel* wrote in his intriguing paper titled "*A new intellectual framework for psychiatry*" (Kandel, 1998) that "the unique domain which psychiatry occupies within academic medicine, the analysis of the interaction between social and biological determinants of behavior, can best be studied by *also* having a full understanding of the biological components of behavior." Fifteen years later, much like surfers who continue a frustrated and longing pursuit for the next "big one" (Cowan et al., 2000; Kandel, 2006), we are, according to *Henrik Walter*, in the midst of the third wave of biological psychiatry (Walter, 2013). Because a wave is, in a physical sense, a disturbance that propagates through space and time while transferring energy, there are at least three reoccurring "thermodynamic sinks" that I would like to *also* emphasize with *Walter* to ultimately better understand the complexity of the human brain in action (Bassett, 2011).

First is the rediscovery of the coequal contributions of emotions and affects toward normal brain functioning (Damasio, 2003; Tsuchiya and Adolphs, 2007). After *Michael Gazzaniga* and *George Miller* "invented" "*cognitive neuroscience*" in the late 1980s (Zorumski and Rubin, 2011), the predominance of a cognition-centered view of "higher" (and perhaps as one facet: more noble?) brain functions was able to again delay necessary and not so new "insults" to our species and misdirect (in its top-down-view of brain functioning) the conceptualization and treatment of mental disorders (Cromwell and Panksepp, 2011; Almada et al., 2013). Neurobiology helps us recalibrate the human wishful thinking we had come to appreciate regarding the "higher" and "lower" of the "conditio humana" imprinted in our (neuro)physiology. As the world divides into facts, there is in fact no *such* hierarchy imprinted in our brains. Rather, the brain seems to favor "dynamic coalitions of networks of brain areas with a high degree of *connectivity*," and these networks - or the *connectome* - should not be conceptualized as being specifically affective or cognitive (Pessoa, 2008).

Second is the rediscovery of the body in biological psychiatry. *Walter* mentions the "4Es" (*embodiment, embeddedness, enactivism, extended cognition*) and the challenge of so-called "*situatedness*" (Walter, 2013). However, the very first step toward valuing the operant inter-wovenness of mind and body might be a simpler one. Interestingly of *ecto*dermal origin, neural tissue emerged enabling motor control in an evolutionary beneficial way. The brain originates in relation to a body that again, in relation to the outer world, actively moves – and, not least, gained the ability to interact with other bodies. Sensory information about the "*situation*," the *reflective* information involved in reflexes, is primarily able to close the loop and help coordinate movement. If *Antonio R. Damasio* is right, there is a need for *emotions* before we can *feel* anything, and these *emotions* are intimately connected with "more or less the complex reactions the *body* has to certain stimuli" (Damsio, 2005). These so-called "*somatic markers*" (Damasio, 1996) apparently make us capable of making predominantly beneficial decisions for self-preservation and the (we have to admit: biologically sexual) preservation of our species. It is *design*ative that the brain is the "*unmoved mover.*" However, changes are also reflected in the brain itself if the "motor-sensory" connections to the body are disturbed, e.g., in paraplegia (Wiens, 2005; Lenggenhager et al., 2012). The fantasy of an ever-dreaming, monolithic (but nonetheless self-conscious) "*brain in a vat*" that could reasonably think (or meaningfully simulate) about "*what is it like to be a bat*" (Nagel, 1974) currently suffers from not only solipsistic but also neurobiological-Darwinistic (so to say "inborn") pitfalls. In this manifold context, it is interesting that today's "modern or third wave" of psychiatry is more willing to pay increased attention to enigmatic *somatic symptom disorders* (other than at first glance mere "brain disorders" such as *schizophrenia*, *depression*, *addiction*, and *dementia*) and attempts to incorporate the body and its imprinted neural representation into a genuine, more holistic understanding of the field. One could interpret it as a new esteem of anciently quirky psychosomatics in biological psychiatry that overcomes its centro-centric monodimensionality.

Third is the rediscovery of the importance of "being in relation" for reasonable neural functioning, especially in terms of social relationships for the human brain. From birth until death, human mammals need the "significant other(s)," and it is perhaps the most integrating framework covering cognitive and affective neurosciences that will give rise to emerging social neurosciences (Eisenberger and Cole, 2012; Singer, 2012). Newly emerging imaging techniques, such as *hyperscanning* (Babiloni and Astolfi, 2012), i.e., the simultaneous recording of brain activity

of different subjects that allows "the study of inter-brain correlations between the cerebral activity of a group of interacting subjects as a unique system" (Babiloni and Astolfi, 2012), will help us understand the brain and perhaps pave the way to a central *second-person neuroscience* (Schilbach et al., 2013). Against this background, and only as one important example, empathy and the question of its quality and quantity in men have gained more and more attention in modern neuroscience (Gonzalez-Liencres et al., 2013). Psychotherapy and its proven impact on mental health (Etkin et al., 2005), before any technical question, fundamentally relies on the quality of the relation between two human beings (like patient and therapist) (Ardito and Rabellino, 2011). One could, again, interpret this rediscovery as the new esteem of anciently subordinate psychotherapy in biological psychiatry.

Finally, after three waves, a fourth wave seems inevitable. I would venture to predict that this "new wave" will belong to the computational neurosciences (Wen et al., 2011; Poldrack et al., 2012) and arise from the background of *information integration theory* (Tononi, 2005). The *Human Brain Project* (Markram, 2012) which was awarded one of the European Union's Flagship grants in 2013, worth more than €1 billion ($1.35 billion) over the next ten years, aims for the first time to *tie* or *link up* all knowledge of and to *simulate* the complete human brain from the molecule to the cortex on supercomputers to better understand how it functions (or even malfunctions), is ultimately the first step into a new era of real *cooperativeness* among neuroscientists and brains (Markram, 2013). Unfortunately, largely without "third wave" psychiatry. Just as affect and cognition, body and soul, the body-bound brain and *the* brain of my conspecific, and psychiatry and psychosomatics grow together, entities that belong together grow together. We have the opportunity to see the emergence of a new, non-reductionist science of fractal brains, as we examine mental orders and disorders differently, in a "brainy way," with more cooperation and integration than ever before. In one word, in accordance with *Henry Markram* (Kandel et al., 2013): exciting!

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On the use and misuse of externalist approaches in psychiatry

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Keywords: externalism, extended cognition, reliability, biological psychiatry, philosophy of science

Walter (2013) argues in that a bunch of externalist approaches examined in the theory of mind is interesting for psychiatry—especially for biological psychiatry. The externalist approaches state that mental states are not only constituted by internal but also by external factors. He subsumes them under the so-called 4E-thesis: embodied, embedded, extended, enacted. His example is concerned with ADHD such that ADHD is only existent "in an environment that favors attentional distraction and punishes hyperactivity."

First, using the 4E-thesis seems to be explanatorily attractive: There are factors of the disorder that can be grouped as external constituents and these constituents are not named by internalist vocabulary. For example, punishment is for sure not part of internalist vocabulary.

But there is a figure of a principal argument against using the 4E-thesis in psychiatry. Consider this cookbook theory of the 4E-thesis:

Cookbook theory for the 4E-thesis in accord with Henrik Walter's multilevel-approach:

1. There is the phenomenon of a psychiatric disorder instanced in the form of multilevel-complexes.
2. There are *embodied*, *embedded*, *extended*, or *enacted* (4E) constituents of cognition that realize the cognitive systems.
3. There are individuation criteria for the 4E-constituents.
4. Coupled systems of individuated 4E-constituents individuate a multilevel-complex.
5. There are individuation criteria for systems of constituents C that realize multilevel-complexes.

For example, with respect to extended cognition, the cookbook theory is the following:

Cookbook theory of extended cognition:

1. There is the phenomenon of cognition of a group of patients in psychiatry instanced in the form of cognitive systems of a specific structure.
2. There are vehicles of cognition that realize the cognitive systems named in 1.
3. There are individuation criteria for vehicles named in 2.
4. Coupled systems of individuated vehicles individuate a cognitive system.
5. There are individuation criteria for systems of vehicles of cognition that realize cognitive systems named in 1.

As one can see, every attempt of the 4E-thesis needs criteria marking where to *stop* adding externalist constituents to the constituent base of the psychiatric disorder. For example, extended cognition-constituents should not include the whole world to make the thesis an interesting thesis—otherwise every phenomenon to be explained by extended cognition is constituted by the same constituents (i.e., the whole world). But what kind of criterion can the 4E-externalist provide us with?

First, the 4E-externalist could give a criterion based on intrinsic properties of systems realizing disorders. But as a detailed analysis of criteria of this kind by Adams and Aizawa (2001, 2010) shows, these criteria are regularly based on finding a mark of the cognitive—which seems to be a hopeless endeavor without being a fundamentalist who just adds *axiomatically* this criterion to the cookbook theory.

But second, the 4E-externalist could maybe give a criterion based on extrinsic properties of systems realizing psychiatric disorders. For example, one could use *reliability* as a stop-criterion for delineating what is coupled (with respect to point 4 in the cookbook theories). What is *reliably coupled* builds a constituent base of a system realizing a psychiatric disorder. But as a detailed analysis shows, this is not the case (Bukow and Will, in press). With respect to different criteria of reliability, there are very different reliable systems. This is not trivial, because in accord with the cookbook theory-approach, Bukow and Will argue:

Argument for the arbitrariness of the reliability-predicate:

1. Assume the cookbook theory of extended cognition.
2. Give a definition of reliability with an operationalization (e.g., reproducibility as an operationalization).
3. Choose an experimental procedure to test for reliability based on interaction between systems (because reliability is relative to this experimental procedure).
4. Choose a value X%, i.e., that the experimental procedure is reproducible in X% of all series of experiments.

There is *no* established decision procedure for the needed choices in points 3 and 4. And whatever procedure you choose, you may get different values for X% which will lead the psychiatrist to different constituent bases of the psychiatric disorder

to be explained. Bukow and Will analyze in detail other candidates for criteria based on extrinsic properties, for example functional roles, causal roles, or heuristics.

Now, there are two horns for the 4E-externalist:

> Horn 1: Use a stop-criterion based on intrinsic properties. The critics of Adam and Aizawa bite the proponent of this kind of criterion. If you add the criterion axiomatically, then you are a fundamentalist and not a scientist.
> Horn 2: Use a stop-criterion based on extrinsic properties. Then, there is the need to show in detail how to use such a criterion—which seems to be hopeless in accord with the investigation above. If you add the criterion without detailed analysis, then you are using *just so-stories*, without guarantee that the criterion fulfills its job.

For these reasons, using the 4E-thesis is tempting but dangerous—there is no well-founded *stop-criterion* in philosophy of mind that a psychiatrist could use—not in a detailed way and not in a rough way. It is arbitrary what type and what token the psychiatrist may use with respect to the selection of constituents of a disorder. In a principal debate, the 4E-proponent may excuse these deficits because nobody may give in principal such stop-criteria. But in psychiatry, principal deficits are no excuse for misuse. Arbitrariness can smooth the way for use—but also for misuse in psychiatry, which is a dangerous zone with respect to rights and needs of patients of psychiatry. And if a psychiatrist can only give arbitrary constituent bases to a patient's disorder, then disorder and treatment will only arbitrarily be defined. This is, as far as I can see, an objection against Henrik Walter's argument for a mature "third wave" biological psychiatry.

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Normative preconditions for the assessment of mental disorder

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The debate about the relevance of values for the concept of a mental disorder has quite a long history. In the light of newer insights into neuroscience and molecular biology it is necessary to re-evaluate this issue. Since the medical model in previous decades was more of a confession rather than evidence based, one could assume that it is—due to scientific progress—currently becoming the one and only bedrock of psychiatry. This article argues that this would be a misapprehension of the normative constitution of the assessment of human behavior. The claim made here is twofold: First, whether something is a mental disease can only be determined on the mental level. This is so because we can only call behavior deviant by comparing it to non-deviant behavior, i.e., by using norms regarding *behavior*. Second, from this it follows that psychiatric disorders cannot be completely reduced to the physical level even if mental processes and states as such might be completely reducible to brain functions.

Keywords: biological psychiatry, norms, reduction, cultural relativity, morality

INTRODUCTION

In the course of the "molecular turn" (Rudnick, 2002) in psychiatry, researchers purport to "provide more objective diagnoses" (Akil et al., 2010, p. 1581) with the help of biological markers. Our traditional diagnoses, they claim, are not only unhelpful but actually a handicap for causal research (Holsboer, 2010, p. 1308). This is why "psychiatric disorders should be reclassified as disorders of the (central) nervous system" (White et al., 2012, p. 1). Even the neurosciences seem to have lost their leading position and appear to have gotten diminished to merely heuristic value since the "real" discoveries are to be expected on the molecular level (Bickle, 2006). While the adherents of the disease (or medical) model of mental[1] disorder purport that psychiatry is at least as value free as all the other sciences, critics claim that psychiatry rests on norms and values *over and above* those being present in, say, physics or chemistry, since it deals with the mental, i.e., the experiences, emotions, and behaviors of persons, and therefore always includes norms in respect to these phenomena.

It would be trivial claiming that even the criteria for something being a brain defect rest on norms and that, hence, the criteria for a mental disorder cannot be norm-independent either because they rest upon brain defects. The claim made here is twofold: First, whether something is a mental disorder can only be determined on the mental level. This is so because we can only call a behavior deviant by comparing it to non-deviant behavior, i.e., by using norms regarding *behavior*, which simply are not applicable to neurons. The brain alone cannot give us the evidence necessary [2]. Second, from this it follows that psychiatric disorders cannot be completely reduced to the physical level, may it be neuronal or molecular. The classification of something as a mental disorder cannot even in principle be free of values and norms and can be "objective" only insofar as norms and values can be seen as objective. This is the case even if mental processes and states might—in principle as well—be completely reducible to brain functions. Hence, for the sake of the argument I will take the latter for granted: there is no behavior or experience, I assume, that does not come from the brain, and there is nothing in the mental realm that could not be reduced to the brain's processes. Nonetheless, whether a certain kind of behavior or experience should be seen as disordered, is *not* reducible to the brain's functions.

Thomas Szasz once stated: "It is not by accident that, in all the psychiatric literature, there is not a single account of voices that command a schizophrenic to be especially kind to his wife" and he continued, "[t]his is because being kind to one's wife is not the sort of behavior to which we want to assign a causal (psychiatric) explanation" (Szasz, 2001, p. 300). Even if we are not devoted adherents of Szasz, this quote should give us pause. There seems to be something peculiar about behavior that is beyond purely physical explanation because the difference between, say, acting kindly and unkindly can hardly be grasped in physical, non-normative terms.

In this paper I neither intend to offer another definition of mental disorder nor do I claim an incompleteness of some sort of neuroscience. Above all, I want to stress at the very beginning that

[1] In the following I will use "mental," "psychiatric," and "psychological" disorder interchangeably. Likewise the term "behavior" is used as a placeholder that stands for "experience, emotion, and behavior."

[2] Imagine a neurologist tapping with her finger on your brain scan and telling you "Oh, look, you were quite depressed last week."

I do not doubt the existence of mental disorders. If you have ever seen a deeply depressed person, or a schizophrenic desperately asserting his responsibility for the destruction of the WTC twin towers, you will not have any doubt about the existence of mental disorders. All I want to show is that mental disorders cannot be determined in a purely physical way.

In the following section I will explain my claim that psychiatric diseases are irreducible to the brain even if the mental as such may in principle be reducible. In the main part of the paper I will first show that psychiatry is embedded in several *normative frames of reference*, and then refer to five particularly relevant normative dimensions of psychiatry. These are the concept of *rationality*, *moral* assumptions, the notions of *harm and distress*, several *cultural* norms and influences, and finally the relevance of—equally normative—*routes of explanation*.

THE PHYSICAL FOUNDATION OF THE MENTAL

There is no behavior that does not arise from the brain. Neither is there something like a Cartesian soul, nor is there full-fledged mental causation. How can one nonetheless regard mental disorders as irreducible to neurobiology? Doesn't this look like wanting to have one's cake and eat it too? It might, at first glance, but things are not that simple.

If biological psychiatry was nothing but an ideology, as some authors claim (Cohen, 1993; Berger, 2001; McLaren, 2010), one would just have to show the irreducibility on this level. But we do not need to make such a principled assumption.

Let's assume every single aspect of our mental and behavioral life could be explained in purely physical terms. In this case it could not only be shown *that* our brains, together with our genetic endowment, are responsible for the way we are, but also *how* this happens, and which mechanisms are involved in producing this or that kind of thought or behavior. Let's further suppose the neurosciences could even explain the so-called phenomenal qualities—the "what it is like" to see red or to be depressed. Since what we call "mental disorder" is without doubt part of people's mental and behavioral lives, it would be explicable in purely physical terms as well. So it seems. To give an example: It would be possible to explain which of the brain's functions and properties make a person feel "depressed." To make the claim even stronger, let's take for granted that environmental influences, too, are explicable mechanistically and that "[e]xploring the mechanisms of gene-environment interactions for depression is not substantially different from understanding how environmental toxins contribute to cancer or how diet influences cardiovascular diseases" as Thomas Insel and Remi Quirion assume (Insel and Quirion, 2005, p. 2221). Would we be able to determine what a mental disorder is by physical means alone? We wouldn't.

This is due to the fact that no behavior or inner feeling has a sticker on it that reads "I'm a disorder!" We have to write those stickers ourselves and attach them to certain feelings and behaviors. It is completely right when Matthew Broome and Paolo Fusar-Poli write:

> "It is by observing how the person behaves with respect to her beliefs, and by witnessing such behavior in the process of the giving and asking of reasons that one suspects delusions, not in viewing a brain scan or a genetic sequence. In other words, the diagnosis of delusions is based on the observation of behavior that violates accepted norms (e.g., of rationality for belief reports)."
> (Broome and Fusar-Poli, 2012, p. 598)

In short, whether something is a mental disorder has to be evaluated, not be discovered. This seems to be a purely Szaszian account, but it is not. According to Szasz, mental disorders are evaluated on a normative basis and not, as it is the case with physical diseases, discovered on the basis of functional or structural lesions. Psychiatric diagnoses "are driven by non-medical, that is, economic, personal, legal, political, or social considerations and incentives" (Szasz, 1994, p. 37). Up to this point I agree with Szasz. But while he claims that mental illnesses cannot be treated by medical means for this reason, I neither maintain this, nor do I dispute their existence. His argument seems to be something like this: (i) only medically discoverable conditions can be treated medically; (ii) mental illness is not medically discovered but normatively evaluated; (iii) mental illness cannot be treated medically. The argument fails because premise (i) is problematic. If we reformulate it into "only physically based conditions can be treated medically" the problem becomes obvious: Szasz confounds the epistemological and ontological side of the issue. All that can be inferred from the fact that mental illness is evaluated and not discovered is—at best—that there are no natural kinds of mental illness. We draw the line between normal and allegedly deviant behavior somewhat arbitrarily. But the question of how we can and should categorize forms (and norms) of behavior is different in kind from the further question of whether mental disorders exist. The first one is an epistemological question, the second one is ontological. Moreover, it is obvious that we can even "treat" completely normal behavior. Psychological enhancement gives the best evidence. This follows not at least from the assumption that no behavior or experience can exist without a brain producing it. Change the brain and you change the mind[3].

While Szasz asserts mental illness does not exist because of its evaluative nature, my weaker claim is that it will never be possible to determine in a purely physical way which of the countless variants of behavior and thinking are disorders, even if we might discover all the physical causes of each and every thought and form of behavior one day. Hence, the irreducibility of mental disorders is not due to the mind-brain problem. But where exactly does the irreducibility come from? In the following section I will give an outline of the main normative aspects that prevent mental disorders from being explained purely physically.

NORMATIVE BEDROCKS OF MENTAL DISEASE

Stating that everything is normative insofar as we have to decide what kind of evidence we want to count as proof for something or what we are willing to accept as an explanation in science would be trivial. It would not be very shocking to claim that, e.g., neuroscientists have to use normative concepts such as the

[3]Paquette and colleagues put it the other way round: "Change the mind and you change the brain" (Paquette et al., 2003). This is, of course, true as well, but not because of some spooky sort of mental causation, but rather because changing the mind *just means* changing the brain.

"correct functioning" of certain brain areas. Nearly everything in the world—including psychiatry—is normative in this sense. A much more provocative claim is that psychiatry is guided by social, moral, cultural and other norms. If this is true, and if it is also true that these kinds of norms are relative to time and place, then psychiatry cannot claim to know what a mental disease is "in itself," where normality ends and mental disorder begins. Again, if the boundary between normality and mental disorder is a social construction such that the question of whether a certain kind of behavior is a disorder can only be judged against the background of this very convention, then the "disorderness" of a condition cannot be found on—and hence not be reduced to—the neuronal level. Psychiatry would have to admit that it serves—to a certain degree at least—not only the needs of patients but those of society as well.

NORMATIVE FRAMES OF REFERENCE

Judgments of psychiatric disorder always need a background of psychiatric *order* without which no diagnoses could be made. A relatively easy way of finding such a background or "frame of reference" is to take a set of diagnostic criteria and turn them (back) into behavioral imperatives. Leising and colleagues have made visible the normative assumptions inherent in the DSM-IV criteria for personality disorders (PDs) in this way (Leising et al., 2009). To give just one example: On the basis of criterion one of Borderline and criteria seven and eight of Dependent PD they formulated the underlying norm "be able to tolerate real and imagined separation[4]." If a person is not able to conform to this and other social standards she may be a candidate for a PD. It may be objected that this only refers to some single criteria while in the case of, e.g., Borderline PD seven out of nine criteria have to be met. This is true, of course. But what about the normativity of the other criteria? What do "unstable and intense interpersonal relationships" (DSM-IV-TR, 301.38, 2), or an "unstable self-image" (DSM-IV-TR, 301.38, 3) mean?

A principled objection against the normativity assumption could go like this: The current diagnostic manuals are indeed deeply misguided, but once we have found the *real* and *appropriate* criteria for psychiatric disorders, we will get rid of the normativity problem. But again, on the basis of what background or reference frame will such an ideal manual function? Since it is always experience and behavior that have to be judged as pathological, we will always have to draw on "average people" to tell apart mental and/or behavioral deviance on the one hand and "normality" on the other.

In particular, four such normative frames of reference can be distinguished (cf. Leising et al., 2009 for the following)[5].

(1) The personal values of a given diagnostician: In the absence of a strong theoretical foundation it is more likely than not that the criteria follow the values and worldview of those who establish them.
(2) Cultural expectations: Diagnoses might not primarily refer to the person but to the mismatch between her patterns of culturally primed behavior and the expectations of her current social environment. For instance, western-style behavior of a girl in rural areas of Turkey may become a candidate for a PD. Conversely, rural Turkish behavior patterns may be seen as an indicator of a psychiatric disorder in the west.
(3) Generalized assumptions about human nature: While it may be possible to determine something like "normal functioning" of the body, e.g., in respect to heart, liver, or the hormonal system, it is quite difficult, if not impossible, to find universal human mental and behavioral patterns. Even if there is a species-typical behavioral setup, it is questionable whether the thresholds to pathological behavior and thinking similarly follow species-typical patterns[6].
(4) *Harm and disturbance*: What constitutes harm for one person does not need to constitute harm for another. In particular, the thresholds to harm and the kinds of issues that are regarded as harmful differ from one culture to another. Therefore, harmfulness is always judged against the background of varying, contingent frameworks.

While these frames of reference are situated on a more general level, Sadler and Fulford have indicated seven normative judgments that are "nested" in the *individual* diagnostic act (Sadler and Fulford, 2006, p. 171 f.). These concern:

(i) a match of the criterion's semantic content against the patient's phenomenal clinical presentation;
(ii) a judgment by the examiner about the appropriate approach to the solicitation of relevant data from a patient;
(iii) an examiner judgment about the prevailing sociocultural norms relevant to a particular criterion;
(iv) an appraisal of the patient's performance (behavior, interview discourse) relevant to said sociocultural norms;
(v) a comparison between the patient's performance and the specific sociocultural norms in determining whether the patient's performance substantively deviates from them;
(vi) the determination of whether such deviance is substantive enough, qualitatively (e.g., idiosyncratic deviance, as in "bizarre delusions") or quantitatively (e.g., as in "excessive" need for reassurance in dependent PD), to constitute psychopathology; and, finally,
(vii) a judgment about whether the criterion-driven behavior and experience is disvalued or for the worse.

Apart from the respective diagnostic manual the diagnostician in a clinical setting cannot but make a whole range of normative judgments in individual cases. It is *in principle* impossible to get rid of this normative aspect of the task, even if the underlying

[4]The original DSM-IV criteria are: 301.38 (1), "frantic efforts to avoid real or imagined abandonment"; 301.6 (7), "urgently seeks another relationship as a source of care and support when a close relationship ends"; 301.6 (8), "is unrealistic preoccupied with fears of being left to take care of himself or herself."

[5]The following four frames of reference are oriented toward those of Leising et al. (2009) but are not completely identical to them.

[6]This holds notwithstanding the assumption of a set of ubiquitous virtues (courage, justice, humanity, temperance, wisdom, and transcendence) shared in all cultures (Dahlsgaard et al., 2005).

biological mechanisms of a particular behavior or experience were completely known.

In the following I will discuss five normative dimensions that are present in psychiatry to varying degrees. The first is "rationality," the role of which is somewhat underestimated in the discussion of the normative preconditions of psychiatry (section Rationality); the second refers to the special case of PDs which seem to be particularly dependent on moral expectations (section Morality); third, there is the problematic notion of "harm and distress" that has already been mentioned above (section Harm and Distress); fourth, we have to ask to what extend the concept of psychiatric disorder is relative to different cultural backgrounds (section Culture); the fifth normative dimension pertains to the relativity of scientific explanatory routes which are no less normative in character (section Routes of Explanation).

RATIONALITY

Even though "irrationality" and corresponding terms are not explicitly mentioned as criteria in the current versions of DSM or ICD, Marie Crowe has pointed out that there are several features to be found in the DSM with which a person's perception of reality must be consistent in order for the person to be attributed with rationality. These include notions such as "impairment in reality testing," "magical thinking," "suspects without sufficient basis, that others are exploiting, harming or deceiving him or her," or "worry about everyday, routine life circumstances" (Crowe, 2000, p. 75). Yet, this does not say what kind of reality is at stake.

There are several concepts of rationality (Bunge, 2007), two[7] of which are of particular interest in psychiatry: The first one is theoretical or linguistic in nature (logical rationality) while the second one is practical in the sense of means-end rationality (practical rationality). When someone concludes from (i) human beings are mortal, and (ii) Socrates is a human being, that (iii) Socrates is *im*mortal, his theoretical rationality has failed. If mental disorder could be characterized by a lack of theoretical rationality, things would be quite easy. Unfortunately, this is not the case. A couple of years ago a study was conducted showing schizophrenic people to be even more theoretically rational than average persons (Owen et al., 2007). Practical rationality, on the other hand, comes in degrees and is not always judged by the same standards. If a person who has become convinced by advertisement that a certain kind of caffeinated drink makes you popular and henceforth consumes it for this reason, we would probably attest a lack of practical rationality. If someone seeks a cure for cancer in prayer, this would be (at least in the eyes of many) a grave lack of practical rationality, too. Now think of a person who washes her hands every 10 min in order not to catch an infection. There are, of course, other forms of practical non-rationality which leave hardly any doubt that something must be wrong with a person. But we have to set the cut-off ourselves, and there is no other way than doing this somewhat arbitrarily.

[7]Bunge distinguishes seven concepts: conceptual, logical, methodological, epistemological, ontological, and valuational rationality (Bunge, 2007, p. 117 f.).

The problem already begins with the assessment of capacity and competence to make treatment choices. While it could be argued that there is an objective way of assessing patients' capacity by testing their cognitive abilities to understand, retain and weigh up information, it is often overlooked that this is accompanied by a number of inherently normative judgments in clinical practice (Banner, 2012). Hence, it is not only the capacity of the patient that can be put into doubt, but also the way she makes use of it. And this aspect, the *way* of using information, cannot be assessed but on normative grounds. One of the most well-known examples in this regard is anorexia nervosa, where patients usually completely understand the relevant information and consequences but nevertheless make choices that other people would regard as problematic (see, e.g., Craigie, 2011).

The assessment of rationality in people's choices is normative in two respects. First, it is not always a precondition for recognizing the autonomy of a person; in some circumstances it is, in some it is not. Let's call this the "Switching-Standard-Thesis" (SST). Second, and connected to the first, the threshold beyond which a certain kind of irrational behavior can be seen as pathologic varies considerably. Call this the "Switching-Threshold-Thesis" (STT).

The Switching-Standard-Thesis

According to SST the standard of rationality to which a person is expected to conform is the higher, the more she is suspected of having a psychiatric condition. As long as someone is regarded as "normal" her decisions may completely unreasonable in the eyes of others. As judge Lord Donaldson pointed out in an often quoted decision, the "right of choice is not limited to decisions which others might regard as sensible. It exists notwithstanding that the reasons for making the choice are rational, irrational, unknown or even non-existent" (Re "T", 1992). In a similar vein, Craig Edwards underscores that if someone ruins his reputation due to mental illness he may end up having to undergo involuntary psychiatric treatment, but if he does so without mental problems, it is his own business and he will not experience (strong) interventions (Edwards, 2009). While ordinary people are allowed to make irrational decisions even in highly important matters without being deemed incompetent (just think of decisions regarding the termination of treatment), patients with a suspected mental problem are at greater risk of being judged incompetent because of the very same "irrationality" (Banner, 2012). It is, therefore, a matter of normative choice and not one of objective judgment whether rationality is regarded as a component of mental health or not. It is usually being judged on normative grounds whether to examine someone's rationality further or not. If a mental disorder is suspected, we do; otherwise we don't. Irrationality is not the indicator of a mental problem. The dependency relationship runs the other way round: a suspected mental disorder is the reason why we take a closer look at someone's rationality and possibly regard a decision as irrational and incompetent that we otherwise would have accepted as competent.

The Switching-Threshold-Thesis

Here it is not asked whether someone's rationality should be subjected to deeper scrutiny or not, but whether irrational behavior

should be seen as indicating a mental problem. We all constantly behave irrationally in everyday life. It therefore has to be decided whether the irrationality of a person should count as part of a mental problem. Edwards lists a whole series of conditions such as greed, jealousy, hatred, or racial prejudice that impair our rationality and that "are sometimes considered to negative impact our well-being and that fall outside of our ability to control as rational agents, yet are not usually considered mental illnesses" (Edwards, 2009, p. 80). The threshold of rationality beyond which someone is being seen as having a psychiatric disorder is varying.

Both cases look very similar, and they indeed point to the same problem from different angles. According to SST, a mental disorder is diagnosed first, and subsequently a standard of rationality is applied that is higher than in everyday life. According to STT, irrational behavior that is judged to be normal on the background of one framework may be seen as indicating a mental disorder in other cases. The assessment of rationality is deeply normative.

MORALITY

I should stress once more that my claim is not that *all* psychiatric disorders are moral in kind. What I do claim is, nevertheless, that many conditions—or conditions in many circumstances—at least *involve* (morally) normative elements and thus cannot be purely value free, non-normative (objective) medical kinds. The moral side of ascriptions of psychiatric disorders is most obvious in Cluster B PDs. Louis Charland uses two arguments to show this (Charland, 2006): The "*argument from identification*" and the "*argument from treatment.*" According to the first one, Cluster B disorders are identified in the DSM through *explicit moral* terms and notions such as "lying," "lack of empathy," or "conning others." It would be hard to explain why a condition that is defined this way should not be moral in nature. His second point is only partly an argument on its own since it relies on the validity of the first one. What he has in mind seems to be that there is an important difference between, say, ceasing to be depressed on the one hand and ceasing to be a liar on the other. The difference is that the first case can be seen as a cure while the second case is "tantamount to a moral conversion" (Charland, 2006, p. 122).

Possible counterarguments to this account are not far to seek. First, one could argue that it is not the morally questionable behavior as such that defines the disorder but the respective person's inability to change it, her irresponsiveness to reasons. Even if this sounds comprehensible, on a closer look it becomes obvious that an immutability criterion like this one only makes sense in connection with a presupposed moral judgment. There is hardly any person in the world that can change her character traits from one moment or week to the other. Character traits which we would not even think of as pathologic can be as "hardwired" as a full-fledged "PD." Think of a particularly polite and attentive man who has become this way through his genetic endowment and parental upbringing. Every morning he tells himself to be a bit more selfish—but he just can't help it. He cannot change his style of behavior, but hardly anybody would suspect a psychiatric problem here. Both character traits as well as dysfunctions cannot be overcome just by choosing to do so. Second, the availability of therapeutic help or treatment that could be seen as a distinguishing factor is not a good candidate criterion either. Edwards emphasizes this pointedly when he states that the "need for, or availability of, treatment does not make something an illness any more than plastic surgery makes a crooked nose an illness" (Edwards, 2009, p. 81). Third, neither are character and dysfunction discernible through underlying causes since wicked behavior is equally due to internal and external biological influences and environmental conditions as mental disorder is. With the appropriate chemicals (or even brainwashing methods) you can "treat" grandma's joy, little Johnny's nosiness, or Martha's politeness as effectively as Bill's full-fledged depression.

Edwards, who regards the concept of psychiatric disorder as morally based, realizes this very tension. His way out is a catalogue of five criteria, each of which is necessary but not sufficient, together with the assumption that there is genuine moral truth in the world. His criteria, formulated as questions, are the following: (a) Is the condition harmful for the person who has it? (b) Is there any reason for legitimizing the condition as a character trait that one can choose to develop or maintain? (c) Is the condition one that can be discouraged through the inculcation of appropriate moral values during childhood? (d) Will applying moral responsibility to the condition help to uphold broader moral values in one's ethical system? (e) Can one have insight into the condition's effect upon oneself and if so, how difficult is it to take an active role in seeking treatment for oneself? (Edwards, 2009, p. 83 f.) As one can see, all five questions can indeed help only if they have answers that are not themselves contestable and/or relative to society, culture, and underlying moral creeds. With his reference to ethical truths Edwards may at least avoid the lurking diagnostic arbitrariness, even if that makes psychiatric diagnostics no less moral. Those however, who do not belief in objective moral truths, are still lost in the wilderness of psychiatric relativity.

In a strictly religious society being an atheist may be seen as a dysfunction of personhood; when our western societies still were (regarded as) strictly heterosexual, homosexuality was regarded as dysfunctional and, hence, a mental disorder; since productivity is highly valued in our busy and buzzing western societies, lack of productivity has become a part of the definition of mental disorders (Crowe, 2000, p. 73).

HARM AND DISTRESS

One could assume that harm is not a normative concept: if a person suffers she suffers, period. In the context of psychiatric diagnosis things are more complicated, however. A first crucial point that illuminates the normativity of harm has been emphasized by Fulford (2002). We just don't realize the value-ladenness of physical harm because most people regard, say, a broken leg as something bad and painful. Values that are shared by most people tend to hide themselves behind their commonness. When it comes to mental suffering our values diverge to a certain degree. Hence, it is not that bodily diseases are value-free whereas psychiatric disorders are value-laden. Both rest on normative assumptions. In one field we simply share them, in the other we don't. As Fulford writes:

> "Thus, the criteria for good and bad heart functioning, for example, paralleling 'good strawberries,' are largely settled and agreed upon, and this is true by and large of all the areas with which

> (acute) bodily medicine is primarily concerned. By contrast, however, the areas with which psychiatry is primarily concerned—emotion, desire, belief, motivation, sexuality and so forth—are all areas in which our values, paralleling 'good pictures,' are highly diverse." (Fulford, 2011, p. 3 f.)

The most prominent author to have included the concept of harm in his theory of disorder is probably Wakefield. According to his "*harmful dysfunction analysis*" (Wakefield, 1992) we first have a function of a certain mechanism that turns into a *dys*function if the mechanism does not properly perform the tasks it was designed for by evolution; and if this dysfunction is furthermore harmful for the respective person, then it becomes a disorder. It is therefore not enough to state a (physical or mental) mechanism's dysfunction, since there are lots of dysfunctions that are not seen as disorders[8]. On the other hand, we all experience many harmful things in life without regarding them as mental disorders. Harm, he rightly assumes, is a value concept because it is relative to cultural assumptions. While this is plausible, turning Wakefield's idea upside-down is plausible, too: It may well be that we first disvalue a condition as harmful and only then search—and find—a mechanism of some sort that has a dysfunction of some sort. This would only be impossible if we could have a look into God's (or the evolution's) model kit.

But there are even more normative aspects in the notion of harm. First, the harm criterion leaves open *who* has to judge whether a person feels harm and distress enough and whether it is pathologic in character. It is one thing to subjectively feel harm and distress, quite another is to judge whether distress is pathologic, and, if it is recognized as potentially pathologic, what degree someone's suffering must reach in order to warrant a psychiatric diagnosis. Second, particularly in the case of Cluster-B PDs it is often the social environment, i.e., other people, who experience harm due to the "patient's" condition while he himself feels fine. A successful, narcissistic person will probably feel no distress at all while the people around him may suffer considerably. Third, harm also can arise indirectly from one's acts and with a temporal delay. If someone in a manic phase makes highly risky and imprudent transactions, the "harm" will (a) be indirect because not the condition itself is harmful or distressing but its consequences may cause harm, (b) the harm caused may initially not represent a problem for the person in question but for his spouse or children, (c) whether a risky and imprudent financial transaction or its consequences should be seen as harmful is clearly nothing we can read off some diagnostic manual. Financial losses are to be judged economically, not medically. Even if the person later deeply regrets what she has done, it remains unclear what degree of regret will warrant a psychiatric diagnosis.

CULTURE

One of the most widely discussed issues in the philosophy of psychiatry is the impact of cultural varieties on the concept of psychiatric disorder. Do different cultures give rise to special forms of disorder experience? Are there mental disorders that are due to particular socio-cultural frameworks? These and other questions have been disputed for a long time. There is one tradition that takes cultural particularities into account. It is called the "emic" approach. In contrast, the "etic" account tries to explain human behavior independently of culture-specific features and to find general, universal traits (for a more detailed explanation of the terms see Morris et al., 1999). Even though human nature has some universal characteristics, there are underlying culture-relative assumptions that make the etic approach inappropriate for psychiatry.

The various normative elements implicit in the assessment of psychiatric disorder overlap, and much of what has been said above about the concept of harm, moral frameworks, and even the question of rationality could have its place in this section as well. Therefore, what I am going to do in this section is only to highlight the various cultural dimensions of psychiatry. These are assumptions and mechanisms regarding the *causes* of mental disorder, the impact of culture on *diagnosis*, specific differences in the individual *experience* of mental disorder, and last but not least the *evaluation* of behavior from the third-person perspective.

Causes

Culture or the character of a given society seems to influence the development and understanding of psychic problems both directly and indirectly; indirectly through the norms and social expectations the individual has to follow, directly through the expected ways of behavior which determine deviance. In an interesting article Catherine Caldwell-Harris and Ayse Ayçiçegi formulated a "*personality-cultural clash hypothesis*" according to which there is a correlation between personality-style, cultural character and mental health (Caldwell-Harris and Ayçiçegi, 2006). They state that "[p]ersonality traits associated with psychopathology will be most frequent in allocentrics living in an individualist society, and in idiocentrics living in a collectivist society." In collectivist societies where strict rules of social behavior have to be followed and social harmony is highly valued, people with an idiocentric (extremely individualistic) personality tend to have poorer mental health with high scores in paranoid, schizoid, narcissistic, borderline, and antisocial PDs. In individualistic societies, by contrast, a distinct allocentric (extremely collectivist) personality is positively correlated with social anxiety, depression, obsessive-compulsive disorder, and dependent personality. In addition to this indirect influence on mental disorder, there is a more direct influence, too. This can best be illustrated by Wakefield's account of cultural relativity:

> "Whereas social phobia is a real disorder in which people can sometimes not engage in the most routine social interaction, current criteria allow diagnosis when someone is, say, intensely anxious about public speaking in front of strangers. [...] This diagnosis seems potentially an expression of American society's high need for people who can engage in occupations that require communicating to large groups." (Wakefield, 2007, p. 154)

In sum, not only has the respective cultural setup an indirect influence on mental health, it also tends to dictate the boundary between the normal and the deviant on the basis of the expected values and virtues of its members. In this respect the impact

[8]I am only mentioning Wakefield's concept of "dysfunction" here without having room for a discussion.

of society on the concept of mental disorder is clearly normative. Whether the indirect influence, i.e., the personality-cultural clash, turns out to be directly normative under the surface after all remains an issue for further scrutiny.

Diagnosis

Culturally specific views on psychiatric problems are harder to detect in our era of mass migration and globalization than in earlier times with more stable national and cultural boundaries. Nonetheless, important cultural differences regarding mental disorders remain, to which I am only able to allude in the following. What is more, the culturally formed experiences of psychic problems are not only to be considered on the patient's side but also on that of the practitioner, as Laurence Kirmayer points out (Kirmayer, 2001). This has also been shown some years ago by a study that compared the diagnostic patterns of American and Japanese clinicians (Tseng et al., 1992).

Three points regarding psychiatric diagnoses should be stressed here. Firstly, many mental disorders indeed really "exist" in the sense that they are modes of experiencing oneself and the world which are extraordinarily burdensome. Secondly, experience and behavior can only be understood against the background of other people's behavior and experience. Social phobia, for instance, presupposes a social surrounding not only because it is the very object of the phobia but also because it constitutes the basis of comparison against which a person assesses her own experiences. Thirdly, since there are "real" disorders on the one hand and dynamic social expectations on the other, it follows that the boundary between average and deviant behavior cannot be but normative. This is *not* just due to epistemological limits. Those boundaries simply do *not exist* by nature. What should psychiatrists do who are in need of a boundary that does not exist? They have to define it themselves (with the help of their social community) and put up a sign that reads "Attention, you are leaving the normal sector!" Seen in this light it is hardly surprising that there appears to be an extreme variance of prevalence rates for, e.g., social anxiety disorder across cultures, ranging from 0.2% in China and 7.9% in the US to 44.2% in rural areas of Udmurtia, a Constituent Republic of the Russian Federation (Hofmann et al., 2010, p. 118). Even if this spectrum should be primarily due to differences in case finding methods and there is in actual fact no "real difference in major psychiatric disorders across cultures and societies" as Andrew Cheng assumes (Cheng, 2001), it nevertheless mirrors all the problems and dependencies of psychiatric diagnosis and, hence, the impact of cultural and other norms and values on it.

Experience

Are psychological problems all the same around the world? If they are, science may be in a position to explain them on a purely molecular level one day. Two very common examples shall suffice at this point for an illustration that this is a vain hope. First, it is well known—even though hotly debated—that depression in Asian societies is experienced more as bodily malaise by the persons affected. The western counterpart of this "somatization" is sometimes called a "psychologization" (cf. Kirmayer, 2001). The Vietnamese language, for example, does not even have words for psychiatry, schizophrenia, and depression (Phan and Silove, 1997). A similar striking cultural difference can be found in the case of social anxiety. While in the western cultural sphere this is connected with the fear of being harmed or offended, in Japan and Korea people are in fear of harming or offending *others* (taijin kyofusho). Admittedly, taijin kyofusho is—along with other culture-specific disorders—at least mentioned in the DSM as well as in the ICD, but whether it is the same social anxiety disorder as in the western world, maybe a cultural-specific expression of it, or a disorder in its own right, is still under debate (cf. Hofmann et al., 2010). If two psychological problems that are quite differently experienced by the patients in different cultures get explained with one and the same molecular configuration, does this not come down to a Procrustean bed into which diagnoses are forced? Both expressions of social anxiety arise from and are judged by social norms.

Evaluation

As repeatedly mentioned in this article, whether a certain kind of behavior or experience counts as deviant and (potentially) as a psychological problem is often (even though not always) due to specific socio-cultural expectations. Somebody who is "dynamic" in one cultural region may be regarded as offensive in another. Remember the abovementioned western girl in rural Turkey (or the other way round). Here, expectations of rationality, morality, harm and harming combine to a normative framework against the background of which behavior is assessed and disorders are diagnosed. That does not mean there are no culturally and normatively independent mental disorders at all. But it would nevertheless be a fallacy to deduce the thesis that norms do not play a significant role in the assessment of mental disorder from their undisputed existence.

ROUTES OF EXPLANATION

Three levels of observation are of particular relevance in psychiatry. These levels exist in other areas as well, but when it comes to mental health and the concept of mental disorder, they have particularly far-reaching implications. These are the *explanatory level*, the *phenomenal level*, and the *interventional level*. One might use "reflection" instead of "observation," but since "reflection" is in some sense too ambitious a word, associated with deep scrutiny and deliberation, "observation" is more adequate, as will become clear in the following.

Let's begin with the *explanatory level*. Here we find all the traditional models of explanation such as *the psychoanalytical* (Freud), the *sane reaction* model (Laing), the *labeling* model (Rosenhan), the *problems of living* account (Szasz), the biopsychosocial model (Engel), or the currently dominating *medical* model. It will make a considerable difference if you claim with Szasz that mental diseases just do not exist, assume with Rosenhan that it is largely a matter of labels, or if you search for purely biological causes. Each of these models of mental disorder constitutes a basic explanatory *norm* since there just is no higher level of objectivity from which we could assess the validity of one explanatory account or the other. Admittedly, we can (and do) use the effectiveness of an

explanation and its respective therapies as a criterion, but whether psychopharmacological means are the most effective ones is open to debate even today. Hence, everything depends on questions of the philosophy of science, ontology, causality and—on an even deeper level—on the question of what constitutes an explanation.

On the *phenomenal level*, what kind of behavior or experience indicates a mental disorder depends on all the factors discussed above. The phenomenal level is in itself independent of a particular mode of (causal) explanation. Often it is just a matter of tradition or even intuition. The important aspect is that pathologic behavioral deviance is assessed through its "being different."

On the *interventional level* mental disorders are seen in the perspective of therapy, i.e., a successful cure is already part of the explanation of a particular disease.

The *routes of explanation* come into play when we ask where to start in order to understand the nature of mental disorders. It is an interesting phenomenon that we may come to quite different results, depending on where we start. If we begin at the explanatory level, psychiatric disorders may disappear if we are followers of Szasz, or turn out to be purely physical if we adhere to the medical model. In the first case mental disorders cease to be, in the second they cease to be mental. In the first case we do not need a therapy, in the latter the therapy will probably be a pharmacological one. We will get similar "start-dependent" results with the psychoanalytical or the biopsychosocial model. What is important here is that what we assume on the explanatory level defines what we believe on the other levels.

The same holds true for the other routes. If we start on the level of interventions and make use of pharmacological therapies, we will probably come to the conclusion that psychiatric disorders are indeed something physical. In this case we are even in danger of getting ourselves into a circle: Why are pharmacological therapies indicated? Because psychiatric disorders are brain defects. How can we know that psychiatric disorders are brain defects? We can conclude this from the effects of our pharmacological therapies (cf. Valenstein, 1998, p. 222). To give a third and last example: If we believe some behavior to be strange and pathologic, we will surely find a cause of it at the explanatory level. So we have come full circle: Remember the quote from Szazs at the beginning, that "being kind to one's wife is not the sort of behavior to which we want to assign a causal (psychiatric) explanation."

EPILOGUE

The fact that our understanding of mental disorders is guided by several kinds of norms does not mean that these disorders do not exist. More precisely, on the one hand there is psychological suffering which can hardly be doubted in its existence, relevance, and "realness." On the other hand there are several cases of mental "disorder" which clearly rest on direct and indirect, open and covert normative assumptions. This has at least two consequences. First, psychiatric disorders are not "out there" and not to be understood as objectively discoverable entities that can always be separated from each other. The boundaries between normal and non-normal behavior and those between one disease category and the other are floating. Second, because of the normative nature of psychiatry, mental disorders cannot be completely reduced to neuronal or molecular processes. Again, more precisely: A mental state as such may well be reducible to the brain, but determining whether this very mental state is (part of) a *disorder* or not is nothing the brain sciences can do. Something will always be lost in translation.

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GENERAL COMMENTARY
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What is wrong with reductionism? On the normative nature of mental disorder

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A commentary on

Normative preconditions for the assessment of mental disorder
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The subject of Marco Stier's article seems to be well-known, as he addresses a prominent topic in the philosophy of psychiatry: the normative nature of mental disorders. Of course, Stier does not attempt to cover this issue extensively, since he focuses rather on the Irreducibilty-Thesis (IT) and tries to show that "psychiatric diseases are irreducible to the brain even if the mental as such may in principle be reducible" (p. 2). Admittedly, such anti-reductionism is not an uncontroversial position (for an overview of the debate see Perring, 2010, ch. 3). Here is not the place to go into too much detail in dealing with this approach, even though I have expressed my sympathies elsewhere (Rüther, in press). Much rather, I want to pick out three points which, in my opinion, wrongly find little or no consideration. The first point concerns a position which Stier ties to IT, namely social constructivism; the second point concerns the argumentative strategy used in defending IT. The third point finally is a general comment on the orientation of the debate, i.e., the question of what we should discuss when talking about the normativity of mental disorders.

THE CONSTRUCTIVIST-THESIS

It is a unique feature of the text that Stier does not stick only to the far-reaching topic of normativity, but also makes a connection to debates on objectivity. He concludes that if IT is true, "psychiatric disorders are not out there and not [to] be understood as objectively discoverable entities that can always be separated from each other" (p. 8). But what are they then? According to Stier, we are dealing with a social construction. For, "[N]o inner feeling has a sticker on it that reads "I'm a disorder!" We have to write those stickers ourselves and attach them to certain feelings and behaviors" (p. 2, also p. 3, p. 7). If we stick to these ideas, then it follows that we have to understand the concept of mental illness as non-objective or, put positively, as a social invention. This last thesis we may call the claim of the social construction of mental illness (CT).

At this point a number of follow-up questions arise, for instance, how one is to spell out the construction metaphor and how plausible this "spelling out" actually is. How does Stier deal with these matters? At times, it sounds as though Stier justifies CT by invoking a conceptual relation to IT: "Again, if the boundary between normality and mental disorder is a social construction [...], then the, disorderness' of a condition cannot be found on - and hence not be reduced to - the neuronal level." (p. 4) But it is clear (and hardly worth mentioning) that such a relation does not exist. In recent years, philosophers have drawn attention to the fact that the notions of normativity and objectivity are different (see the locus classicus Wiggins, 1976; McDowell, 1985, 1987). Thus, it requires separate arguments to defend CT. In Stier, too, we find passages in which this is acknowledged and at least one such argument is to be found. This argument is well-known in the debate and starts with the descriptive assumption that the question what mental illness happens to be, is relative to a given context. This can be seen in various discourses, for instance, by researching the causes (p. 6), diagnosis and explanation (p. 6 et seq.), or the experiential quality of mental illness (p. 7 et seq.). However, we should recognize the following state of affairs: It seems suspect to make claims about the nature of mental illness by referring merely to of how mental illness is understood de facto. Empirical judgments and judgments about the nature of mental illness are independent of one another. A difference that Stier explicitly concedes and makes use of himself (see his defense of IT below). So, perhaps we should understand the pointer toward empirical variance in another way, maybe not as a direct inference to CT, but as a call for explanation. In this case, one might claim by abduction that CT is the *best* explanation for the factual diversity. Indeed, we can find several indications for such a reading, for instance, when Stier explains the "extreme variance of prevalence rates for, e. g., social anxiety disorder" (p. 7) by the fact that psychiatrists themselves define what an illness is by "put[ing] up a sign that reads, Attention, you are leaving the normal sector!'" (ibid.) But is such an inference really persuasive? I have doubts, in particular, because I cannot see that the objectivist counter-position has a *worse* explanation (see e.g., Rüther, 2013, ch. 13.1). Why shouldn't we claim that many divergences are based on distorted patterns of perception, for instance, on psychological, semantic or logical fallacies? In most cases this would be even more intuitively cogent than using the metaphor of construction. In this manner, it seems that the constructivist can at most achieve an argumentative draw. Ideally, he can offer an explanation that is comparable in quality to that of the objectivist. But if things are like this, doubts arise whether the difference-argument for CT actually can

reach its aim. The mere fact that different beliefs about mental illness exist is not a sufficient reason to take constructivism to be true.

THE ARGUMENT FOR ANTI-REDUCTIONISM

As we have seen, Stier's main aim is to defend IT. He writes: "All I want to show is that mental disorders cannot be determined in a purely physical way" (p. 2). This is not a modest aim, but rather a highly complex one and the literature on the topic is vast. Seen from this angle, one might expect a detailed engagement with proponents and opponents of IT. But taking a closer look at the text, we get a different picture. For the most part, the text points to fields and areas in which we can assume that psychiatry carries heavy normative baggage (key words are: "frame of reference," "normative dimensions of psychiatry"). Accordingly, the text does not, strictly speaking, argue for IT, but offers a description of the normative phenomena at issue. Of course, this description is also a comprehensive project, and Stier's extensive and sophisticated comments are worth noting in this manner. Yet, pointing to our phenomenology is not sufficient for his claim that IT is true. What we do need is not only a description of the data, a common ground to which reductionists and non-reductions can apply their approaches. We also need *a reason* that counts against reduction. But at times, Stier comes close to the claim that assembling normative preconditions is enough. For he speaks of the "normative bedrocks of mental disease" (pp. 2–8) and claims that "[i]t is in principle impossible to get rid of this normative aspect of the task, even if the underlying biological mechanisms of a particular behavior or experience were completely known" (p. 4). In this regard, Stier owes us a reason why the irreducibility of the normative might be the best explanation for the discussed phenomenon, particularly if one takes into account the present state of the debate and the extensive literature on the various counter strategies of the reductionist.

THE FOCAL POINT OF THE DEBATE

However, why should we bother with the complicated dialectics between reductionism and anti-reductionism in psychiatry at all? Might it not for some reason be sufficient to point to the normative preconditions and assume that this is enough? Surely, for some purposes it might be sufficient that we do not have to get entangled in the reductionist counter arguments. Nevertheless, I would suggest that there is at least one reason to deal with these arguments. This is mainly that we can get a grip on the matter of what really is at issue when we spell out the dialectic between the two opponents. And if we do so, we can see that both parties, at bottom, are actually arguing about different conceptions of *how* to analyse philosophical problems. This is, of course, not a novel conception of what the debate on reductionism is about (see e.g., Keil, 2008). The reductionist is, at least in a common reading, a philosopher who tries to accommodate the phenomena in question into a natural framework which is mainly investigated by the natural sciences. In contrast to that, the anti-reductionist is suspicious about this unification and rejects this as a vicious simplification that has unbearable costs, e. g., it leaves something important out or, even stronger, comes close to a self-contradiction. Framed in this way, we can see that the stakes are higher than we might previously have assumed, for now we are concerned not only with questions internal to the philosophy of psychiatry, but with fundamental questions about the nature of philosophy and its methodology. Perhaps, this background also explains Stier's implicit target and the intensity with which he, and others with him, reject reductionism. Of course, questions like these are complicated and call for further explanations of the terms already used, for instance, the reductionist terms "method," "natural framework" or "modern science." But in any case, it is worth being aware that these explanations also lead us away from the narrow philosophical context of psychiatry and point toward a metaphilosophical reflection.

CONCLUSION

What has been said so far? First, it was shown that Stier's constructivist claim is not sufficiently supported. It can neither be deduced conceptually nor is the proposed auxiliary argument of any help. Here we obviously need further arguments in order to support it.

Second, it was noted that the claim of irreducibility is explicated but not defended. It was shown in which way normativity plays a role, but not why it resists any form of reduction. Here one should draw out the dialectic and hear the reductionist position.

Third, it should have become clear that the debate on reductionism is not only a debate specific to the philosophy of psychiatry, but belongs to the wider field of metaphilosophy. The debate about IT is, at its core, concerned with the nature and methods of philosophy. Thus, if one wants to follow up on the debate about normativity in psychiatry, here might lie a field for promising future research.

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On the concept of the normative in the assessment of mental disorder

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In Marco Stier's article "Normative preconditions for the assessment of mental disorder," the concept of the normative occupies a central role (Stier, 2013). Stier states that mental disorders have an irreducible normative element built in, expressible through various "normative frames of reference" they are tied to. Following his two main theses, he thinks that these frameworks shape what counts as deviant as well as non-deviant behavior. He takes this as evidence that we have to specify mental disorders at the mental level, and thus will never be able to give a purely physical account of them.

Unfortunately, he nowhere makes clear what he takes to be the content of the concept of the normative, although he gives some hints about his understanding at various passages. In what follows, I will explore three of his implicit suggestions on the essential linkages his concept of the normative bears to other concepts: the non-natural, the non-objective, and the relative. I shall argue that it is questionable that this understanding leads to the conclusion Stier aims at—that the specification of mental disorders cannot be succeed on the physical but only the mental level due to the impact of normative considerations in this enterprise.

THE NORMATIVE AND THE NON-NATURAL

Regarding the relationship between the normative and the non-natural, Stier argues that the normative cannot be grasped in naturalistic terms. Integrating this alleged fact into his ontological dichotomy between the mental and the natural, it follows for him that the normative must belong to the realm of the mental, for "[t]here seems to be something peculiar about behavior that is beyond purely physical explanation because the difference between, say, acting kindly and unkindly can hardly be grasped in physical, non-normative terms" (p. 1).

Both thoughts appear to be problematic. With respect to the first, there is a whole bunch of philosophers out there that intend to explicate all kinds of normative facts related to human behavior in naturalistic terms, therefore reducing the normative to the natural. What Derek Parfit calls "Analytical Naturalism" (cf. Parfit, 2011, p. 295) precisely aims at redefining normative notions in terms of natural notions. Parfit mentions Nicholas Sturgeon and Frank Jackson as prominent proponents of this type of naturalism (Parfit, 2011, p. 365).

Whether these people are right or wrong is certainly subject to discussion, but their efforts at least suggest that taking phenomena at face value and not even mentioning competing accounts can hardly be the adequate strategy. Indeed, Stier himself gives a prominent example of a *prima facie* irreducible ontological domain besides the physical: The mental itself is often seen as an important challenge for the hard-boiled naturalist. Nevertheless, Stier grants (at least for the purpose of his paper) that "every single aspect of our mental and behavioral life could be explained in purely physical terms" (p. 2)—from which he of course explicitly excludes mental disorders. But if the vast majority of mental phenomena could be explained in natural terms—why should not the same be possible for normative ones?

Secondly, there seems to be no intuitive way for marrying the fact that something is non-natural with the fact that it is mental. A great many deal of things that may serve as paradigmatic examples for non-natural entities are not perceived as necessarily being mental in the way that is of interest here: God, the number seven, or human dignity—the latter being clearly a *normative* idea. God, for instance, is almost by definition non-natural, but his concept surely does not demand from us that he has to be imagined as something mental.

Indeed, that a certain normative entity cannot be grasped in natural terms does not mean that it has to be reducible to any other domain at all. Alternatively, the normative aspects of mental disorders that prove not to be analyzable in natural terms can be just that—irreducible normative aspects. For large parts of the normative domain, this is a common option. Russ Shafer-Landau, for example, argues that we should "introduce into our ontology a *sui generis* category of values" that can explain the normativity found in morality (cf. Shafer-Landau, 2003: 55). If one succeeds in providing a similar account for the normative aspects of mental disorders, there is no need to suppose that mental disorders have to specified at the mental level.

THE NORMATIVE AND THE NON-OBJECTIVE

Let us consider next the non-objective. When analyzing the various sorts of normative frames of reference, Stier frequently uses his assumption of a certain fact's being normative as a reason that is must be non-objective.

I will confine myself to two examples. When investigating evaluations of rationality and their bearing on the attribution of mental disorders, he states that "[i]t

is [...] a matter of normative choice and not one of objective judgment whether rationality is regarded as a component of mental health or not" (p. 4). Since we often use irrational behavior as an indicator for the presence of a mental disorder, the attribution of the latter, if based on the former, also becomes a non-objective judgment.

One of Stier's arguments for this verdict is based on the general observation that one and the same person can be subject to a different treatment by others and the authorities, depending on whether a certain irrational behavior by her is regarded as due to a mental disorder or not. Thus, according to Stier's "Switching-Standard-Thesis," judgments of rationality are not an "objective" standard for measuring one's mental health.

But this argument fails, for all Stier's observation shows is that depending on the supposed source of the irrational behavior (which might be subject to an argument from the best explanation), our reaction varies: If we have reasons to think that the person's irrational behavior is due to a psychological urge she does not recognize as part of her own personality, we are rightly more eager to intervene. Therefore, it is not as if the normative judgment about the person's rationality does change its validity from "true" to "false," or as if the fact that she behaved irrational is somehow ontologically dependent on subjective elements; the change is only in the practical reasons both provide us with. And we have heard no argument that these reasons are not objective, only that their content depends on further considerations whose status is yet unclear.

Stier discovers an analogous fault when moral considerations are used for the assessing of mental health: "I do claim [...] that many conditions [of psychiatric disorders]—or conditions in many circumstances—at least involve (morally) normative elements and thus cannot be purely value free, non-normative (objective) medical kinds" (p. 5).

Stier's assumption that many kinds of mental disorders have a moral evaluation built in and are thus not "objective" in the sense of "out there independently of our subjective evaluation" seems to confuse people's opinions about the right morality with morality itself. He writes, for instance, that "[i]n a strictly religious society being an atheist may be seen as a dysfunction of personhood" (p. 5). True – but unless one subscribes to a flat subjectivism of the form "thinking that something is right makes it right," *opinions* about morality are not necessarily true. Consequently, a diagnosed mental disorder partly based on observed moral misbehavior might be just false instead of arbitrarily (but nevertheless correctly) attributable.

THE NORMATIVE AND THE RELATIVE

Finally, I want to take a look at Stier's assumed relationship between the normative and the relative. He uses the concept of the relative at various places and cites it as evidence that the value laden nature of mental disorder cannot be regarded as open to a purely natural analysis of the former. In sum, he states that "[p]sychiatry is guided by social, moral, cultural and other norms. If this is true, and if it is also true that these kinds of norms are relative to time and place, then psychiatry cannot claim to know what a mental disorder is "in itself," where normality ends and mental disorder begins" (p. 3).

One passage where he substantiates this last quotation is his explication of the influence of *cultural* relativity for what counts as mental disorder. He distinguishes a direct from an indirect form of this influence. The former refers to the "cultural setup" of a society that creates norms its individuals have to follow. Depending on their personality, individuals might find it very difficult to cope with the expectations expressed in these norms and thus receive higher scores on certain criteria for specific psychological disorders.

That the frequency of the occurrence of psychological disorders correlates with cultural character and personality is hardly surprising and "relative" only in the most uninteresting sense of the term. It is a well-known fact that water's boiling point is dependent on a number of factors, including air pressure. This does not prevent it from being a perfect natural as well as objective fact so far. And neither cultural character nor personality is something that can be influenced at will by the individual to any degree relevant for most psychological disorders. Following this reasoning, Stier's demand that psychological problems have to be "all the same around the world" to receive a scientific explanation (cf. p. 7) is far too exaggerated.

With "direct influence," Stier points to the thesis that culture characteristics may not only trigger the development of mental disorders in people with certain personalities; it also "tends to dictate the boundary between the normal and the deviant on the basis of the expected values and virtues of its members" (p. 6). Again, the example Stier uses makes only sense when assuming that there is no way to evaluate the criteria put forward for mental disorder in a given culture in terms of their appropriateness. That "[s]omebody who is "dynamic" in one cultural region may be regarded as offensive in another" (p. 7) just does not tell us whether she indeed *should be regarded* as offensive.

Stier thinks that there exists no "final answer" to this question, stating that different models of explanation in psychology all constitute "a basic explanatory *norm*" for determining what a mental disorder is. Thus, "there just is no higher level of objectivity from which we could assess the validity of one explanatory account or the other" (p. 7). To justify this bold thesis, he argues that even seemingly "hard" criteria such as the "effectiveness of an explanation and its respective therapies" cannot provide us with a solution, since which of the competing models of explanation "are the most effective ones is open to debate even today" (p. 8). However, the fact that a debate is still in an ongoing state as such is no legitimate criterion for thinking that it never can be closed via rational exchange, as proponents of the argument from relativity already admitted 30 years ago (Mackie, 1977, 36f.).

In sum, seeing mental disorders as having certain "normative aspects" does neither have to mean that they are only explicable at the level of the mental, nor that they cannot be stated objectively, nor that they exist or are recognizable only relative to cultural norms. The failure to provide a throughout "natural" characterization of them may be completely compatible with the view that

they are, as Stier puts it, "out there" and have to be discovered rather than construed. And inasmuch not every normative aspect may be in need of being reducible to the natural, the mental, or anything else, nothing might be lost in translation.

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Mental disorders, brain disorders and values

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The debates about the normativity of mental disorders and about the distinction between somatic and mental disorders have long been closely linked. This is very obvious in Szasz, who claims that there can only be brain disorders, no mental disorders and that so-called mental disorders are really problems in living. The implication of the latter claim is that people who have mental disorders are really people whose behavior and emotions depart from societal expectations. One might therefore be tempted to think that the normativity claim and the claim that mental disorders are really brain disorders stand and fall together. This is indeed what Stier claims. "Because of the normative nature of psychiatry, mental disorders cannot be completely reduced to neuronal or molecular processes." (Stier, 2013, p.8)

But how close is the link between normativity and irreducibility really? I agree with Stier that ascriptions of mental disorders are intrinsically normative, and that what counts as a mental disorder has to be decided at the mental rather than at the brain level is also correct. However, the normativity claim and the claim that physicalism does not imply that all mental disorders are brain disorders can and should be separated for two reasons: First, we do not need the appeal to value judgments to justify the importance of the mental level in description and explanation. Second, we need to invest significant normative judgments in any kind of ascription of disease or disorder, not just in the range of the mental.

MENTAL DISORDERS AND BRAIN DISORDERS

As Schramme (this issue) and others rightly point out, we cannot do without the mental level of description because the decision what counts as dysfunctional is made at the level of behavior and mental states. This holds even for disorders which are commonly understood as brain disorders, such as for example Alzheimer's. Alzheimer's counts as a disorder because of the problems with memory it is associated with. The importance of the mental level of description secures its continued relevance: "The claim that all instances of S have the property of realizing a disordered neurophysiological process is only possible at the psychological level of explanation" (Schramme, 2013, p. 5). It is therefore conceivable that we might end up being able to identify the physiological correlates of various mental disorders while still classifying these as mental disorders, as what marks these conditions and their corresponding brain states as disordered is a psychological defect.

One might think that the fact that mental disorders are physically based implies that mental disorders are *ipso facto* brain disorders and that what counts as a brain disorder becomes dependent on what counts as a mental disorder. In this way, the close link between mental disorders and brain disorders would be retained, but the concept of brain disorder would become dependent on that of mental disorder. But this inference is not licensed. It presupposes that there cannot be separate criteria for what counts as malfunction in the brain and what counts as a malfunction in the mind and that patterns of dysfunction can be pursued all the way down. However, this is by no means conceptually necessary. As has been pointed out in the literature, physicalism does not entail that where there is mental dysfunction, there is always a physical dysfunction. The computer analogy is sometimes invoked to illustrate the point that just as software problems do not imply hardware problems mental problems do not necessarily imply corresponding physical problems. As Boorse puts it "Whether and how a computer program, or a mental state, is dysfunctional need not be evident from any of its physical properties" (Boorse, 1976, p. 68).

This is not to say that there cannot be a close link between what is labeled as psychological dysfunction and what gets specified as brain dysfunction. While there are some clear cases of brain problems such as lesions which are specifiable without reference to the mental level, some of our conceptions of brain dysfunction are derived from the psychological level, rather than from independent conceptions of what counts as a brain malfunction. An example for this is the fact that the anomalies in the functioning of the amygdala found in psychopaths are labeled as dysfunctions because of the mental and emotional impairments they are associated with.

To summarize, whether dysfunction at the mental level is best described as dysfunction at the brain level is an empirical issue and it is by no means clear that very strong correlations can always be established. It may well be that in some cases, the way a certain disorder is realized in the brain is so disparate that no explanatory value is achieved by labeling the mental disorder in question as a brain disorder. Whether something should count as a brain disorder or a mental disorder will depend not on considerations regarding physicalism and the ultimate nature of the mind-brain relation but on what is explanatorily primary, the level of the mental or that of the

physical. This is not a new thought, and it has been forcefully argued for in the context of the autonomy of the special sciences (cf. Fodor, 1974). But it is worthwhile reminding people that diagnoses of mental and brain disorders cannot simply be inferred from our ontological commitments but serve the purpose of explaining and treating these disorders. The importance of the first point has been stressed by Schramme and Stier, I hope to have helped shed some light on the latter.

NORMATIVITY

I now want to turn to the normativity of mental disorders, a point Stier is particularly focused on. What he has in mind is not merely that all conceptions of disorder are at least minimally normative because they make reference to such notions as dysfunction and "correct functioning." Rather, Stier endorses what he calls the stronger claim that psychiatry is guided by social, moral, cultural and other norms. That psychiatry is de facto a value laden discipline and that psychiatric diagnoses are to an extent dependent on the values that practitioners in the field and the surrounding society endorse is undeniable. I take it that Stier does not merely intend this as a true but fairly innocuous description of psychiatric practice. Rather, to have any philosophical bite, the claim must be that this is an essential feature of psychiatry which cannot be given up (see Rüther's comment in this issue).

One might think that subjectivity in norms is unproblematic if there are no objective values in the first place. According to this line of thought, a subjective or culturally relative diagnosis could only be wrong if there was an objective standard against which it could be measured as wrong. If this is not the case, then there is nothing troublesome in the difference of values which lead to different conceptualizations and classifications of disorder. However, I do not believe that a strongly relativist perspective is internally coherent. It would assume that the cultural relativist would have to concede that what is a disorder in their own society is not in another. For example, a homosexual would change from being disordered to being healthy simply by moving from a culture where homosexuality is seen as a mental disorder to one where it is not. But it does not seem credible to me that anyone would actually concede this degree of relativity for their own ascriptions of disorderedness. A certain amount of vagueness is of course unavoidable, but when we ascribe mental disorders, we try to get it right and worry that we may end up wrongly labeling something as a disorder.

The knowledge that ascriptions of mental disorder are in fact often culturally biased is troublesome because diagnosing someone with a mental disorder has far reaching practical consequences. If people can be sectioned because of acute mental disturbance, we do not want decisions as to what constitutes mental disturbance to be culturally arbitrary. Otherwise, social deviance could be labeled as mental disorder and used as a way of stigmatizing or discrediting people, or even withdrawing their personal freedom. For all of its weaknesses, the general definition of mental disorders in the DSM tries to address this issue, stating that "Socially deviant behavior (e.g., political, religious, or sexual) nor conflicts that are primarily between the individual and society are mental disorders unless the deviance or conflict results from a dysfunction in the individual, as described above"(American Psychiatric Association, 2013, p.20). Unfortunately, this does not really solve the problem, as it brings us back to the question what a dysfunction is. There needs to be a standard according to which we judge whether calling a certain condition pathological is valid or not.

The consequence some writers, in particular Christopher Boorse, have drawn from this is that we need to purge psychiatry of values and endorse a scientific notion of disease, whereby any residual normativity can be explained in biological terms. He proposes that we can define disease in terms of dysfunction and dysfunction in terms of deviation from normal species-typical functioning. Normal functioning is described as typical contribution to survival and reproduction. The problem with this type of account is that it only seemingly avoids normativity. Unless it is a purely statistical notion, talk of dysfunction presupposes that there is something the mind or body should be doing, not merely something it normally does. A statistical notion of dysfunction and pathology is too thin to be useful for medical practice. This does not mean that it cannot be extremely useful in describing various conditions. But arguably, the move from describing something as anomalous to describing it as a dysfunction always requires a commitment as to how something is supposed to function which goes beyond observations on what is statistically normal.

Fortunately, we need not assume that being evaluative and being objective are mutually exclusive unless we are given a convincing argument to the contrary. Rather, we can look for some specification of harms ensuing from anomalous mental conditions which we can use to justify that a certain condition is indeed a mental disorder. An example for such an attempt is Gert and Culver's proposal for a definition of mental disorder drawing on the list of harms given in the DSM IV TR. They point out that the list only contains states considered to be harms cross-culturally, such that there is an objective, broadly shared notion of harm. "The agreement of rational persons in all societies about the universality of the basic harms is extremely important, for it establishes the objectivity of the concept of a disorder" (Gert and Culver, 2004, 421f.). It should be pointed out that, just as de facto disagreement and subjectivity does not automatically entail the subjectivity of all value, neither does de facto agreement automatically establish objectivity. Nevertheless, a theory of value does well to take the things people actually value and agree on as a starting point.

In conclusion, ascriptions of disorder are normative, but we should strive for objectivity in our evaluations. Furthermore, there is no need to fear that advances in the brain sciences will make the level of the mental redundant.

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Antireductionisms with regard to mental disorders: some caveats. A commentary on Marco Stier

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Keywords: mental disorders, non-reducibility thesis, medical model of mental disorder, psychiatric diagnostics, value-dependence

INTRODUCTION

With his article "Normative preconditions for the assessment of mental disorder" Stier (2013) is presenting a thought-provoking piece of work and I agree with many of his conclusions. This is certainly true of Stier's main thesis that the demarcation line between mental health and mental disorder cannot plausibly be gained on the level of neurobiology alone, but is in need of additional value judgments. However, I think that this specific "antireductionist claim" holds true also in somatic medicine. Hence, the "medical model," rightly understood, seems to be fully appropriate for assessing mental disorder. Moreover, I suggest to be very restrictive in discussing the concept of psychiatric disease in the language of reductionism, since this might, contrary to Stier's own intentions, be easily misunderstood as water on the mills of methodological antireductionism in psychiatry.

SETTING THE STAGE

Making use of Ayala's (1974) influential differentiations between reductionism (and the corresponding debates) on the levels of metaphysics (ontology), epistemology, and methodology, reductionist concerns vis-à-vis psychiatry primarily refer to the last level. As a practical science, psychiatry is mainly concerned with *methods* or strategies of preventing, ailing, or curing mental disorders. These strategies in turn are interrelated with methods of properly explaining and diagnosing such disorders. In contrast, whatever psychiatrists or their critics hold on the level of ontology or epistemology seems relevant to psychiatric work (only) in so far as it determines outlooks on methodology—especially in interacting with patients and in treating their disorders. When it comes to the latter, matters of causation play the crucial role. And here, I urge, one should distinguish between two questions: (i) how mental dysfunctions (e.g., delusions, depression, mania, decrease in cognitive functions etc.) is/can at all be "caused" by brain dysfunction; (ii) how relevant systemic brain dysfunction is caused by neurobiological processes on lower levels—e.g., on the levels of circuits, cells, or genes.

The first question points to the central and perennial problem of the mind-brain debate and from here cuts throughout psychiatry (so also Kendler, 2008, p. 9). For these problems and questions, it ultimately does not matter whether we talk about healthy or disordered minds and brains. I do not know whether psychiatry might make a genuine contribution to solve these problems. Likewise, we most often do not know what proponents or critics of biological psychiatry hold in these matters. Beyond the shared views that the "mental realm," disordered or healthy, is (a) both very real and very important to ourselves and (b) brain-based, there exist many conflicting views and intuitions. Key problems seem to be the questions of mental causation, agent causality, and free will. In this paper, Stier does not address them in their own right, but he suggests assuming full explicability of the mental in "purely physical terms" (p. 2).

The second question lies at the bottom of what mainstream neuroscience, and in fact life science in general, is doing today. Here, scientists successfully try reductionist strategies to count for certain biological phenomena by explaining them on a relatively lower level (circuits, nerve cells, synaptic spaces) and by isolating them from as many relevant background conditions as seems fruitful[1]. Here again, Stier is ready to accept—if only for the argument's sake—"that environmental influences, too, are explicable mechanistically" (p. 2). Making such (non-eliminative) reductionist assumptions upon both questions, he rightly emphasizes that the truth of his "anti-reductionist claim" regarding the notion of mental disease does not depend on metaphysical or methodological anti-reductionism with regard to the mental.

A PARTIALLY NORMATIVE CONCEPT OF (MENTAL) DISEASE

Stier holds that "mental disorders cannot be completely reduced to neuronal or molecular processes" (p. 1). His justification for this "anti-reductionist claim" is the above stated "main thesis" which holds that in the field of neuropsychiatric disorders, the borderline between health and disease is value-laden. Unable to argue for this in any detail, I wholeheartedly agree with the view that the concept of mental disorder is partly normative. Being mentally diseased means (or should mean) to be in some or other dysfunctional and unwelcome mental state that thus should ideally be prevented or treated. Imprecise as these stipulated evaluative criteria and their originators are, I also agree with the view that individual and social value judgments cannot be read off from mere neurobiological facts[2]. We principally cannot tell from scratch

[1] See Kaiser (2011) for a diligent analysis.
[2] See Barker and Kitcher (2014), p. 70ff. for a defense of value invention.

whether some functional neurobiological state corresponds to a mental disease or not. Rather, we can only do so within a partly evaluative background frame.

However, do these insights not hold true for diseases in *general*, for disorders within *and* without psychiatry? For so-called somatic disorders, this might not always be as obvious as in the realm of psychiatric diseases. Take an infection that, if untreated, would rapidly lead to death without any other adverse symptoms. One might argue that premature death is a purely descriptive term independent of it's being unwelcome to most people. But the same could be said about neuropsychiatric disorders that lead to permanent coma or benign delusions. Where single disorders, in the mental as well as in the non-mental sphere, *seem* to be explicable without recourse to values, the gist of the whole concept of disease refers to unwelcome malfunctioning (including the functions of living or being conscious) and can be traced, I think, in each of its subtypes. Unable to further argue in favor of a partly normative concept of disease at this occasion, let me at least emphasize that this is one of the standard views (often referred to as partial "constructivism") in the contested field of theories of health and disease (see Murphy, 2008). The current tendency to blurr or to give up the distinction between psychiatry and neurology could, by the way, be seen as yet another indicator for the non-exceptionalist status of mental disorders (see Perring, 2010).

THE MEDICAL MODEL

Stier refers to the "medical model" (MM) without giving a complete explicit definition. In the literature, MM is indeed a commonly used paradigm; it is seen, however, to allow for "minimal and strong interpretations" (Murphy, 2010, pp. 3–13). Stier's understanding of MM comes in pieces. On a purely descriptive level it is said to stand in competition with psychoanalytical and other explanations of mental disorders (p. 7) and to substantially parallelize body-environment interaction in the genesis of cancer and brain-environment interaction in the genesis of depression (p. 2). Critically, MM is accused of inadequately explaining psychiatric disorders: "psychiatric disorders [. . .] may turn out to be purely physical if we adhere to the medical model [. . .] and cease to be mental" (p. 8). But why should this be the case?

One possible answer could be MM's alleged tie to a value-neutral concept of disease. However, this is not only contested by many and with good reasons (see above), but also by Stier himself. He clearly admits that "[. . .] it is not that bodily diseases are value-free whereas psychiatric disorders are value-laden. Both rest on normative assumptions." But then he continues: "In one field we simply share them, in the other we don't" (p. 5). Both observations of the last sentence seem questionable: Quite a number of "bodily" conditions are contested with regard to their "diseasedness"—e.g., limited reproductive or sexual functions, moderately decreased hearing, or moderately diminished memory capacities in "normal aging." Arguably, it is normative aspects that will determine demarcations. In any case, MM does not seem committed either to value neutrality in the concept of disease, or to the indisputability of the underlying values. On the other hand, value dissent in the psychiatric domain is by no means ubiquitous. After all, delusions, anxiety disorders, depression, or addiction do not appear very attractive, neither from inside nor from outside.

Hence, contrary to Stier, MM should in my eyes be properly understood as *rightly* holding a thoroughgoing non-exceptionalist view toward the explicability of psychiatric disorders. This view indeed seems to be the main stream position in neuroscience. It implies optimism with regard to neuroscientific contributions to diagnostic and therapeutic progress in psychiatry. But, again, it does neither imply viewing the concept of psychiatric disorder as value-independent nor viewing the mental realm as eliminable by neurobiological approaches.

PSYCHIATRIC DIAGNOSTICS

Suppose, you diagnose an individual patient with certain symptoms as suffering from mental disorder Z. In an idealized nutshell this presupposes: (1) a multi-dimensional demarcation between mental sanity and mental diseasedness, where those symptoms indicate disorder; (2) a taxonomy of *specific* psychiatric diseases, one of them called Z; (3) valid *indicators* and tests for Z; (4) positive *testing* for indicators of Z in the concrete patient. Each of these steps has its problems. But only (1) seems value-dependent in the way described by Stier, i.e., relative to human flourishing and human interests. With regard to (2) there is malleability and ongoing change in both the bodily and the psychiatric dimension of medical practice: fine-tuning and re-tuning according to some symptoms or other, to locations, or to (assumed) underlying causal paths. The main values that reign nosology are coherence and therapeutic success, I think. (3) is, again, an ongoing process according to medical evidence, having repercussions to (2) and being reigned by the very same values of coherence and therapeutic effectiveness. Finally, diagnosing a given patient should involve testing her according to best available parameters, with results of presuppositions (1) to (3) in the back. Hence, in psychiatry, a patient showing up with certain behavioral symptoms could conceivably be tested for neurobiological indicators, resulting in the diagnosis Z—without loosing sight of the mental. Determining a mental disorder in this way is not guilty of any problematic reductionist credo.

THE INNER LIFE OF PSYCHIATRIC PATIENTS

Granting potential causal relevance to a multitude of external influences, psychiatrists would finally be ill advised to look for brain function in isolation rather than in context. But turning external effects—e.g., psychologically stressful life events—into background conditions of pathogenesis, does not imply neglecting their causal role. Nor does it imply ignoring the importance of preventing such adverse factors in the first place, or excluding psychotherapy from the agenda of psychiatry. Likewise, nothing in a methodologically reductionist approach to psychiatric research compels scientists or doctors to ignore or belittle the enormous importance of patients' conscious experiences. If such unfortunate "practical reductionisms" nevertheless occur, they can neither legitimately be nobilized nor criticized as a sequel of biological psychiatry.

From all we know and foresee, detailed knowledge about one's inner mental life needs first-person experience or, as a weak approximate substitute, third-person encounter. Listening to psychiatric patients' directly or indirectly describing their subjective experiences thus seems irreplaceable for assessing the subjective impact of mental disease as well as for an understanding interaction with patients. Nevertheless, using neurobiological tools for diagnosing and monitoring treatment might in principle be possible and helpful.

SUMMING UP

Stier holds that the classification of certain mental states as disorders is value-dependent and therefore cannot be read off from neurobiology. Contra Stier, however, this plausible view does in no regard discredit the medical model (MM) as "the one and only bedrock of psychiatry" (p. 1). Rather, MM is uncommitted to a naturalist theory of disease. As Stier himself admits, values can be seen as indispensible also in demarcating bodily diseasedness. Some of these diseases and values might be as contested as in psychiatry. MM's upshot is a non-exceptionalist view on the explicability of psychiatric disorders—and subsequently on their diagnostic and therapeutic in-principle accessibility on a biological level.

Finally, framing and selling a partially constructivist position regarding (mental) disease as an *anti-reductionist* view, is both unusual and misleading. Affirming such constructivism should not get confounded with common and problematic objections that blame biologically oriented psychiatry as metaphysically or methodologically reductionist. Yet another distinct problem might be an unfortunate practical negligence of patients' inner life within modern psychiatry. Such "practical reductionism" can and should be defeated *within* a neurobiological orientated psychiatry.

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Medical criteria of pathologicity and their role in scientific psychiatry—comments on the articles of Henrik Walter and Marco Stier

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The articles of Walter (2013) and Stier (2013) refer to some central problems of philosophy regarding the concept of disease. Among them are the questions whether psychiatric diseases exist at all, how they are to be distinguished from mere problems of everyday life, which underlying theoretical concept of disease is assumed, and what kind of normativity—if any—is associated with their definition. In this commentary, I am going to commemorate the way medicine in general identifies and defines diseases and kinds of diseases, and the role theoretical concepts like *pathologicity* and *disease entity* play in medical epistemology and concept formation. The fundamental ideas and principles of modern medical pathology (German: *Krankheitslehre*) are the following ones:

1. Diseases are circumscribed, partial processes and conditions of the life process of human individuals as a whole. They are not a necessary part of life, i.e., a life without any disease or pathological condition is theoretically possible. Processes and conditions that are necessary, inevitable parts of life (e.g., developmental stages like childhood) are, in themselves, never pathological conditions (but of course they may be *pathologically altered*).
2. *Diseases* are generally distinguished from healthy, normal conditions insofar as they meet at least one *disease criterion* (or *criterion of pathologicity*).
3. There are five primary criteria of pathologicity:
   (a) Shortening of lifetime expectancy, or immediate lethality
   (b) Pain, and other specific somatic or vegetative complaints
   (c) Infertility, i.e., inability of biological reproduction
   (d) Inability or impairment of living together in human symbiotic communities
   (e) Non-universal *disposition* of the organism to develop a condition that is pathological according to one or more of these criteria (this clause covers also conditions that are usually called risk/risk factor, disability, impairment, or handicap).
4. Diseases originate from circumscribed, first or primal causes (or complexes of first causes) that interact from the outside (i.e., from the environment or the parental generation) with the life process of the individual.
5. All partial processes of a disease are *causally* connected. Particularly and retrospectively, they would have occurred even without or against the individual's conscious, intentional volition.
6. In medical theory, the entirety of possible pathological processes forms a huge but definite, manageable system of pathological conditions and causal pathomechanisms that represents the subject matter of General and Special Pathology (including Pathophysiology and Pathobiochemistry).
7. In medical theory, the entirety of diseases may be systematized and classified according to their causal structure and clinical features, and is classified into species (kinds) of diseases that are called *disease entities* and arranged taxonomically by Nosology.

These seven principles represent core theoretical ideas of modern medicine. Around these core ideas the entire existing medical theory of diseases has developed in history and can be reconstructed by philosophy[1].

In the present context of theoretical pathology several remarks may deserve particular attention:

1. Criteria of pathologicity do not express valuations or normative assessments, but form criteria for the use of *discriminating* and *classifying* conditions of life and living organisms. Particularly, this applies to conditions of pain and bodily or vegetative complaints and discomfort: Indeed, these kinds of sensation are experienced as negative, unpleasant or even unbearable and insufferable events, but their "negativity" is not the result of a free, intentionally eligible evaluation, or of a socio-culturally established norm or convention, but is *determined by nature* (viz. the nature of the human organism). It has developed and been selected in the course of natural evolution and phylogenesis of the species *homo sapiens*. This fact is not contradicted by a different fact: that pain and discomfort of this kind are, *additionally*, in most—but, interestingly, not in all—cases of occurrence *evaluated* negative by the affected

[1]The account of theoretical pathology in this section is a *very* condensed one. You will find far more elaborated versions in Hucklenbroich (2013, 2014).

individual, and are subsumed by the socio-cultural environment under the *norm* of being in need of treatment. These evaluations and normative assessments form an additional overdetermination of the natural, physiologically determined sensations, and they may, in distinction to the natural sensation, be denied (e.g., in asceticism) or even inverted (e.g., in self-punishment or self-mutilation).

2. The above principle 2 postulates a strict dependence of pathologicity on criteria 1-5. But this principle is overwritten by a theoretically more elaborate account of pathology and nosology: There are single instances of disease entities that do not meet any primary criterion of pathologicity. Nevertheless, they are classified as diseases, because they meet the *defining criteria of the respective disease entity*. These defining criteria may be, e.g., definite changes in cell structure, or in composition of blood, that can be ascertained by pure lab findings. In such cases, theoretical classification overrides and overwrites phenomenological classification. This procedure that is common in medicine shows that, in principle, identification of diseases and of pathologicity has "emancipated" from the pure phenomenological level and the level of pure clinical symptoms, and will give priority to the theoretical system in ambiguous cases.

Recent philosophical debates on the concept of disease have been paralyzed by the controversy between naturalism and normativism. Naturalism attributes this concept to differences given in nature, while normativism ascribes these differences to individual or socio-cultural valuations and preferences[2]. The reconstruction of medical theory of disease sketched above shows this dichotomy and antagonism to be misguided: In the first instance, the conditions and circumstances addressed in the criteria of pathologicity are naturally given possibilities resulting from the structure and organization of human organism and its embedment into its natural environment:

- mortality and lethal vulnerability are determined by the very structure of living organisms
- negative sensations like pain, nausea, tussive irritation, pruritus (itching) etc. belong to protective mechanisms that are universal features of human organisms
- ability of biological reproduction is naturally (biologically) represented in the male and female reproductive systems consisting of specialized organs, hormones, functions, and mental affections
- living together in (human) symbiotic communities is prerequisite for survival of every human individual, from birth and maternal care to life-long cooperation and mutual assistance.

The last feature, of living together, may be realized in very different, culturally determined and historically changing ways. This openness to variance shapes deeply the form of symbiosis but does not suspend the universal necessity of symbiosis at all. Thus, criteria of pathologicity possess a foundation in nature and are not constituted by valuations or social norms, but by the natural, bio-psycho-social lifeform of homo sapiens. However, defending this position does not imply that one is bound to deny, ignore, or underestimate the cultural and historical embedding and variability concerning forms of thinking and social life dealing with phenomena of disease. On the contrary: Cultural values and traditions as well as individual convictions and preferences are decisive regarding the way disease phenomena are interpreted, evaluated, and regulated by norms and institutions. Help and assistance for ill persons, emergence of the professional role of physician, and the development of public health systems are intentional and socio-cultural *reactions* to the natural phenomenon of disease. In this sense and in the final phenomenon, disease—also, mental disease—is *simultaneously* determined by natural and by socio-cultural factors. One-sided naturalism as well as one-sided normativism both are misleading.

The article of Henrik Walter provides an impressive account of the development and actual status of biological psychiatry. Only the status of the concept of disease remains somewhat ambiguous. First, it is stated that the concept *dysfunctional* "inevitably involves normative judgments" (2). Later on, the concept of normativity is connected to the concepts of suffering and of clinical relevance, and its readability from "biological facts" or "biological measures" is denied (6). Finally, Walter defines *disorder*, following Graham, by being *harmful* and *undesirable for the subject* but adds the remark that these "normative criteria" are not dependent on the subject's appreciation (7)! Who, then, is the subject of these norms and valuations (appreciations), and where do their objectivity or legitimacy stem from? If one accepts the above solution regarding the alleged dichotomy of naturalism and normativism, this apparent aporia may be dissolved.

Marco Stier reports and discusses in his article a lot of arguments from current philosophical literature concerning the status of mental diseases. In my opinion, many of these arguments are in need of a critical examination. But I want to confine myself to an examination of Marco's first and main thesis: That diagnosis of mental disorder is dependent on the acceptance of socio-cultural norms and values. Slightly reformulated, his proof runs as follows:

1. Mental disorders are defined by the presence of *deviant behavior* (experience, emotion) and/or of *suffering*.
2. Deviance can only be recognized by comparing with (mental) *normality*.
3. Mental normality is defined by social and cultural norms.
4. The same holds true for the concepts of suffering and harm: What is recognized to be suffering or harm depends on socio-cultural norms and values.
5. Therefore, the diagnosis of mental disorder is dependent on the prevalence or acceptance of certain socio-cultural norms, and is varying relative to socio-cultural differences and changes.

Premise 1 is equivalent to the assumption that behavioral deviance and suffering are the decisive criteria of pathologicity in psychiatry. I am going to show that this premise is misguided in two respects, and leads to an inadequate view of biological psychiatry.

[2]This controversy, meantime, lasts about several decades. For an overview and review see, e.g., Gottschalk-Mazouz (2008).

1. As soon as psychiatry succeeds in clarifying the etiopathogenesis of a mental disorder or disease regarding also its biological aspects, diagnostics of this disease no longer depends on behavioral or mental criteria: Once *pathognomonic somatic* markers or criteria of a disease are recognized, diagnosis of this disease may be secured or excluded by purely biological tests and procedures. To say more: If the disease in question is a disposition—a dispositional disease like "social phobia" or "tendency to panic attacks" –, then it is not even necessary that the patient at hand has shown the respective symptoms (behavior) at all, because diagnosis may be ascertained beforehand, e.g., by lab findings. The same shift from symptom-related diagnostics to biologically objectifiable methods is usual in somatic medicine, and is in accordance with the aforementioned principle that theoretical classification overwrites and overrides phenomenological criteria.
2. *Behavioral deviance* and *suffering* (or *harm*) are not genuine criteria of pathologicity. Already in the pre-scientific sense, there is no necessary or cogent connection between deviance and pathologicity. Instead, there may be many different causes and reasons of deviance, most of them without any relationship to disease. Additionally, deviance *may be*, but by no means is bound to be a *result* of some diseases, mental or somatic. Also, suffering and harm, as rather abstract categories, are not criteria of pathologicity, because these concepts are far too broad und undifferentiated. Rather, there are several definite, specific kinds of suffering that represent psychiatric criteria of pathologicity. To mention just a few examples:

   - the overwhelming, flooding kind of fear and angst that is typical for panic attacks
   - the "feeling of unfeelingness" in major depression
   - hallucinations, anhedonia (inability to experience pleasure), feelings of self-alienation and depersonalization, and catatonia in schizophrenics.

Symptoms of these kinds represent very specific forms of experience that, indeed, might be described as kinds of suffering, but are criteria of diseases and diagnosis only by their very special characteristics, not because they are cases of abstract suffering or harm. What is most important in the present context: These specific forms of experience and behavior are characterized by a very stable *cultural invariance* of their appearance and presentation; they do not vary relating to even very different cultural contexts. This is a well-ascertained insight of psychiatry, and it diametrally contradicts the thesis of Marco Stier.

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On the autonomy of the concept of disease in psychiatry

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Does the reference to a mental realm in using the notion of mental disorder lead to a dilemma that consists in either implying a Cartesian account of the mind-body relation or in the need to give up a notion of *mental* disorder in its own right? Many psychiatrists seem to believe that denying substance dualism requires a purely neurophysiological stance for explaining mental disorder. However, this conviction is based on a limited awareness of the philosophical debate on the mind-body problem. This article discusses the reasonableness of the concept of mental disorder in relation to reductionist and eliminativist strategies in the philosophy of mind. It is concluded that we need a psychological level of explanation that cannot be reduced to neurophysiological findings in order to make sense of mental disorder.

Keywords: concept of mental illness, mind-body problem, identity theory, reductionism, eliminativism, Szasz, Kendell, biological psychiatry

For some time, especially in the 60s and 70s of the twentieth century, psychiatry was under pressure because it did not seem capable of showing that mental illness actually exists. Can there really be such a thing as a "disease of the mind"? At the time skeptics such as Thomas Szasz (1974) wrote against the "myth of mental illness." The emphasis of this debate lay for a long time in the scrutiny of the associated norms, i.e., the question whether one can differentiate between sick or healthy mental phenomena. One still visible effect of this debate, which has more or less reached its conclusion, is the avoidance of the term "disease" in the official nomenclature of psychiatric medicine. Nowadays we speak of *disorders*, not diseases, for example in the Diagnostic and Statistic Manual of Mental Disorders (DSM), or in the International Statistical Classification of Diseases and Related Health Problems (ICD), in which the psychiatric classification is also described as "mental and behavioral disorders." One cannot help feeling that the attempt had been to avoid a definition that is already all too close to somatic medicine, which would have required a corresponding "hard" validation of the respective categories, or at least a set of explanations of the nosological units in these classification systems. Because contemporary psychiatry barely meets this idea, one could get out of this whole affair with a "weaker" term[1].

The downside of this difficulty is visible in the likewise widespread somatization of psychopathology, which—put briefly—is the identification of mental disorders as diseases of the brain, a practice that has a long tradition in the history of psychiatry. All efforts are undertaken to establish psychopathological phenomena as "real," precisely because many psychiatrists share the view that psychiatry has to be scientifically demonstrable and ought to put in place generally testable criteria for pathological conditions, and because it is somewhat embarrassing for them to separate "disorder" from the disease concept in such a way. Because the real is in turn considered to be one and the same as the observable, from this point on the material disorder of the brain counted as what ideally should be proven. The move had some initial plausibility, in that one more aspect of the original skepticism toward the concept of disease in psychiatry could be undermined. This doubt was fed by the supposedly non-existing location of the disturbance: the spirit or the psyche. How can one rationally assume scientifically valid criteria of pathology when the impaired or damaged object apparently does not exist, or at the very least cannot be affected by disorders? The answer lay in tossing away significant reference to the psyche, the mind, or to mental objects, and talking mainly about neurophysiology, the nervous system, and the brain; for instance when schizophrenia was regarded as a disorder of dopamine and serotonin levels. The somatization could therefore solve both problems of psychiatry: its ostensibly poor scientific grounding and the supposed lack of reality of its phenomena. One astonishing result of this development lay furthermore in the fact that leading proponents of psychiatry now claim—just as their strongest critics once had—that mental illnesses do not actually exist, because only brain disease exists. From a philosophical perspective such a conclusion seems unreasonable[2].

The proponents of the concept of disease in psychiatry, however, seem to be left indeed in an uncomfortable dilemma: If they emphasize the bodily manifestation of mental diseases, then they save the analogy to somatic disease and therefore the medical terminology. At the same time they stand to lose through this strategy the uniqueness of mental disease, which is reduced to somatic disease. If one wants on the other hand to keep the distinctive manner of speaking of *mental* disease (or disorder), then this is apparently only possible when one at the same time postulates a sphere of the mind that is distinct from the body. This strategy, again, seems to lead to a mind-body-duality in the mode that has become unpopular in philosophy. The supposed

[1]In this paper I use "disease," "illness" and "disorder" synonymously.

[2]In this contribution I draw on considerations that were published first in German in my book Schramme (2000).

dilemma for proponents of the concept of mental disease therefore consists in the choice between the Scylla of reduction and the Charybdis of dualism. "Psychiatry is left with two seeming alternatives: either to say that personal, psychological, and emotional disorders are really states of the body, objective features of brain-tissue, the organism-under-stress, the genes or what have you; or else to deny that such disorders are illnesses at all" (Sedgwick, 1973).

As already indicated, there are authors, especially on the side of psychiatry, who are ready to solve this dilemma by consistent somatization and consequentially abandoning the concept of mental disease. One prominent example for this is Robert Kendell (1993, 3), a well-known British psychiatrist: "[. . .] it follows that there is, strictly speaking, no such thing as disease of the mind or mental disorder and that Griesinger was right—mental illnesses are diseases of the brain, or at least involve disordered brain function—because all mental events are accompanied by and dependent on events in the brain. (Thomas Szasz was also right; mental illness is a myth, though not for the reasons he believed.)." A surprising alliance between biological psychiatrists and skeptics has been formed; in the end both positions are ready to give up the concept of mental illness.

The task of this article is therefore to search for compelling arguments in favor of a distinctive concept of mental illness. As already indicated, I cast doubt on the presented dilemmatic structure as a defense of this concept. Rather, I shall try to show that there are well-reasoned positions somewhere between the two horns of the dilemma. The question—can we see "*mental* illness" as an autonomous concept without relying upon unpleasant theoretical premises?—is, as I have said, based on an important philosophical issue: the mind-body problem. This problem can be formulated as follows: Are there really mental phenomena, and if so, how can they be explained and how are they connected with physical phenomena? The first part of this question may sound rather strange, since what are we more sure of than that mental states such as pain, wishes and beliefs exist?[3] In the course of this investigation it will become clearer—so I hope at least—why questioning the existence of mental phenomena is not as strange as it seems. The second part of the mind-body problem, the question of the nature of mental phenomena and their relation to physical states, has of late, with the study of the brain and the nervous system, received a strong empirical orientation and even a new twist. The idea that the nervous system and especially the brain are the basis of mental states is nowadays no longer seriously disputed. For this reason the mind-body problem is sometimes reformulated as the "mind-brain problem." The assumption that mental phenomena are based upon physiological processes suggested itself after the observation of people with brain injuries. Since the introduction of new imaging techniques to study the brain's physiological processes, this theory has been strengthened and refined. Today we can identify connections between regions in the brain and specific mental capabilities, even if definitive relationships between them are not yet possible. And ultimately we are still far away from being able to formulate a generally accepted solution to the mind-body problem.

In this article I will first briefly focus on Thomas Szasz, the main critic of the concept of mental illness. Here I will examine especially his arguments that are related to the mind-body problem[4]. Second, I will apply reductionist and eliminative theories in the philosophy of mind in order to find out whether they might rule out the concept of mental illness.

THE SCEPTICAL ARGUMENTS OF THOMAS SZASZ

Like no other theorist, Szasz has dealt with the concept of disease in psychiatry in a very intensive way, and has attempted to demonstrate particularly the different disanologies between the concept of disease in somatic medicine and psychiatry. The concept of disease—as Szasz has put it in the argument that interests us—cannot be applied to mental phenomena, because the expressions "body" and "mind" belong to different logical categories. When we speak of mental *illness*, we are merely using the term as a metaphor.

In this argument Szasz relies upon the British philosopher Gilbert Ryle (1949), who, in his book *The Concept of Mind*, attacked the "official doctrine" of Cartesianism, in which every human being possesses both a body and a mind, which exist as independent entities. Ryle also called this position the "dogma of the ghost in the machine." He wanted to refute this position by showing that it is based on a so-called category mistake. Szasz uses Ryle's approach in order to demonstrate that the "official doctrine" of psychiatry commits an identical category mistake, in virtue of assuming that there is both bodily illness and mental illness. The mistake consists in the claim that the mind can, just like the body, be affected by illness.

Ryle explains his conception of a category mistake by way of several examples. Suppose that a student comes to Oxford for the first time. There she is given a tour of the many colleges, libraries, and administrative offices. After the tour she says: "I now know the individual colleges and where the books are kept, and I have also seen where the administrators work. But now I want to be shown the university." Naturally this wish cannot be fulfilled, because colleges, libraries, etc. *are* the university. There is no other point of interest that is called "university." Apparently the student does not know that "university" belongs to a category separate from "New College," "Bodleian Library," etc. It would be a similar mistake to ask, after one had been shown the functions of the

[3]This assumption in reference to the existence of the mental on the basis of its experiential aspects has also once again gained some weight recently in philosophy of psychiatry. To this end new phenomenological approaches, such as those from Fuchs (2004), Gallagher and Vaever (2004), or Ratcliffe (2008), have taken up the experiential attributes of mental disorders. From this perspective we could claim that the challenge of the mental by somatization is itself drawing upon a dualistic starting-point. In virtue of doubting distinct properties of the mental realm, somatization accordingly even results in a rejection of one aspect of human existence, i.e., the mental, which, however, cannot be separated from human corporeality. The contrasting holistic perspective becomes prominent in the concept of Leib (lived body), which accordingly plays an important role in the phenomenological tradition.

[4]A more extensive critical evaluation of Szasz's position, and of other critics of the concept of mental illness, can be found in Schramme (2004).

defender, the forward, and the goalkeeper etc., whose function it was to contribute the team spirit in a football team.

The mistake of Cartesianism is given, according to Ryle, in such a mixing of categories, in that "mind" is classified in the same logical category as "body." Only in this way could it be claimed that a human being has both a mind and a body. But one could only combine expressions in linguistically correct conjunctions when they belong to the same category. Therefore, one cannot, from a linguistic point of view, state, for example that one has seen New College, Bodleian Library, *and* Oxford University.

Ryle does not claim that the mind cannot be said to exist as a matter of principle. But if this claim is made, one would need another meaning of "exist" than in the assertion that bodies exist. Because mind and body belong to different categories, it cannot be reasonably claimed that mind *and* body exist in the same way. Nor can it be expressed in a logically compelling way that *either* the mind *or* the body exists, because this disjunction is just as inadmissible when expressing different categories. To say, "Either I have visited the New College, the Bodleian Library etc. or seen the university" is patently absurd.

At this point Szasz (1974) wants to show that *because* "body" and "mind" belong to different logical categories, *in principle* there can be no such thing as a mental illness. This theory and its basis—the category mistake argument—are important for Szasz's work and are mentioned again and again, even when there is no direct relation to Ryle, such as when Szasz says that an illness can only affect the body.

As previously mentioned, Ryle's category mistake argument is directed at the "official doctrine" that had prevailed after Descartes and that postulated two distinct entities: the body and the mind. The special feature of Ryle's argument as compared to other critiques of dualism is his claim that asking about the relationship between mind and body is already by itself non-sensical (Ryle, 1949, 23). But even if this view is correct, the category mistake argument alone does not support Szasz's theory, because the mere suggestion that "mind" and "body" belong to different categories does not allow the conclusion that a mental illness cannot exist. The argument does not express whether mental phenomena exist or not, but rather casts doubt on Cartesianism.

Moreover, illness does not exist independent from organisms. If one abandons the notion of an independent existence for illness, the idea of actual mental illness is no longer implausible, because we need not claim that the mind *has* an illness, but rather, as one could say for example, a disorder of mental capacities—however specified—*is* a mental illness. In order to make this claim one would not have to postulate a separation between mind and body. Substance dualism is not required for the maintenance of the concept of mental illness. Therefore, Szasz cannot show the absurdity of this concept in principle through casting doubt on the Cartesian separation of mind and body.

THE MIND-BODY PROBLEM

There are many different theories about the relationship between mental and physiological states. These theories range from strict separation of the two spheres to their identification, or lead all the way to the claim that the mind-body problem is principally unsolvable. This situation and the enormous scale that the respective literature has since reached make it necessary to restrict this examination to a cursory treatment and to keep as strictly as possible to the issue at stake. For this reason I will largely restrict myself to negative statements in order to defend the concept of mental illness, and will attempt to show accordingly that the theories that doubt the explanatory independence of mental phenomena are not adequately justified to reject such an autonomy [5]. Positive criteria of such evaluation of theories would be their scientific adequacy—this spoke against Cartesian dualism—but also their philosophical plausibility, of course. Still, I cannot deal with all of the many contributions in this area of research.

Theories of the relation between mind and body that lead to a questioning of the mental disease concept are largely of two kinds: Either they are *reductive*—they lead to an explanation of mental phenomena through reference to physiological states; or else they are *eliminative*—they relegate mental phenomena to the sphere of myth that is, dispute the very existence of mental states. I naturally do not want to claim that the respective authors actually want to discard the notion of mental illness. Ultimately their theories are aimed at a different question. But it is possible to scrutinize the independence of the mental disease concept on the basis of such theories and in doing so to ultimately scrutinize the identity of psychology and psychiatry. And I think furthermore that implicit or explicit theories about the connection between mind and body can have an impact on psychiatry's research focuses, its methods of treatment, and its classifications.

From this point on I will take it for granted that the problems of substance dualism, such as those exemplified in Descartes, are already well-known. In the following I will first analyse the reductive theories on the mind-body problem. Then I will examine the eliminative theories. The result will show that the rejection of an independent concept of mental illness does not succeed. A conceptualization of mental disease by referring to mental states is possible without having to advocate an awkward dualism. Nevertheless, we should not go so far as to generally repudiate somatic approaches in psychiatry, but rather emphasize their one-sidedness and their need to be complemented. Psychiatry should be neither "mindless" nor "brainless".[6]

IDENTITY-THEORY

The mind-body identity theory makes reference to mental states, which underlie our actions, and therefore it does not reduce them

[5]Unfortunately there is still very little cooperation between philosophy and psychiatry regarding this specific issue as well as the underlying mind-body problem. The psychiatric literature is relatively unaffected by the elaborate debates in philosophy. In turn, a theory in philosophy will still seldom be examined against concrete examples, such as those found in psychiatry. It would be important to achieve stronger cooperation here, and on the whole I see this article to be an attempt at mediation. There are in the meantime some interesting publications that are headed in this direction. To highlight just a few: Wilkes (1988); Graham and Stephens (1994); Griffiths (1994); Northoff (1997); Ghaemi (2003); Radden (2004); Murphy (2006); Bolton (2008); Kendler and Parnas (2008); Graham and Stephens (2010), and Graham and Stephens (1994) founded periodical Philosophy, Psychiatry & Psychology; see also Schramme and Thome (2004).

[6]Eisenberg and Lipowski coined the terms "mindless" and "brainless" psychiatry (compare Sullivan (1990), p. 271).

to observable behavior, as behaviorism had done previously, ultimately leading to a theoretical dead end. Identity theory permits explanations of behavior that appeal to desires, beliefs, pain, etc., and seems in this regard adequate to work as a basis for an explication of the mental illness concept. However, the theory posits the ontological identity of mental states and neurophysiological states. In this way identity theory rules out substance-dualistic assumptions. Mental states are identical to physiological states of the nervous system and the brain. There are not two different substances, only one, namely matter. The mental does not have any properties that go beyond the physiological[7]. One often cited example for this is the identity of pain with the stimulation of C-fibers.

Identity theory is superior to behaviorism not only thanks to its appeal to inner states, but also through its ability to acknowledge different mental phenomena in cases of identical observable behaviors. For identical behaviors can have many different underlying mental states, which are themselves identical to specific neurophysiological states. Therefore, for instance the same behavioral abnormality could in one case be accompanied by a neurophysiological irregularity that is completely missing in another case. On the basis of such a theory this suggests searching for the boundaries between mental normality and illness on a neurophysiological level, because on this level different mental states manifest themselves. The first step to a theory about mental illness as a brain illness is therefore fulfilled. Identity theory also solves Descartes's problem of explaining mental causation, for instance the causation of the action of going to the fridge by a desire to drink and a belief that there is a drink in the fridge. On the basis of substance dualism such a straightforward explanation becomes quite difficult to achieve, because mental phenomena are regarded as non-material in Cartesianism, and non-material things lack the force to cause anything physical. However, if mental states are identical to neurophysiological states, then there is no need to postulate an obscure non-mental causality. Mental states can cause material changes to the body in virtue of their material existence.

We should clarify what the identity theorists mean by "identity." J. J. C. Smart (1959, 171), one of the prominent proponents of identity theory, speaks of "strict identity" and uses the identity of lightning and electric discharge as explanatory example. The thesis is stronger than a mere claim to a correlation between brain states and mental phenomena. This type of identity is often explained using Leibniz's law of the identity of indiscernibles: X is strictly identical to Y if they are indiscernible from one another, i.e., if every property of X is also a property of Y, and vice versa. In the present case of an identity claim, this means that there are no properties of the mental that are not also properties of the nervous system. Hence there is no unique property of the mental.

The proposition of identity theorists is not tantamount to the claim that statements about mental and neurophysiological states have the same meaning, and are therefore synonymous. The identity theory merely claims that the state being referred to is one and the same. We can elucidate this distinction with the following example: The Evening Star is identical with the Morning Star (both have the same properties of the planet Venus, and so refer to the same object), although the terms have different meanings, because there are "different modes of presentation" at hand (Frege, 1892).

Smart (1959) emphasizes the difference between synonymity and identity in order to avoid one obvious objection to identity theory. For there's room to claim that mental and physical states are not identical, because someone who has no idea of neurophysiology can nonetheless refer to his mental condition. If the two states were identical, so this argument goes, then we could substitute the proposition in a statement such as "I know that my foot hurts" with the proposition "my brain is in state X" while retaining the truth value. Since this clearly does not work, the states cannot be identical. This only shows, however, that statements about mental states have a different meaning than statements about physical states, not that they are not ontologically identical. Therefore, this kind of argumentation against identity theory is invalid.

In the distinction between meaning and reference there is also the implication that the postulated identity of mental and physical statements is informative (i.e., not *a priori*). In its differentiation between types of identity statements such as "a bachelor is an unmarried man," "a square is an equilateral rectangle," and the like, identity theory contains an assertion of a contingent, empirically verifiable fact. Mental states could also be identical with completely different states, but it has been established by science that they are identical with neurophysiological states.

Still, the critique of identity theory was not hard to come by. At this point two variations of objections can be distinguished[8]. Firstly, the neurophysiological side of the identity proposition has been the starting point for objections. Here the argument of "multiple realizability" is especially pertinent. In the case that indistinguishable mental states could each be realized through different brain states, they would not, contrary to the proposition of identity theory, be identical to specific neurophysiological states after all. Secondly, it is questionable whether mental states in their usual taxonomy can be rendered at all as identical with any underlying brain states. The terminology, with which we categories mental states as desires, hopes, etc., has been around long before anyone ever had any insight into brain processes, and it seems therefore unlikely that clear equivalents can be found.

The identity theorists postulate a strict identity of mental states and neurophysiological states. The proposition is that indistinguishable mental phenomena each correspond to the same physiological states. But, as the objection goes, it is not only conceivable, but even extremely likely that many animals experience states of consciousness such as for example pain. Yet the nervous system and brains of many animals vary widely from those of

[7]It is meant thereby that every mental state can in principle be fully explained exclusively in reference to neurophysiological processes. Compare for example Smart (1959) p. 54.

[8]Here I ignore a whole line of objections, which deal with so-called qualia that is, the specific, felt qualities of a mental state. These objections are not limited to the identity theory introduced here, but rather are in part turned in general against physicalism, disputing therefore that mental states possess exclusively physical attributes. The best known versions of these objections come from Nagel (1974) and Jackson (1982, 1986).

humans. Therefore, it is completely unlikely to ever be able to identify specific neurophysiological states with mental states. A bird's feeling of pain, or that of a perhaps unknown creature, could be based on radically different physiological states. This is one possible way of formulating the argument of "multiple realizability."

The objection itself was first advanced by Hilary Putman (1967). For strong forms of identity theory it has proven to be fatal, but not necessarily for weaker ones. Putnam supports identity theory with a strong proposition. He claims that the theory has to show, for example that a feeling of pain can always be identified with a specific neurophysiological state. If, however, different physical states can accompany the same mental state (feelings of pain), then this proposition falls apart. "Consider what the brain-state theorist has to do to make good his claims. He has to specify a physical-chemical state such that *any* organism (not just a mammal) is in pain if and only if (a) it possesses a brain of a suitable physical-chemical structure; and (b) its brain is in that physical-chemical state. This means that the physical-chemical state in question must be a possible state of a mammalian brain, a reptilian brain, a mollusc's brain (octopuses are mollusca, and certainly feel pain), etc. At the same time, it must *not* be a possible (physically possible) state of the brain of any physically possible creature that cannot feel pain" (Putman, 1967, 53).

The philosophers that support identity theory have only inadequately explained what they understand as mental states. It would be, for example, conceivable not to simply identify unspecified feelings of pain with one neurophysiological state, but rather "sharp" pain, "dull" pain, etc. It would be equally possible, naturally, to realize the classification of species-specific mental states. Then human pain could be identified with the neurophysiological state X, octopus pain with mollusc state Y, etc. Such species-oriented reduction seems to weaken Putman's argument. But even if we restrict ourselves to examples of mental phenomena in humans, it remains unlikely to be able to carry out identification at a higher level of mental states. Consider, for example, a desire to travel to London. This desire realizes itself clearly within an entire web of other mental states: for instance the belief that London lies in England; the belief that London is beautiful; the knowledge that one has an important appointment there. Even given all that we know about the brain, it appears hopeless ever to be able to match such a state one-for-one with a neurophysiological state. The brain (and also the respective mental state) is simply too complex to be able to ascribe such identities[9].

There is still the alternative of weakening statements of identity by making the classification of mental states more finely granulated. Hence it could be the case, for example that we can identify a specific neurophysiological state with the mental state "sensing an orange in a veiled and darkened room." Yet here it would be unclear what advantage would come of this kind of reduction. The more that identity theory specifies the types of mental states that they want to identify, the more they lose its original advantage, namely its simplicity. But it does not necessarily follow from the lack of plausibility of such type-identification that mental phenomena are not, on particular levels, after all identical with physical events. This is the proposition of the so-called token-identity. This theory attributes the identity of every single mental event (token), for example the belief of person X at time *t* that it will rain today, to a single neurophysiological event. This is admittedly an extremely weak identity thesis, but it guarantees the maintenance of a non-dualistic position. Still, it apparently prevents any *reasonable* reduction of the explanation of mental states to physiological states, and is therefore inadequate as an argument against the autonomy of the concept of mental illness.

There is a yet another way to understand type-identity: If every realization of a type of mental states could be subsumed to a class of neurophysiological states, then the argument of multiple realization would be defeated. Suppose the belief that Berlin is the capital of Germany would be realized in the respective neurophysiological states N1, N2, N3, etc. Due to this assumption multiple realizability would be safeguarded, because the belief would not always be identical to one and the same neurophysiological state. If, however, N1, N2, N3, etc. could be subsumed to a class, then a kind of type-identity would be saved (Rosenthal, 1994, 351; Hannan, 1994, 21f.).

Maybe we could identify particular mental illnesses with distinct classes of neurophysiological states, in the sense that all realizations of a mental illness would fall under a neurophysiological class. The following is an example: Suppose that schizophrenia in person X were realized in neurophysiological state N(X), in person Y in state N(Y), etc. Now it appears that N(X), N(Y), etc. all belong to a distinct class S. Then it would evidently be possible to reduce the explanation for schizophrenia to the class S. Whether we can show that I cannot say, but the chances seem (theoretically) not so bad [10]. On the other hand the question remains whether through S we have really explained and reduced the mental phenomena-type schizophrenia completely. This is arguably not the case. One important reason to me seems to be the following: To subsume such mental phenomena as "schizophrenia" assumes that they have in common a particular property, namely that they are cases of disorders of "normal" *mental* states. Even if it were indeed the case that S underlies all these states, the mere reference to S does not explain the pathology of the schizophrenic realizations (see also Margolis, 1991). We only show that there is a *correlation* between the mental states and a type of neurophysiological states. The claim that all instances of S have the property of realizing a *disordered* neurophysiological process is only possible on the psychological level of explanation. The fact that specific

[9]Beckermann (1996, 6) points out that after injuries to the brain many mental processes of other parts of the brain, which previously were not involved, can be realized. To an extent this would be an intrapersonal variation of the argument of multiple realization. In my opinion the neuroplasticity of the brain is a general argument against the possibility of simple identification, which, however, I will ignore.

[10]Practically there are certainly significant problems, for in order to show that the single neurophysiological processes actually fall under a certain type one has to acquire a detailed knowledge of them. However, "The more precisely one wants to establish neural state N as the origin of a behavior, the greater does one change the brain and the overall situation of the test subject . . . " (Tetens, 1992, p. 121). Tetens describes this as a "fuzziness relation" of neurobiology, and argues "Already for this reason naturalistic descriptions of the human being are not practically realizable alternatives to the mental-psychological descriptions."

mental phenomena count as a mental illness cannot therefore be explained exclusively through brain physiology. In this respect I take the assertion that mental illnesses are brain *illnesses* (or diseases, for that matter) to be truncated. The assertion conceals that being ill is explained and only recognizable on the psychological level. A psychological explanation of mental illness is in this regard autonomous in relation to physiological attempts at explanation.

Even if all counterarguments up to now are not sufficient for a complete repudiation of identity theory, can the theory ever show itself to be true? The second of the objections to be presented here focuses on the mental side of the identity thesis in replying to the question introduced, i.e., the question about the possibility of reducing the mental to the physical level[11]. Mental states are normally grouped into so-called propositional attitudes (for example desires, beliefs, hopes, which all have propositional content) and sensations (pain, sensual perception, etc.). We use this classification in order to make actions comprehensible. If we see someone running in the train station, then we understand this behavior in that we ascribe to the person certain beliefs—for example that the train is about to leave—and desires—that he wants to board the train before it leaves. We can even make our own actions comprehensible with these categories, such as in the following example: "I took my hand away because I suddenly felt a sharp pain." At the same time there are certain principles that guide our explanations, such as, for instance, if a person would carry out action A when she has the chance, because she has the desire X and believes that A will achieve X. Together these categories and principles amount to an apparently useful basis for the explanation and prediction of behavior. This is not an especially complicated psychological theory that requires its own field of study, but rather an accumulation of concepts and rules, which are regularly used on an everyday basis. This tool has been given the short-hand name "folk-psychology"[12]. Already for hundreds of years—and long before anyone ever knew about brain processes—this has been used by people, at least in a similar form, in order to interpret their behaviors. Yet identity theory claims that there is a one-to-one correspondence between neurophysiological processes—any knowledge about which we have only ever gathered in the last few decades—and the taxonomy of folk-psychology. It seems more than unlikely, however, that categories that originated long before our knowledge of the brain and nervous system can be precisely identified with corresponding neurophysiological types of processes. It would be just about as likely as a correspondence between the disease taxonomy of Paracelsus and the newest insights into physiological processes in the human body.

ELIMINATIVE MATERIALISM

The results of this examination up to now suggest a certain skepticism toward reductive theories in philosophy of the mind. To be sure, mental states are not on the one hand non-physiological states, since they are apparently realized through physiological processes in the brain. On the other hand the world of the mental is not entirely physiologically explicable. "The desire to drink a beer causes him to go into the pub." "The detective followed the murderer because he believed he wanted to hide the weapon of the crime." It is questionable how such complex situations could be described meaningfully on a purely physical level. In this regard we have levels of explanation that are independent and irreducible. Folk-psychology works on this level. But are its explanations correct? Proponents of Eliminative Materialism (EM) answer this question in the negative[13]. Going further, they actually come to the radical conclusion that these states, which we and other creatures are ascribed to within the framework of folk-psychology, do not exist. There are no desires, beliefs, fears, etc. This thesis contains the eliminative side of EM. Propositional attitudes (and even states of experience like feelings of pain, here depending on what EM exactly sees as elements of folk-psychology)[14] are removed of their ontological legitimation, whereby they are relegated to the sphere of myth.

This is a truly radical proposition, and I will try to illustrate why it is not as unintelligible as it may at first seem. Certainly it should be clear that—if EM is right—it would have equally radical consequences for our conception of mental illness. If the sphere of the mental, in the way that we describe and explain it every day, is non-existent in the strictest sense, then this would really revolutionize its conceptualization as well as the categorization of single types of illnesses. Whether the mental disease concept in its own independent framework would also be lost depends on which level of explanation is chosen for the corresponding phenomena. If there were a (future) scientific psychology that would then replace the eliminated folk psychology, then talk of *mental* disease would—in my opinion—still be warranted[15]. However, because the main supporters of EM want to see folk psychology eliminated to the benefit of a (future) *neuro*physiology, it is questionable whether there could still be *mental* illnesses in the strictest sense[16].

[11] It can be found for example in Churchland (1988 p. 27 f.) This objection is, as it will later become clear, not merely advanced against the plausibility of identity theory, but also serves as an argument for eliminative materialism.

[12] Sometimes common-sense-psychology is also being used, because the expression "folk psychology" has a pejorative connotation.

[13] The term "eliminative materialism" stems from Cornman (1968). It was already used earlier in the sixties by Feyerabend (1970) and Rorty (1965) in a similar form. The main supporters of a "modern" EM are Patricia and Paul Churchland.

[14] Sometimes only propositional attitudes are attributed to folk psychology. Hannan (1994, p. 45) points out that supporters of EM are often less critical of qualitative states. But Churchland (1994, (308) explicitly specifies pain for example as a component of folk psychology).

[15] Naturally I do not want to claim that the future psychology will manage without any reference to physiological and chemical processes in the brain. Already today the borders are fluid. The independence of the concept of mental illness would be kept alive in the maintenance of a genuine psychological terminology in this science. An attempt to discuss the theory of EM from a psychiatric point of view is undertaken by Harrison (1991).

[16] In this regard EM as such is not a threat to the mental illness concept (even if for today's existing classification of mental illnesses), but rather only in a certain version. Some arguments for why a future science of mental phenomena should not be exclusively located on a neural level can be found in Kitcher (1996). To be sure, it is not always clear from the Churchlands' writings whether they expect the elimination of psychology in general or of only folk psychology.

The discussion concerning EM's persuasiveness is therefore linked primarily to the status of folk psychology. Ironically the latter's irreducibility becomes a symptom of its superfluity. Because mental states are based on physical states, a neurophysiological explanation should support the folk-psychological explanation. That was also the claim of identity theory. Now it has been shown, however, that explanations on the level of folk psychology cannot be reduced to a neurophysiological level. And precisely because this irreducibility relation exists, it can be shown that folk psychology and neurophysiology are incommensurable. Temperature could be successfully reduced to molecular kinetic energy, and therefore can continue to be seen as existing. Conversely, no one could find a scientific explanation for witches that were commensurable with superstition, and so witches count as non-existing. The more we learn about the functionality of the brain the clearer it becomes that there are practically no equivocations to be made from the folk-psychologically explained mental states to neurophysiological levels. But when two approaches of explanation are incompatible, then it is to be taken that at least one is false, in which case it should be discarded. The claim of EM is naturally that folk psychology is false and therefore has to disappear. To this degree mental states go the way of witches: They are expelled from the scientific ontology.

In short, EM consists of two premises and one conclusion (cf. Stich, 1996, 4). Premise 1: Folk psychology is a theory. Premise 2: Folk psychology is false. Conclusion: The mental states postulated by folk psychology do not exist and can play no role in any future explanation of behavior.

Which objections are addressed toward EM?[17] Its first premise pertains to the form of folk psychology: Does it really represent a theory? This claim is essential for EM's proposition, for otherwise it would be questionable why we should drop folk psychology. The assumption of EM is that folk psychology aims to provide explanations and predictions of behavior, and therefore contains certain claims and supports certain principles, etc. If this were not the case, then it would not compete with a neurological explanation of the same issue, and would therefore not be an eligible candidate for elimination. It seems to me, on the basis of the already discussed need for a psychological perspective to establish distinct mental disorders that we cannot abandon the corresponding vocabulary and its related theoretical constructs.

The second premise of EM states that folk psychology as a theory is false, because it leaves many phenomena unexplained and is therefore incompatible with the underlying natural sciences: "(. . .) what we must say is that FP [folk psychology, TS] suffers explanatory failures on an epic scale that it has been stagnant for at least twenty-five centuries, and that its categories appear (so far) to be incommensurable with or orthogonal to the categories of the background physical science whose long term claim to explain human behavior seems undeniable. Any theory that meets this description must be allowed a serious candidate for outright elimination" (Churchland, 1981, 212).

Churchland sees shortcomings to the explanations of folk psychology in the areas of, e.g., creativity, intelligence, sleep, memory, sensory illusions, and—especially interesting for our analysis—in relation to mental illness. This suggestion of Churchland's is not easy to counter. It seems true that we can learn fairly little about mental illness through the help of folk-psychological conceptions and principles. But does folk psychology even have this aim? A theory can only be untrue in that area where it claims to have explanatory value. Otherwise it does not at all seek to compete with other theories such as neurophysiology or a "scientific" psychology.

Churchland's claim that folk psychology breaks down or fails as a theory apparently stems from the assumption that it does *compete* with neurophysiology. Then it would be an appropriate candidate for elimination. But this assumption is not straightforward. First the role and aim of folk psychology would have to be clarified. There is much to indicate that even when it is not all that successful as a theory, neither is it so false that it has to be eliminated, nor does it have to be replaced by a (future) neurophysiology. Even if it is replaced, it would be done so by a scientific theory that is situated on the same level of explanation—that is, a psychological one. Folk psychology works on a macro level, and neurophysiology on a micro level. In this regard the irreducibility of folk psychology does not necessarily lead to its incommensurability with neurophysiology. Each explains phenomena, but simply on two different levels of abstraction.

Psychiatric explanations of disease phenomena would not manage without psychological conceptions. Which role folk psychology will keep in the process is still undecided; but to eliminate it is neither necessary on theoretical grounds nor advisable on practical grounds, for it is through its coarse grain that in the end the psychological perspective facilitates our access to the mental world.

CONCLUSION

The reductive and eliminative theories of the mind-body problem are not convincing. The reductive versions largely fail for the lack of correlation between types of mental and neurophysiological states. Identity theory seems to apply on the level of single mental events. Thereby, however, only an ontologically non-dualistic position is implied, and not at all the superfluity of mental terminology. On the contrary, mental illness is one of the very phenomena that would be unexplainable on an exclusively neurophysiological level, because here no explanation of a single event (token) is being sought, and because the ascription of pathology itself can only happen by taking the mental level into account. The eliminative position fails largely due to its ascription to unreasonably high expectations on the part of folk psychology. Sure it is unlikely that we can adequately explain mental illness with folk-psychological terminology alone. But this finding neither makes folk psychology as such superfluous

[17] One objection to EM that I do not find convincing, but will mention for the sake of thoroughness, is that it is self-defeating: The theory of EM contains the premise that beliefs, etc. do not exist. It is objected that one can only sensible formulate this premise, however, with recourse to the affected states themselves, for example beliefs. To me this objection seems to be begging the question, because it assumes the same manner of speaking that is put in question. An expression of the theses of EM may very well radically change, once folk psychology is eliminated. Hannan (1994, p. 62 ff.) puts more faith in this objection. For a defense against this charge see Churchland (1986, p. 397 f.); Churchland (1988, p. 48).

nor excludes the possibility of a scientific and therefore *psychological* (and specifically psychiatric) explanation of mental illness.

The concept of mental illness was drawn into question on the basis of reductive and eliminative theories. As a consequence the rejection of these accounts leads to the possibility of an independent conceptualization of mental illness—so long as no further, more convincing objections are brought forward. To be sure, the difficulties of substance dualism prevent any possible explanation of mental illness to be completely independent of physiological knowledge. Mental and bodily phenomena do not belong to *principally* separate areas. In this regard both the general rejection and the one-sided restriction to brain-physiological explanations of mental illness fail. In brief, even if we have accepted that theories of mental illness are not allowed to be in conflict with the knowledge of neurophysiology, it does not follow that the single (let alone the best) explanation of mental illness can exist on a neurophysiological level.

Even if for many disorders of the mental apparatus no corresponding disorders of brain-physiological processes were found, the language of mental illness does not seem to me to have necessarily failed. The desire for "objective" signs of disease, as they are supposed to be in the brain or in observable behaviors, is understandable. Nevertheless, this is in the end a methodological problem that is posed for a future scientific study of mental illness. It should not lead to discounting any of the two—neurophysiological or mental—perspectives. Mental illness is not reducible to brain illness, even when mental phenomena have their basis in the brain.

The recent publication of the fifth version of the *Diagnostic and Statistical Manual of Mental Disorders* should cause occasion for the underlying philosophical aspects of the language of mental disorder to make itself clear within the psychiatric trade. The chairpersons of the working committee that put together the DSM-IV had still formulated the following misgivings: "There could arguably not be a worse term than *mental disorders* to describe the conditions classified in DSM-IV. *Mental* implies a mind-body dichotomy that is becoming increasingly outmoded (. . .)" (Frances, 1994, VIII). We should have expected that such mistaken misgivings—which are after all concerning the very foundations of psychiatry—would be resolved by now. A cursory look at current psychiatric publications, however, gives us cause to fear that the same error in reasoning will be made as before: "'Mental' implies a Cartesian view of the mind-body problem that minds and brains are separable and entirely distinct realms, an approach that is inconsistent with modern philosophical and neuroscientific views" (Stein et al., 2010, 1760) ([18]). In this respect we surely have to be skeptical that psychiatric thinking will have seriously progressed. Instead the cure will very likely be looked for in neurobiology. In this article I have attempted to show why psychiatry will not find here what it is looking for and that there is no need to look for a supposed cure, since the concept of mental illness is autonomous from somatic medicine. For philosophers this is no surprising realization; for many psychiatrists, however, the insight seems to remain closed off.

[18]This article was mentioned on the DSM-5 website as background for the planned revision of the definition of "mental disorder": http://www.dsm5.org/proposedrevision/Pages/proposedrevision.aspx?rid=465# [Accessed 14. September 2012]. The reference has since been deleted from the website.

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Mental disorders are somatic disorders, a comment on M. Stier and T. Schramme

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In his paper, Marco Stier argues that mental disorders are irreducible to the brain and cannot be determined in a purely physical manner (Stier, 2013). Instead, he argues that mental disorders can only be determined on the mental level since behavior can only be termed deviant by comparing it to the norms of non-deviant behavior. Thomas Schramme proposes that a psychological conceptualization of mental disorders is necessary since the concept of mental illness is autonomous from somatic medicine and that psychiatry will not find the cure it is looking for through neurobiology (Schramme, 2013).

In the following, I present my personal argument for: (i) why neurobiological research offers the potential for identifying curative therapies for mental disorders despite the lack of a valid explanatory model on the mental level (albeit not to the extent psychiatrists may wish); and (ii) that such research is needed to establish more valid disease models on the mental level. In other words, an understanding of the biological underpinnings of the mental disorders is a prerequisite for a valid disease model. If the definition of deviant behavior requires norms, these norms, and the degree of deviance, will not only depend on the observed behavior *per se* but also on our understanding of its biological underpinnings and its variance. To illustrate this point I will cite two examples. Firstly, phenotypically identical hallucinations are conceptualized differently depending on whether they are the consequence of a high fever or the symptoms of schizophrenia, and whether effective treatment is readily available or not. Secondly, the inability to spell correctly due to dyslexia is judged differently to poor spelling that is attributable to a lack of care or effort (and dyslexia has for the majority of the past 2000 years of our history almost totally escaped diagnosis). Thus, a concept which is not supported by knowledge of the etiological underpinnings can only be partial and, as Hanfried Helmchen points out, potentially dangerous for patients (Helmchen, 2013).

Furthermore, I argue that the concept of "mental disorder" should be abandoned or at least not confined to the so called mental/psychiatric disorders, as in my opinion mental disorders are somatic disorders, and the so-called somatic disorders also associated with varying numbers of psychiatric symptoms.

As a psychiatric geneticist whose research aim is to identify genes involved in the development of psychiatric disorders, I believe that all of the observed clinical symptoms have a biological correlate. The extent to which different societies in different time periods will conceptualize these symptoms as "pathological" will depend on the society in question. Factors such as the severity of the individual's suffering (e.g., paranoia, anxiety) or the degree of severity attributed to given symptoms by society (e.g., obesity, gambling, sexual deviations) will play a role. Nevertheless the biological underpinnings of deviations from the so-called "normal" will, in many cases, be identifiable. Although I personally consider it unlikely that science will ever provide an exclusively neurophysiological explanation of mental disorders, I am convinced that diagnosis will eventually be based upon assessment of the physiology of the individual patient.

At present however, no such biology-guided diagnoses exist. Although research has established that mental disorders are complex and that their development involves interactions between genetic and environmental factors, their etiology remains largely unknown. Current psychiatric classification systems, such as DSM and ICD, define psychiatric disorders as distinct disease entities. According to these diagnostic systems, a diagnosis should be assigned when a given number of symptoms have been present over a specified period of time. Despite high diagnostic reliability between psychiatrists and evidence from family studies that relatives of index patients have an increased risk of being assigned the same diagnosis, the clinical presentation of psychiatric disorders differs widely between patients, and diverse courses and outcomes are observed within diagnostic categories. Furthermore, no single clinical symptom is either pathognomonic of, or necessary for, a given psychiatric diagnosis, and considerable symptom-overlap is observed between diagnostic categories.

As, in contrast to somatic disorders, no objective laboratory measures are yet available to refine psychiatric diagnosis, the establishment of a diagnostic system that is biologically based will require a more comprehensive knowledge of the etiology and pathophysiology of psychiatric disorders and/or their presenting symptoms.

Since Griesinger, biological psychiatry has conceptualized psychiatric illnesses as disorders of the brain. However, other brain disorders such as migraine, epilepsy, and neoplasms are generally treated by neurologists rather than psychiatrists. In cases where a causal biological reason for psychotic or depressive symptoms has been identified, the disorder ceases

to be a mental disease in the strict sense of the term. This is exemplified by endocrine conditions (e.g., porphyria, hyper-, and hypothyroidism); metabolic conditions (e.g., hypoglycaemia); hepatic, renal, or autoimmune conditions; and viral infections. Indeed a DSM criterion for assigning a diagnosis of schizophrenia is that "*The disturbance is not attributable to the physiological effects of a substance or another medical condition.*" Similarly, a DSM diagnosis of psychotic disorder requires that "*no specific and direct causative physiological mechanisms associated with a medical condition can be demonstrated.*"

Recent findings in psychiatric genetics may provide insights into how mental disorder should be conceptualized. For many years, whole genome screening and the process of relating millions of genetic variants with a complex disorder while taking into account environmental factors and personal life-experiences were considered impossible. However, these processes are now available to researchers.

In recent decades, extensive efforts have been made to identify susceptibility factors for psychiatric disorders such as schizophrenia, bipolar disorder, autism, attention deficit hyperactivity disorder, major depression, and alcohol dependence. The results of formal genetic investigations, e.g., family, twin, and adoption studies, have provided unequivocal evidence that environmental factors as well as inherited genetic variation play a substantial role in the etiology of these disorders. Heritability estimates suggest that genetic factors account for 75–80% of the variability observed in susceptibility to schizophrenia, bipolar disorder, autism and attention deficit hyperactivity disorder and 35–50% of that for alcohol addiction and major depression. For other common complex disorders, such as diabetes, breast cancer, and Crohn's disease, heritability ranges between 55 and 70% (Sullivan et al., 2012). Thus, the contribution of genetic factors to schizophrenia, bipolar disorder, and autism is relatively high. Schizophrenia is also one of the complex common disorders that account for the majority of genome-wide significant findings identified in genome-wide association studies (GWAS) since their introduction in less than a decade ago.

Recent calculations indicate that around 6000–10,000 common variant are involved in the etiology of schizophrenia, and that these variants are not confined to genes expressed in the central nervous system (Ripke et al., 2013). Rather, these variants are located across all chromosomes. It is therefore very likely that the influence of these variants is not restricted to the brain. Thus, a given variant could act as a risk factor for both, a mental as well as a somatic disorder. Many somatic and mental disorders display co-morbidity, and the question therefore arises as to whether these conditions should be viewed as two separate diseases, or whether such states represent a single disease with somatic as well as mental manifestations. Formal genetic studies of co-morbidity between cardiovascular disease and depressive disorders, e.g., suggest the latter. Furthermore, initial molecular genetic studies suggest that stress, for example, is a risk factor for both depression and cardiovascular disease: the influence of a major risk gene (FTO) for obesity is particularly pronounced in depressed persons, and genetic variation in the" stress gene" NPY modifies weight gain under conditions of stress.

Interestingly, among the most significant findings for schizophrenia are variants located in the Major Histocompatibility Complex Region, a locus which hosts, among others, genes responsible for immune reactions. This evidence underlines the finding of formal genetic and candidate gene studies that genetic as well as environmental factors contribute to these disorders.

I argue that all mental disorders are somatic disorders, and that what we currently term mental disorders are actually somatic disorders for which the somatic component is too weak to be detected. That is, due to the high sensitivity of the human brain, and the extreme level of functioning demanded of it in modern life, even harmless somatic changes may have a detrimental influence on brain function.

An example may serve to illustrate this point. Genetic and biochemical studies indicate that immunological processes play an important role in depression, or at least in a subset of them. Viral infections such as a common cold in turn also involve immunological reactions and can present with all of the symptoms required to assign a diagnosis of MD (e.g., markedly diminished interest or pleasure in almost all activities, fatigue, diminished ability to concentrate, loss of appetite, hypersomnia). If such an infection escapes diagnosis it is possible that the patients will receive the diagnosis of depression. I personally have seen patients who had been assigned a diagnosis of depression and who actually had suffered from unrecognized infections such as borreliose and hepatitis. New infectious agents are identified each year, and it is possible that a proportion of patients who are diagnosed with depression today are being misdiagnosed since the causal agent has not yet been identified. But the question is whether this really should be considered a simple misdiagnosis.

While I would argue that all mental disorders are in fact somatic disorders (including the brain as an organ), this implies neither that the cause must originate in the soma, nor that conclusions may be drawn concerning the optimal mode of treatment.

Numerous studies have shown that factors, such as poverty, stressful life events, and child abuse, are major risk factors for depression (although the effect of a given environmental factor can differ substantially depending on the genetic make-up of the individual). Furthermore, the environment can have long lasting effects in terms of which genes will be expressed. This in turn will influence how that particular individual will respond to a future environmental stimulus. A decade ago, research in rats demonstrated for the first time that post-natal maternal care could influence stress hormone receptors, and that this predisposed the affected animal to more pronounced reactions to stress in later life (Szyf et al., 2005). These findings were subsequently replicated in humans. Furthermore, a human genome-wide methylation study by our group revealed that prenatal maternal stress impacted on the methylation pattern in the newborn. These results were then replicated in studies of monkeys and rats. Thus, *environmental* factors can impact on mental well-being through the soma, and can therefore predispose to further reactions

to environmental factors through *somatic* signatures. Further knowledge is needed to define whether the optimal therapy for the resulting depression should be delivered on a direct somatic level or through psychotherapy.

To conclude: I argue firstly, that all mental disorders are somatic disorders, and that somatic disorders present with varying proportions of somatic and/or mental symptoms; and secondly, that both the identification of the underlying genetic and environmental factors as well as optimal causal therapies is important and feasible. This neurobiological approach is promising even in the absence of a valid disease model on the mental level and will in turn inform such a model. Independent of current disease models, it is important for therapists to remember that patients suffer from their symptoms *per se* rather than from the underlying causes of these symptoms. Thus, the management of patients who display mental symptoms—with or without somatic symptoms—requires both an understanding of the nature and subjective impact of these symptoms and appropriate therapeutic empathy.

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On the relation of psychiatric disorder and neural defect

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The debate about the relation between psychiatric disorder and neural defect has produced different argumentative strategies for and against the identification of these two phenomena. I'll coin these strategies as (a) an ontological strategy, (b) an extensional strategy, and (c) an intensional strategy, on which I will focus in this article.

The first, ontological strategy takes the long road over a detailed characterization of the nature of psychiatric disorders and of neural defects. It then goes on to argue for a relation between these two ontological kinds. One anti-reductionist proposition of an ontological strategy can be found in Stier (2013). He provides a sketch of a theory of the nature of psychiatric diseases, claiming *inter alia* that psychiatric diseases are social constructions and intrinsically normative. He goes on to infer a non-reducibility thesis from these more or less ontological characterizations: "if the boundary between normality and mental disorder is a social construction such that the question of whether a certain kind of behavior is a disorder can only be judged against the background of this very convention, then the "disorderness" of a condition cannot be found on—and hence not be reduced to—the neuronal level" (Stier, 2013, p. 3). While Stier's argument fluctuates between an epistemic and ontological non-reducibility thesis, I take him to be talking about the nature of psychiatric diseases and thus about an ontological issue foremost.

The second, extensional strategy investigates the phenomena that fall into both categories in order to relate the categories. In principle one would have to identify all psychiatric diseases and their pathways in the brain. If one could identify at least one neural causal pathway for each psychiatric disease, all psychiatric diseases could be assumed to be brain diseases. Alternatively one could use a falsificatory strategy and look for one psychiatric disease, which does not have a neural causal pathway. Obviously both are only in principle and not practically viable options. Universally quantified statements and claims of non-existence are wellnigh impossible to prove. Typically, the extensional method is applied in exemplary research projects. Proponents of the identity or reducibility of psychiatric disease to neural phenomena try to show that a certain psychiatric disease can be explained with reference to neural phenomena. Opponents of reducibility try to identify psychiatric diseases for which an explanation via some neural, causal pathway is improbable. The extensional strategy has to combat severe methodological challenges as discussed by Kapur et al. (2012) cf. the discussion in Walter (2013): the power of neuro-psychiatric studies, the limited replication of studies, the reliance on extreme comparisons and of course, as Walter (2013) mentions, the ethical issues of research on the living brain.

The third, intensional strategy takes a slightly shorter road to elucidating the relation between psychiatric disorders and neural defects by discussing their explanatory roles: looking at the way the categories are defined and applied.

Turning to intensional methods requires some restriction in the use of the categories and concepts in question. Thus, instead of talking loosely about mental, psychiatric or psychological diseases or illnesses and physical, neural or brain based diseases or states the focus will be on psychiatric disorders and neural defects. The term "psychiatric disorder" will be used for two reasons: (1) "disorder" is the term used in the ICD 10 and in DSM IV and V; (2) I do not want to talk about the alleged mental/physical divide, but about the categorizations of the relevant scientific disciplines, in this case psychiatry; thus I do not use "mental disorder." The term "neural defect" will be used for two similar reasons: (1) "defect" seems to be the weakest functional term (more on functional terms in a moment). (2) I use the word "neural" instead of "neuroscientific" because I want to refer to the broader category of defects detected by neurosciences as a whole, not just in single cell analyses (neuronal) or brain anatomy.

The contrast between the intensional strategy and the ontological and extensional strategies can be observed in Schramme (2013). While he does provide some details on the ontological positions in the debate of the mind-body problem in a quite extensive part of his article, his persuasive key arguments pertain to the explanatory roles of mental phenomena, especially psychiatric disorders, and require only a brief part of the text. Schrammes two convincing arguments for an irreducibility of psychiatric disorders to neural defects are (1) even membership in a neurological category will not explain, why the mental states realized by certain neural states are pathological. The ascription of pathology or of being disordered is dependent on the psychological level of explanation. (2) In an aside he observes that neurophysiological explanation does not even seek explanations of single event tokens.

According to Schramme's first argument, the identification of a neurological class of states all of which realize some psychiatric disorder would not suffice to explain the psychiatric disorder in question (Schramme, 2013, 5 f.). This argument can be further supported by some details on how the concepts in question are embedded in their explanatory projects.

"Psychiatric disorder" and "neural defect" are concepts from quite different disciplinary contexts, which slowly coalesce: "psychiatric disorder" is first and foremost a concept of a "-iatric" discipline, namely psychiatry. "Neural defect" is mainly a concept of neuroanatomy and -physiology. Both are functional concepts that serve a disciplinary purpose and have been shaped in order to do so. A concept is a functional concept by virtue of its embeddedness in functional explanation.

The functional explanatory strategy consists in decomposing a specific explanandum into a set of distinct parts and trying to show how the parts and their forces account for the original phenomenon: "[. . .] the analytical strategy proceeds by analyzing a disposition *d* of *a* into a number of other dispositions $d_1 \ldots d_n$ had by *a* or components of *a* such that programmed manifestations of the d_i results in or amounts to a manifestation of *d*" (Cummins, 1975, p. 759).

The process of decomposition will be iterated during a functional explanation of complex systems, especially in explaining the behavior of an organism. The crucial question at each onset of decomposition is what to pick out as the phenomenon to explain. This decision recurs in every iteration of the decomposition procedure. In a decomposition of arm movement one has to decide whether to analyze the behavior of the muscles or that of the tendons or bones. On the next deeper level one chooses whether to analyze the behavior of cells or that of extracellular transport systems etc. Thus, just as the specific explanandum depends on the background theory, the explanatory path in functional explanation depends on the decompositional decisions.

Two distinct sub-types of this method give rise to the concepts of a psychiatric disorder and of a neural defect. Their differences are threefold:

(1) Context of detection. Psychiatric disorders strike the observer as something to be explained and treated: Usually psychiatric disorders are abnormalities of behavioral patterns observed in terms of folk and scientific psychology. Either they themselves are the reason for an analysis of the behavioral pattern or the pattern has been the target of prior interest in psychology or cognitive science.
Neural defects are further removed from casual attention: Neural defects are abnormalities in the working of causal pathways, in the parts and forces making up a phenomenon. They can only be found after an analysis. The abnormalities in these pathways can but need not result in abnormalities in behavior.

(2) Context of action. The analysis of psychiatric disorders is driven by the desire to understand a behavioral abnormality and if possible to find a therapy or workaround. Neural defects in contrast are found in analysis driven by a purely explanatory research interest.

(3) Last but not least, the context of explanation. Neural defects turn up when physiological phenomena are analyzed into physiological parts and their tempo-spatial relations. A neural defect primarily is defined within a mechanistic explanation of some neural phenomenon (Bechtel, 2009; Craver, 2013). Mechanistic explanation typically elucidates relatively complex behaviors of biological systems by the actions and interactions of their constituting subsystems. The actions and interactions of the subsystems in turn are explained by actions and interactions of their respective subsystems; insofar mechanistic explanation is a type of functional explanation as mentioned above (Craver, 2013). Mechanistic explanation is a special type of functional explanation however, because it strictly sticks to componential analysis, that is: the subsystems stand in a physical part-whole relation to the system, which gets explained. The main interest in ascribing a function and noticing a defect is explanatory. Something is a function because it contributes to some complex behavior in most homologs, which a scientist aims to explain. The word "function" could be replaced by "normal causal role." Something is a defect, because in the more numerous homologs the causal pathways work differently. The word "defect" in this context could be replaced by "abnormality."

A similar type of analysis can be found in cognitive science, with the not so minor variation that what gets explained are cognitive abilities and behavior, and they typically get analyzed into cognitive and affective sub-tasks and capacities. There are for example theories of long term memory, distinguishing it into subtasks of encoding and consolidation, storage and retrieval as well as into different subtypes like episodic, semantic, procedural, and priming memory. The explaining subsystem and the system, which get explained do not stand in a physical part-whole-relation. The decomposition is cybernetic and not componential.

While neural abnormalities can be considered defective only relative to explanatory interests, it seems to be possible to identify cognitive or affective defects beyond such an interest (contra Stier, 2013). If a person can't grasp objects in her right visual field, or can't remember words for more than a few moments, that seems to be a defect no matter what. This alleged obviousness of there being a defect stems from the close interdependence of cognitive science and psychiatry as regards their phenomena as well as their methodology. The psychiatric diagnosis of a defect and thus the psychiatric ascription of function and dysfunction is often prior to analysis in cognitive science. The discussion how to define functions in psychiatry is still on-going and vast [for an overview cf. Schramme (2010)]. None of the suggestions, however, takes recourse to analyzing complex behaviors into physical parts and forces as is done in mechanistic explanation. The explanantia of both methods, psychological and psychiatric, do not stand in a physical part-whole relation to the system and behavior explained.

To conclude: Neural defect and psychiatric disorder are defined within different types of analysis. One is componential, decomposing complex phenomena based on purely explanatory interests, ascribing function on the basis of comparison to homologs. The other is non-componential, decomposing complex phenomena based on interventionist interests, ascribing functions on the basis of systemic goals. The categories are thus neither identical nor bear an obvious intensional relation. As Schramme concludes, there is

no reason for "discounting any of the two—neurophysiological or mental—perspectives. Mental illness is not reducible to brain illness, even when mental phenomena have their basis in the brain" (Schramme, 2013).

It is highly implausible that two categories based on different methods, research interests etc. are homomorphic, that is, can be related in a one to one style. As Schramme (2013, p. 6) points out as well, it would be more than surprising if for every taxonomic class of folk psychology, or, as I must add, of psychiatry there were one related type of neural defect or the other way around. As results in cognitive neuroscience, neuropsychiatry and related disciplines already show, the relation between neural defect and psychiatric disorder is much more complicated: different neural defects can result in the same disorder, sometimes a psychiatric disorder is caused by several coincidental neural defects etc. Thus, even if the metaphysical thesis, that all mental states are token identical to physical states is true, and I take it to be true, that does not help one bit in explaining or treating any of them.

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Mental realities—the concept of mental disorder and the mind-body problem

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A commentary on

On the autonomy of the concept of disease in psychiatry
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From a philosophical point of view, the subject of Thomas Schramme's article seems to be well-known as he addresses one of the most prominent debates in both classical and contemporary philosophy: the mind-body problem. Of course, Schramme does not attempt to broadly cover this general issue. He rather focuses on the neglect of philosophical approaches within the context of the search for a sound definition of mental disorders that led to conceptual as well as theoretical problems for psychology and psychiatry. As a result of this neglect, Schramme argues, psychiatry is facing a make-believe dilemma that implies either Cartesian dualism or reductionism/eliminativism when trying to save the notion of "mental disorder." In a nutshell, this apparent dilemma for current psychiatry goes as follows: Either we try to save the notion of mental disorder by claiming an independent sphere of the mental and end up with the implausibility of substance dualism. Or we attempt to avoid this problem by means of consistent somatization and a naturalistic reduction of mental terms and phenomena, thereby in fact disposing of any substantial meaning of *mental* disorder. Hence, psychiatry seems to be stuck "between the Scylla of reduction and the Charybdis of dualism" (Schramme, 2013, p. 2).

As Schramme convincingly shows, the prevailing acceptance of this alleged dilemma in psychiatry is due to some fundamental misconceptions and the "limited awareness of the philosophical debate on the mind-body problem" (Schramme, 2013, p. 1). He demonstrates this claim by discussing two prominent positions in the philosophy of mind which—albeit in quite different ways—eliminate the level of psychological explanation and at the same time any significant meaning of mental disorder: identity theory and eliminative materialism. While his discussion necessarily remains cursory, it covers the most important objections against both theories. For philosophers, the most surprising aspect in Schramme's analysis of this rather well-known controversy consists of the fact that psychiatry has so far to a large extent ignored important conceptual differentiations that could help to avoid false conclusions like the idea that the concept of mental disorder compellingly implies "a Cartesian view of the mind-body problem, that minds and brains are separable and entirely distinct realms, an approach that is inconsistent with modern philosophical and neuroscientific views" (Stein et al., 2010, p. 1760). A closer look at those "modern philosophical views" would have shown that there is no necessary connection between "mental" and substance dualism but rather different (e.g., phenomenological and narrative) approaches that try to define and describe a rich concept of mental illness without falling back to Cartesianism (for an overview see Perring, 2010).

Schramme succeeds in demonstrating the general problems of reductive and eliminative theories and shows that both types of theories do not provide compelling reasons for rejecting "the possibility of an independent conceptualization of mental illness" (Schramme, 2013, p. 3). Only to a lesser extent, however, does he address the specific features of mental illness that determine its conceptual autonomy and immunize it against scientific naturalism and reductive explanation. As an extension to Schramme's line of argument, I will therefore briefly discuss the case of *posttraumatic stress disorder* (PTSD).

PTSD has become well-known in the context of war veterans who were—passively or actively—involved in extreme forms of physical or psychological violence. After having returned from mission, sometimes years or even decades later, some of them start to re-experience certain episodes, for instance the unintended killing of civilians. These episodes appear as very lively, uncontrollable autobiographical memories that emotionally affect the patient and inevitably arrest his attention. By forcing the patient to relive the traumatic experience again and again, such memories create "black holes" (Pitman and Orr, 1990, p. 469) in the narrative reality of the person, unintentionally attracting his attention without being able to successfully integrate the remembered event into his life story. While being an inerasable part of the *historic reality* of the person, it cannot at the same time be accepted as truly belonging to oneself and therefore cannot be integrated into the persons' *narrative reality* (Jungert, 2013, p. 202). Thus, there remains a *foreign body* in the life story of the person that constantly causes flashbacks and induces the persons' suffering from his past (Hampe, 2007, p. 92).

Why is PTSD a good example for the irreducibility of psychological explanation that Schramme seeks to defend? Most notably, because it reveals the fundamental problems that result from

any attempt to reduce the internal perspective of human minds to the external perspective on human brains. As described above, traumatic disorder can be understood as a break into a persons' history caused by the traumatic event. The awareness of this break is tied to the categories of meaning and subjective reality—the idea of something being true *for* someone—which again can only be captured if one assumes some kind of mental reality and the existence of an internal perspective. It is *by reconstructing these internal perspectives* that psychiatry is able to get access to traumatic disorders and—at least in some cases—to find a way of dealing with them conjointly with the patient.

As a matter of principle, even the most sophisticated neuroscience or biological psychiatry would not be able to approach mental disorders like PTSD appropriately, because the recognition or analysis of internal perspectives is not part of their methodological repertoire, nor can it be grasped by its basic concepts. Instead, by trying to describe psychological phenomena exclusively by using somatic terms and categories, they in fact eliminate those perspectives, because "nothing is true for somatic structures on their own, i.e., they cannot be treated as something with an internal perspective" (Hampe, 2007, p. 100). Saving the sphere of internal perspective, however, does not necessarily imply to invoke substance dualism. In consonance with Schramme's reasoning, it is enough to consider the mental and the somatic dimension as phenomenologically different, but complementary aspects of one substance.

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Commentary to the articles of M. Stier (Normative preconditions for the assessment of mental disorder) and T. Schramme (On the autonomy of the concept of disease in psychiatry)

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A commentary on

Normative preconditions for the assessment of mental disorder
by Stier, M. (2013). Front. Psychol. 4:611. doi: 10.3389/fpsyg.2013.00611

On the autonomy of the concept of disease in psychiatry
by Schramme, T. (2013). Front. Psychol. 4:457. doi: 10.3389/fpsyg.2013.00457

Both authors agree that:

> "*Mental illness is not reducible to brain illness, even when mental phenomena have their basis in the brain*" (Schramme, 2013, p. 8) and
>
> " *…because of the normative nature of psychiatry, mental disorder cannot be completely reduced to neuronal or molecular processes. […] A mental state as such may well be reducible to the brain, but determining whether this very state is (part of) a disorder or not, is nothing the brain sciences can do.*" (Stier, 2013, p. 8).

Therewith the authors deny the tacit assumption according to which disease in its proper sense can only be somatical but never psychic. This posit, which was propagated under the common term of "*Somatosepostulat*" by the German psychiatrist Kurt Schneider (Schneider, 1950) who dominated the post-war psychiatry. Strangely enough the content of Schneider's posit was later on ascribed to a falsely abridged citation of Griesinger according to which mental diseases were nothing but brain diseases (Griesinger, 1861). In fact he stressed in addition that mental states ("*Seelenzustände*") may not be reduced to functional disturbances of the brain ("*Funktionszuständen des Gehirns*").

Definitely no one who is familiar with the second edition of Griesinger's textbook would call this distinguished author to witness psychiatry as a special kind of neurology. Moreover, if a mental disease is nothing but a neurological or a somatic disease respectively, it was logical mandatory to speak of "*Somatic Psychiatry.*" Therefore it is disconcerting that this term is absolutely unfamiliar. Instead, Schneider's posit goes under the disguise of the semantically inappropriate term of "*Biological Psychiatry.*" Following this rational way of thought, the unanswered question arises what actually could be intended by the title of the reviewed symposium "*Biologism within Psychiatry?*" (*Biologismus in der Psychiatrie?)* whereas—strictly speaking—one ought to use the unusual or even inexistent term of "Somatologism."

By the way, to the present author whose duty consisted only to comment on a freely chosen article (out of six) it is cloudy that neither in the workshop nor in any one of the later on prepared articles an answer was searched for the core issue contained in the workshop-title whether psychiatry suffers from biologism (or not). This seemed to be avoided like a hot potato since it would have implied a debate on the outstanding semantics of "biological," biologistical or "somatical."

Schramme states, that the recent publication of DSM V should give occasion for the underlying philosophical aspects of the language of mental disorder to make itself clear within the psychiatric trade. He criticizes DSM IV for using the term "*mental disorders*":

> "*Mental implies a Cartesian view of the mind-body problem that minds and brains are separable and entirely distinct realms, an approach that is inconsistent with modern philosophical and neuroscientific views.*" (Schramme, 2013, p. 8).

One cannot but agree that the progress of psychiatry depends on a logical and semantical consistent terminology. But it is just as disputable that this aim can be reached by simply eliminating the colloquial term "*mental*" being used as a synonym of "*psychic*" and/or an antonym of "*somatic.*" Why should *the concept of mental illness* be *autonomous from somatic medicine*, as Schramme claims?

The real problem to be solved is not an outdated Cartesian view of substance dualism, being scarcely advocated by any of the contemporary psychiatrists, but the prevailing materialistic monism or eliminative reductionism (e.g., Paul and Patricia Churchland, Armstrong, Quine Ryle, Skinner, Crick etc.) which is being camouflaged by the term "biology/biological" being ill-posed because opposite to the sense intended.

There is only one epistemological solution which goes back to Spinoza (1890).

Among the contemporary exponents of this solution the best known is Habermas (Habermas, 2004) who prefers to speaks of "*Epistemic dualism.*" Another notion of this concept which dispels the myth of the unsolvable mind-brain problem is that of "*Aspektdualismus*" (Ulrich, 1990, 1997, 2006a,b, 2013).

About a decade ago a German psychiatric chair holder wrote about his vision of an integrated clinical-neuroscientific field with psychiatry as a special focus but no longer existing as an autonomous field (Maier, 2002). The Psychiatrist was redefined as a Clinical Neuroscientist or Clinical Psychopharmacologist.

Recently the director of a renowned Max-Planck-Institute for Psychiatry confessed in an interview that he surely was a better chemist than psychiatrist (Holsboer, 2005). By an editorial, entitled: "*Are we still in need of psychiatry as a special field within medicine?*" Ulrich (Ulrich, 2006a) demanded that psychiatrists should discourage any attempt to abandon psychiatry as a distinct discipline. He referred to the demand of *Aspect Dualism* being valid for medicine as a whole. Accordingly, "either-or interrogations" have to be replaced by "as-well-as" ones. Thus, it was undue to beg the question whether a hysteria is a brain disease or a psychological disease, or whether a depressive disorder is a biochemical or a psycho-social disorder. Such nominalistic definitions are equally misguided as the question whether an altarpiece should be labelled as an antique or a sacred object. By a recent monograph Ulrich defined Psychiatry both as a Biological Natural Science *and* a Humane Discipline (Ulrich, 2013).

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The depressive situation

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From a naturalistic perspective on mental illness, depression is often described in terms of biological dysfunctions, while a normative perspective emphasizes the lived experience of depression as a harmful condition. The paper relates a conceptual analysis of "depressive situation" to an analysis of the lived experience of depression. As such, it predominantly aims to specify depression as a harmful condition in lights of normative perspective on mental disorder, but partially refers to empirical research, i.e., naturalistic perspective on depression, to exemplarily stress on the methodological merits and limits of relating phenomenological considerations closer to empirical research. The *depressive situation* is further specified with an examination of the evaluative dynamics by which individuals meaningfully relate to themselves, others and the world. These evaluative dynamics emerge out of the interplay of pre-reflective and reflective processes, which are significantly altered in depression. Such alterations of the evaluative structure are inextricably intertwined with significant distortions of practical sense in depression. From a phenomenological perspective, these distortions of practical sense show in characteristic experiences of evaluative incoherence and impairments of agency. Finally, this paper focuses on an examination of "evaluative incapacity," which has the integrative potential to capture a range of typical changes of meaningful relatedness that determine the depressive situation.

Keywords: depression, *existential situation*, experiential synthesis, *practical sense*, habitus, narrativity, evaluative coherence, caring

INTRODUCTION

The paper aims to concretize some phenomena involved in depression, according to which it is conceptualized as harmful condition from a normative perspective. Rather than positioning my analysis of the depressive situation in fundamental opposition to the value-neutral perspective on depression provided by naturalism, it rather affirms the value of both perspectives from a methodological perspective. Although a conceptual analysis and phenomenological considerations of the depressive situation clearly face some methodological limitations in accounting for the empirical research on depression, this approach might indicate an alternative view on depression that can also inform naturalistic accounts. The lived experience of depression is often neglected in the descriptions of depression in terms of biological dysfunctions (cf. Jacobs and Walter, 2011). This can be counterbalanced in relating empirical research on depression closer to phenomenological considerations, for instance, in relating an analysis of particular depressive experience to those theories that aim to provide the neuropsychological correlates to it. Inasmuch as my analysis of changes in the evaluative experience of self and world in depression is exemplarily substantiated by autobiographic narratives, this illustrates, in which ways depressive experience differs in many respects from non-depressive encounters with the world. From a phenomenological perspective on depression, these narratives are authentic expressions of the lived experience of depression and for this reason also provide an additional diagnostic value for clinical diagnosis. The reported phenomena in depression narratives point beyond the well-known clinical cluster of classificatory criteria for depression. The *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision* (*DSM-IV-TR*; American Psychiatric Association, 2000) and *International Classification of Diseases, Tenth Revision* (*ICD-10*; WHO, 2010) criteria of depression often do not reflect the experiential heterogeneity, as well as the subtle differences in the experience of depression. Such phenomena, which are neither captured by the clinical diagnostic manuals, nor become transparent as lived experiences from a naturalistic perspective, are, for instance, significant changes in one's sense of reality, alterations in the experience of one's abilities (changes of practical sense), as well as a range of other experiential alterations in one's sense of meaningful relatedness to the world. Consequently, my analysis aims to enrich the clinical picture of depression drawn by the *DSM* or *ICD* with respect to a more detailed analysis of the underlying evaluative dynamics that shape the depressive situation. In doing so it may contribute to a reconciliation of the scientific and everyday characterization of depressive symptoms, and may show how a normative understanding of depression as *illness* completes a naturalistic conceptualization of depression as *disease*[1].

Before explaining the particular evaluative dynamics that underlie these experiences in depression in greater detail, one can start with some conceptual considerations on the term "existential situation," which addresses those processes in which individuals situate themselves in the world in a particular evaluative way.

[1]For a detailed discussion of the distinction of "illness" and "disease," see Boorse (1975) and Schramme (2003).

But how are individuals situated by the ongoing dynamics of evaluative self- and world-disclosure that emerge out of the general interaction between organism and world? It is assumed that these dynamics are the structural prerequisite placing individuals already in some sort of *meaningful* relation to the world. This bond of meaningful relatedness, this structure, which is instantiated by and maintained in proceeding evaluative experience and practice, points to the inherently evaluative dimension of self- and world-disclosure, thus, to the normative depths of existential situatedness.

With the concept of "depressive situation," I allude to what Karl Jaspers has coined *border situation* (German: *Grenzsituation*), a notion which can be used to describe psychiatric disorders as an exceptional state of existence, *converting* "situations of daily life" (German: *Alltagssituationen*) to border situations, inasmuch as fundamental alterations of evaluative processes of self- and world-disclosure are involved (cf. Jaspers, 1925, 1973; Jacobs and Thome, 2003; Fuchs, 2008). The concept of *evaluative self- and world-disclosure* (German: *Selbst- und Welt-Erschließung*) allows us to refer to those evaluative processes in which things in the world, other people and also (aspects of) oneself and one's actions become intelligible to someone by virtue of being part of a world that provides a background of meaning to one's (practical) encounters (cf. Kompridis, 2006). The main hypothesis of this paper is, that the existential situation of the depressive type – the depressive situation – is characterized by such structural changes in evaluative processes, which contribute to characteristic experiences and modes of enaction that differ significantly from non-depressive ones. These dynamics of self- and world-disclosing significantly alter in depression, i.e., what appears as intelligible to one often dramatically changes in depression: depressed persons often report that they feel disconnected from the world, that it appears as an empty place deprived of all meaning, that other people and activities formerly enjoyed are no longer of interest, that they get stuck in deliberative processes of rumination and indecisiveness, etc. Inasmuch as the sheer variety of experiences symptomatic of depression cannot become fully addressed here, I predominantly focus on conceptual considerations about evaluative self- and world-disclosure. These may prepare the grounds for testing, which particular experiences and clinical symptoms point to an inherently evaluative problematic of the depressive situation.

EXISTENTIAL SITUATION – THE DYNAMICS OF EVALUATIVE SELF- AND WORLD-DISCLOSURE

For a start, it has to be mentioned that in contrast to approaches suggesting *affective* intentionality as the conceptual core of meaningful self-world-relatedness (Stephan and Slaby, 2011), or which focus predominantly on *feelings* as providing this evaluative structure for modes of relatedness (Ratcliffe, 2008), I argue for a *broader notion of intentionality*, which can account for a greater variety of evaluative modes that structure one's existential situation in general, and by which in particular he heterogeneity of changes in depressive evaluation, and not just particular changes of affect, is addressed. It is assumed that processes of evaluative self- and world-disclosure cannot be reduced to or equated with a mere affective (or: "felt") dimension, albeit affectivity (e.g., experiencing an emotional episode) certainly has a self- and world-disclosing function and, as such, provides one way to address a certain mode of evaluative self-world-relation, respectively (cf. Jacobs, 2013a).

To argue for a broader notion of intentionality then also implies to consider the whole *bodily corporal* dimension as irremissibly constitutive of particular modes of meaningful relatedness (cf. e.g., Ratcliffe, 2009; Fuchs et al., 2010). This likewise does not imply to restrict the self- and world-disclosing function of the lived body to a pure feeling dimension in terms of affectivity. Evaluative self- and world-disclosure through the lived body neither depletes in affective states, nor does it solely rest on *felt* evaluations (cf. Helm, 2002). Rather the dynamic interplay of these with different types of incorporated social structures (e.g., values) and particular "knowledge" (practical skills, unconscious desires, embodied memories, etc.) has to be reconsidered as significantly contributing to the respective modes of evaluative relatedness provided by the bodily corporal dimension.

Generally, the body as *Körper* expresses several forms of incorporated practice or roles, e.g., an actor can, for instance, incorporate (*verkörpern*) a certain role. While this kind of incorporation is rather the product of intentional processes (e.g., learning to incorporate a role), there are also specific forms of incorporation that "enter" the body through social structures, education, etc., which rather tacitly shape our situation as embodied individuals. Contrastingly, the *Leib* is the lived body, the transparent medium for world-disclosure that enables us to engage with the world (cf. Jäger, 2004). Pierre Bourdieu's theory of embodied practice paradigmatically outlines how external (social, cultural, moral) structures become incorporated in a literal sense. With his notion of *habitus*, he has elaborated individual (pre-)dispositions in terms of an embodied, embedded and enacted perspective on individual situatedness (cf. Bourdieu, 1979, 1980). This reading of *embodied practice* is the presupposed rationale for my analysis of the evaluative dynamics of psychopathological self- and world-disclosure (cf. Jacobs, 2012, p. 251, 2013a).

Inasmuch as it is rather the complex *interplay of different types of evaluations and evaluative states* – which together contribute to how someone actually experiences a specific situation, and is directed toward the world and others in a meaningful way – this interplay can be further explained by the procedural dynamics of evaluative processes. These considerations provide the prerequisites for the analysis of how these procedural dynamics of evaluative processes change in depression, and how this, in return, contributes to characteristic changes of meaningful relatedness.

PRE-REFLECTIVE AND REFLECTIVE PROCESSES

Generally, the procedural dynamics of evaluative self- and world-disclosure can be specified in pointing to the particular interplay of the pre-reflective and reflective sphere of one's existential situation (cf. Jacobs, 2012, 202ff). The pre-reflective sphere is – broadly construed – that according to which all intentional encounters with the world are pre-structured by a set of individual predispositions (for instance perceptional schemes, embodied skills, tastes, capabilities, etc.). It has its counterpart in a sphere of reflection, while it is particularly by self-reflexive processes[2] that individuals

[2]Normally, self-reflection is understood as a mode of intentionality that improves one's practice in the future through a *retrospective* analysis of action. The difference

structure their life as autonomous agents (cf. Jacobs, 2012, 175ff; pp. 221–229; 242ff).

These modes of self-directedness in acts of reflection, of course, form a special kind of evaluative self-relation, which differs from that provided by the pre-reflective sphere: inasmuch as individuals are able to develop an objective *stance* (German: *Haltung*) toward their own beliefs, desires, feelings, behaviors and actions, it is in virtue of their self-reflexive capacity that they *situate themselves as autonomous agents* in fields of social (e.g., moral, cultural, etc.) practice (cf. Rothacker, 1941, 55ff; Jaspers, 1973, 203). In contrast to theories of practice, in which the self-reflexive stance and associated rational strategies of self-constitution, for instance, in deliberative processes play rather a secondary role (e.g., Bourdieu, 1979), one can emphasize the experiential and practical modes of meaningful relatedness provided by these. This implies that a person can influence by which particular evaluative stance, and by which particular modes of practice, she relates to herself, others and the world. These modes of being directed to the world (and others) then have their vital counterpart in the evaluative modes of self- and world-disclosure provided by the pre-reflective sphere. Both spheres equally contribute to how one's existential situation enfolds in the procedural dynamics of evaluation. Consequently, the depressive situation can be explained by focusing on structural changes of evaluative processes that take place on the pre-reflexive and reflective/self-reflexive level.

THE INTERPLAY OF DIFFERENT TYPES OF EVALUATION

If one aims to account for the *interplay of different types of evaluations* that equally shape particular evaluative experiences and modes of meaningful relatedness, and if one further stresses the complex evaluative phenomenality and intentionality provided by the dynamics of both spheres, one has to differentiate at least those types of evaluations that rather refer to a pre-reflective sphere, from those that predominantly structure the more reflective encounters with the world: the pre-reflective sphere provides one with an experiential structure and particular modes for self-world-relation that can be described as stemming from rather spontaneous and immediate evaluations (e.g., in terms of bodily appraisals). Often this pre-reflective sphere is described as an evaluative dimension, which rather tacitly structures one's daily encounters with the world. This evaluative "rather taken for granted-structure" has been specified, for instance, in terms of basic existential feelings (cf. Ratcliffe, 2008; Jacobs et al., 2013). These background orientations provide one with a basic sense of belonging or a sense of reality, thus, with an evaluative structure that normally is rather the unquestioned evaluative basis for the more reflective processes of self- and world-disclosure. They come to the focus of someone's attention especially when they significantly alter, e.g., due to a slight experiential fracture of one's sense of reality as in a *déjà vu*-experience. Other classes of evaluative types that rather seem to belong to the pre-reflective realm are, for instance, incorporated memories and unconscious desires.

to "self-reflexivity" becomes relevant with regard to when this process of introspection exactly takes place, as self-reflexivity refers to the *ongoing evaluative process*, for instance in deliberation.

What unites all these different types of pre-reflective evaluations is that they have to be reconsidered for their constitutive role for agency, i.e., as (partially) constitutive of self-reflexive evaluative processes. The discrimination of a pre-reflective vs. reflective evaluative sphere of one's existential situation thus is a purely analytical one, as this paper emphasizes the *dynamic interplay of both spheres* that establish the evaluative processes – the evaluative structure – by which the meaningful bonds between self and world emerge. Claiming a primacy of self-reflexive evaluative processes for (e.g., moral) self-constitution as agent does not imply to dismiss that both classes of evaluations contribute to such structurally complex modes of evaluative directedness.

THE SYNTHESIS-VIEW – THE INTEGRATIVE UNITY OF EVALUATIVE EXPERIENCE

It may have become transparent that it is by experiencing a particular type of evaluative state (e.g., a desire) that we are already directed to the world and, as such, imbue the world with meaning. Consequently, the intentional and the phenomenal dimension are no longer separable in evaluative processes, but rather must be conceptualized as essentially *unified in the evaluative experiences of self and world.*

From what has been said so far follows that the particular evaluative modes of existential situatedness can be characterized *neither* solely in terms of mental states (mere intentionalism), as the concept of intentionality has been broadened by considering the whole bodily corporal dimension of individuals as something essential to the respective (evaluative) experience itself. *Nor* are the evaluative modes by which the self-world-relation is instantiated are understood as just being comprised of separable *components*, whose relational connection often raises conceptual problems. From the perspective of such component theories, evaluative states (e.g., emotions) are described as having a certain kind of intentionality (world-to-mind/mind-to-world/direction of fit/or both, in being "*janus-faced*" cf. de Sousa, 2002) *plus* an other component to account for the phenomenological dimension of evaluative states. Whatever might be considered to be a defining factor of a certain type of evaluation seems to be simply *added on* as a further component to its intentional content.

One might object that my account faces this common problem, in failing to provide the required sufficient explanation of the interrelation of the components by which a particular type of evaluative state (for instance, an emotion) must be comprised. And inasmuch as different types of evaluations amount to the more complex evaluative meta-structure of one's actual existential situation, one has to present a theory that equally can account for the structural connectivity of evaluative content, too.

This can be tackled the following way: rather than focusing on an analysis of how these "components" either combine to constitute a particular evaluative state, or how different evaluative types exactly must be combined to form more complex evaluative patterns, these factors must not be treated as disjointed components: neither for the case of a particular evaluative state, nor for the case of a complex evaluative pattern, which structurally integrates the phenomenality and intentionality of more than one evaluative type in the process of evaluative experience. One can rather aim to explain, how these elements are necessarily interrelated due to

a *synthesis of evaluative experience*. This "synthesis-view" aims to take the phenomenology and intentionality as an integrative unity in evaluative experience fully into account[3] . Then the various "aspects" of being, for instance, in a particular type of evaluative state are perceived rather as *dimensions of the unifying structure of evaluative experience itself*. If one expands the synthesis-view, one can address the structural complexity of someone's actual evaluative situation *as a whole*: the different types of pre-reflective and reflective evaluation thereby are perceived as building the structure of more complex evaluative patterns by which several types of evaluative content become integrated. This means that different types of evaluations (e.g., desire, emotion, or the normative evaluation in case of belief, etc.) become structurally related with each other to form a more complex evaluative pattern for evaluative self- and world-disclosure. With this, one can conceptually address the evaluative totality, and, simultaneously, the procedural dynamics of evaluative self- and world-disclosure.

NARRATIVITY AS THE STRUCTURING PRINCIPLE OF EVALUATIVE EXPERIENCE

But one may still ask how such a *structuring principle* that guarantees for the experiential unity in processes of evaluative self- and world-disclosure may be described. This has to be explained in more detail, as I argue in the following steps that many forms of depressive experience can be explained due to fragmentations of this experiential unity.

THE NARRATIVE STRUCTURE OF EVALUATIVE EXPERIENCE

This is exemplarily outlined in Voss (2004, 185ff) thoughtful narrative account, in which she elegantly solves the component problem for the particular subclass of evaluative states, namely the emotions. In her analysis, *evaluative narratives* are carefully distinguished from *narration*. While "narration" is the result of purposive writing and speech, the evaluative narrative is the meaningful structure, which can become the *object of a narration*. Insofar as narration is an appropriate medium for transporting evaluative content, one can argue, in line with her account, for the *narrative structure of evaluative experience* in general. This plays a constitutive role for uniting phenomenality and intentionality in evaluative experience: narration is then the appropriate or inappropriate description of a desire, belief, (bodily) feeling, emotion, etc., which already points to the narrative structure of the particular evaluative content of a specific evaluation. More precisely: it points to the evaluative narrative as the *situational instantiation of the formal object of a certain kind of evaluative type*. The proposition of an evaluative content is never isolated from a corresponding, more detailed narrative structure of a specific evaluative state. Accordingly, the idea of a narrative structure of evaluative states suggests, that one therein refers to the specific evaluative content and structure of these states. As such, one refers to certain types *of significance or import*, which are instantiated in a specific situation. Thus, experiencing a particular type of evaluative state is equivalent to experiencing significance, to which a particular valence is always integral. This valence dimension has been exemplarily discussed with regard to the emotions, in particular, for its motivating role (e.g., Helm, 2010, 2001, 99ff). These experiences – that something or someone is experienced as having import, is of significance or is experienced as meaningful – are not solely provided by the reflective sphere, e.g., in virtue of self-reflexive processes (e.g., in terms of "caring," cf. Frankfurt, 1988; Jacobs, 2012, pp. 219–228), but also by the pre-reflective sphere, e.g., in terms of bodily appraisals (cf. Prinz, 2004, p. 77, 173) and other bodily corporal processes that positively or negatively reinforce individual practice of meaningful relatedness.

THE EXPERIENTIAL UNITY

The constitution of a *phenomenal unity* and its respective situational occurrence in the experience of a particular evaluative states then can be explained the following way: certain types of evaluative states are linked with evaluative concepts and their respectively embedded propositional content, which form the underlying "core"-intentional structure of an particular evaluative state; while the whole narration (e.g., in form of an oral or written report) conveys the structure of that evaluative content. The "evaluative narratives" are the more fine-grained structures, which contribute to the narrative *coherence* of the more "robust," single evaluative concepts of evaluative experience (cf. Slaby, 2008, p. 274). Such evaluative concepts (like, for instance, "threatening," "dangerous," etc.) that relate to the formal object (e.g., psychopath) of the respective evaluative type (e.g., anxiety) thereby amount to the *all-encompassing conceptual structure of a particular evaluative state* (e.g., of an emotion), which is expressed (e.g., communicated) in narration. Consequently, one can also account for the structural connectivity *of different evaluative states* in complex evaluative situations by pointing to *narrativity* as an adequate structuring principle. Then the complex phenomenality and intentionality of different types of evaluations become integrated into the *all-encompassing conceptual meta-structure of one's evaluative situation as a whole*. This refers to a synthesis according to which different experiential modes of evaluation become structurally interrelated in complex evaluative experience. Experiencing, e.g., anxiety when standing face to face with a psychopath then becomes structurally connected with isochronally occurring evaluative experience, for instance, with the desire to run away, the belief that one has to protect the child one hides behind one's back, with bodily feelings of being close to collapsing, etc., according to which someone's actual evaluative situation enfolds. This strengthens the hypothesis of a procedural dynamics of evaluation, i.e., it points to the continuous processes by which the evaluative structure of one's existential situation develops and by which one experientially traverses one's actual existential situation in multifaceted modes of meaningful relatedness.

THE NARRATIVE PARADIGM AND DEPRESSION

Although there are alternative ways to conceptualize the unity of evaluative experience, and albeit I do not claim narrativity to be a necessary condition for the conceptualization of the experiential

[3]I have elsewhere provided an answer in terms of a synthesis-account in order to tackle some problems of classic cognitivist and component theories of *emotion* (cf. Jacobs et al., 2013). This has been the background for claiming that an adequate theory of evaluation in general – and not only theories of emotions – has to explain both, the intentionality and phenomenology of evaluation (cf. Goldie, 2000; Helm, 2001; Döring and Peacocke, 2002; Stephan and Slaby, 2011), and how these form an *integrative unity in processes of evaluative self- and world-disclosure*, in particular.

synthesis in evaluative experience, it presents as a quite reasonable paradigm; especially, if one aims to explain the (structural) changes in depressive evaluation. One can describe on a conceptual level, how particular experiences of *evaluative incoherence* can be traced back to *inconsistencies* (typical deformations) in the evaluative structure itself. These cause "fragmentations" of the experiential unity in processes of evaluative self- and world-disclosure in depression[4]. Very often, such fragmentations of the experiential unity are already reflected in the incoherent styles of writing and speech in depressed patients (cf. Hilken, 1993; Hunsaker Hawkins, 1999). As such, *pathography* (Möbius, 1907) provides a very good insight to recurrent and significantly altered evaluative patterns of experiences in psychopathology (cf. Habermas, 2011). Consequently, I refer to narratives of depression to exemplify such experiences of evaluative incoherence. These may put some flesh to the conceptual bone of "depressive situatedness."

Moreover, narration is a particular type of sense-making-practice, in which evaluative content (experiences of import) is transformed – e.g., by verbal expression – to a speech *act*, and, as such, provides one way to address how evaluative experience instantiates and expresses in individual meaningful practice. The important role it plays in processes of coping, identity formation (cf. Bruner, 2001; Habermas, 2012) and self-knowledge (cf. Bruner, 1987) points also to a therapeutic value of the narrative paradigm (cf. Richert, 2006). This already has been pointed out by Freud (1905, p. 7) who characterizes neuroses as "gaps" in autobiographic narratives.

Given the plausibility of such a holistic, dynamical perspective on the individual evaluative setting, my analysis affirms a synthesis-view as essential for a description of experiential and practical changes of meaningful relatedness in depression.

THE DEPRESSIVE SITUATION – CHANGES OF PRACTICAL SENSE AND MEANINGFUL RELATEDNESS

It is next outlined what characterizes the *depressive situation*. I suggest that those processes in which different types of evaluation/evaluative states normally become structurally interrelated and integrated to consistent evaluative patterns for self- and world-disclosure are more prone to distortion in depressive as in healthy individuals. I focus in the following on how the otherwise "smooth" and flexible procedural dynamics of evaluation – denoting the openness of one's existential situation – come unstuck and therein amount to symptomatic depressive experiences of changes of meaningful relatedness, i.e., to particular experiences of evaluative incoherence and incapacity. These are addressed as distortions of practical sense in depression.

EXISTENTIAL OPENNESS AND ITS RESTRICTION IN DEPRESSION

Inasmuch as we situate ourselves in the world in ongoing processes of evaluative self- and world-disclosure, our existential situation normally *remains receptive* for experiential changes and different modes for meaningful relatedness, respectively. In contrast, the depressive situation rather points to the opposite of an existential openness.

Generally, one's meaningful self-world-relation evolves out of the proceeding dynamics of the pre-reflective and reflective evaluative spheres that provide the evaluative architecture of the existential situation. With these procedural dynamics one can, for instance, account for the coming and going of different evaluative states in a specific situation, as well as for the individual variances in evaluative reactions to the very same event, i.e., how individuals flexibly adapt to contextual constraints in a specific situation. In the continuous processes of registering, adopting, maintaining, reflecting or rejecting certain beliefs, desires, feelings, fantasies, values, ideals, etc. one navigates through the world as an evaluative being in more or less meaningful ways.

Habitual attunements

It has to be mentioned that a set of *relatively stable* (not rigid!) evaluative patterns for self-understanding and world-orientation become individually acquired. These evaluative patterns form a *habitual evaluative repertoire* that narrow a total existential openness, as it terminates the individual space of evaluative possibilities to a realm of actual evaluative capacity an individual has in specific situations. It naturally determines, for instance, the range of actions that might count as reasonable to one in a specific situation, and someone's evaluative responsiveness to a certain event. As such, these evaluative patterns already provide a (pre-)normative basis according to which we come to appraise (pre-)reflectively what makes sense to us or what is of importance in specific contexts of social interaction. This evaluative repertoire can also become the object of reflection, for instance, in processes of contemplation about the values, ideals, concerns, etc. that often deeply impregnate one's self-understanding and world-orientation.

Inasmuch as the existential situation in principle remains receptive to the changes of the evaluative structure, an essential structural requirement is provided for not experiencing oneself as fundamentally exposed to these processes. We normally experience ourselves not as fully determined in thought, feeling, desire, and by our bodily (pre-)dispositions, but rather as autonomous, thus, responsible agents (cf. Jacobs, 2012, 2013a). In contrast, the *depressive situation* is often shaped by the experiences of being existentially exposed, particularly, when depressive individuals register how their meaningful relation to the world, to others and their self-understanding has significantly changed. Such a decrease of existential openness exhibits, for instance, in a generally reduced sense for one's actual possibilities in life, e.g., due to a changed outlook on the world filtered by negative bias. It reveals in experiences of evaluative incoherence, for instance, in moments of being torn between conflicting desires, emotions, and in particular experiences of failures of intentional action (i.e., specific *inabilities*), or problematic coping strategies for dealing with such experiences of "losing grip" on one's life.

Depressive situation and (evaluative) incapacity

It is generally useful to differentiate between different realms of (in)capacity for a systematization of specific types of impairments

[4]Inasmuch as my account focuses on experiences of fragmentation due to significant structural changes in the evaluative processes in depression, it is evident that I do not stipulate such fragmentations as the experiential norm. It has to be discussed elsewhere, whether emphasizing on the latter would require to reconsider narrativity to be a necessary structuring principle, as it is, for instance, suggested by Ricoeur (1996, p. 174), who emphasizes on "discordance concordance" that likewise accounts for a synthesis of heterogeneous aspects of one's evaluative situation in virtue of narrativity as a (necessary) structuring principle.

involved in psychopathology, while the concept of "evaluative incapacity" is special for its integrative potential (Jacobs, 2012, 141f). One can argue for conceptual primacy of evaluation/evaluative incapacity in depression, insofar as specific affective/emotional, conative/volitional, and cognitive/rational disturbances in depression either present as particular subtypes of evaluative incapacity and/or significantly contribute to it. This mirrors the conceptual claim that the significant changes in the evaluative structure in depression, neither can perceived as exclusively based on irrationality (it is not just a matter of false beliefs), nor exclusively stemming from an emotional-affective, or predominantly volitive, or mere bodily corporal dimension. Insofar as different kinds of evaluations become interrelated and shape the special evaluative architecture of the actual depressive situation, one can account for different kinds of inabilities that specify the dimensions of evaluative incapacity in depression.

A prominent way to address from a naturalistic perspective on mental disorder what underlies such experiential and practical changes in depression is to refer to altered information processing in depression. One can exemplarily stress on cognitive *bias*[5] (e.g., cf. Beck, 1963, 1987; Clark et al., 1999) in reasoning and in processing of emotional information, including attention and memory (e.g., cf. MacLeod et al., 1986; Williams et al., 1997; Gotlib and Neubauer, 2000; Gotlib et al., 2004; Joormann and Gotlib, 2007), and *distortions* in logical thinking (e.g., Ellis, 1962) in depressed individuals. These count either as vulnerability factors, or as already symptomatic for the depressive situation. These descriptions of involved dysfunctional processes provided by clinical research on depression can be related more closely to the phenomenological analysis of depressive experience, insofar as these refer to the neuropsychological correlates. In the following section, I address those as "pathogenic restrictions of existential openness" in depression. These restrictions appear more specific in lights of these biases, for instance, the "rigidity" of certain evaluative patterns for self- and world-disclosure.

Depressive bias and rigid evaluative patterns

This rigidity can be explained in terms of negative biases which have to be considered for both, their role in implementing the rigidity on a structural level of evaluation, and for simultaneously representing it. Depressed individuals show a tendency to focus explicitly and exclusively on their own alleged negative traits, inabilities and failures. These biases already represent how certain affects, beliefs and desires that formerly might have stood rather in contrast to one's evaluative patterns for self- and world-disclosure and self-understanding, deeply infiltrate a persons' evaluative system and install a different evaluative dynamics. As such, biases represent and contribute to a reduction of the existential situation to only a fractural amount of experiential and practical possibilities for meaningful relatedness, as there have been before the onset of depression. Many of the evaluative patterns that have provided one with possible and actual modes for evaluative self- and world-disclosure so far lose their practical significance, because other evaluative pattern stake over the depressive person's life.

It has become transparent that not only specific *feelings*, but also characteristic *beliefs*, i.e., their normative evaluative content ("I am a terrible, ugly, selfish, unworthy, etc. person and deserve to suffer") and *desires* ("I just want to die") together with the evaluative bodily corporal rationale (e.g., psychomotor agitation, pain, loss of appetite, etc. cf. DSM-IV-TR (American Psychiatric Association, 2000), are considered characteristic types of evaluation that shape the self- and world-relation of the depressed. Being in such a way attuned to the world, depressed persons find themselves evidenced by very single negative experience. These confirm the structure of evaluation represented by the biased view. Such experiences, and respective appraisals, loop back, i.e., become re-incorporated, to the very evaluative structure from which they have arisen. The development and manifestation of the depressive self- and world-relation follow the logic of self-priming looping dynamics: the more one experiences and behaves in a "depressed" way, the more these experiences become woven into one's personality structure, which then contributes simultaneously to the (re-)production of certain behavioral patterns. It is this kind of vicious circle by which the depressive situation becomes manifest in rigid patterns for world- and self-disclosure.

Besides vulnerability factors and individual difference in one's habitual (pre-)dispositions, of course, specific experiences, e.g., such of reinforcement by the environment and one's own individual practice of dealing with these experiences influence these self-priming dynamics. In order to alter both, incorporation and enacting of these structures, something from the "outside" has to come (e.g., therapeutic invention) or from the "inside" (e.g., self-reflexivity or introspection) that may eventually re-shape these perpetuating dynamics. Consequently, it is an aim of therapy to "crack" the structural rigidity in order to alter the experiential and practical modes of relatedness stemming from these so that depressed persons are able to restore a kind of experiential and practical openness of their existential situation.

The following quote illustrates that this may mean hard work, as to be existentially situated in a non-depressive way often has become literally unthinkable for the severely depressed. Alternative modes of being related to the world stay out of experiential and practical reach in feeling, thought, desire, embodiment and action:

"When you are depressed, the past and the future are absorbed entirely by the present moment, as in the world of a three-year-old. You cannot remember a time when you felt better, at least not clearly; and you certainly cannot imagine a future time when you will feel better. Being upset, even profoundly upset, is a temporal experience, while depression is a-temporal. Breakdowns leave you with no point of view." (Solomon, 2001,p. 55).

The quote from Solomon illustrates how the fundamental restriction of one's existential situation becomes transparent in terms of temporality, too. The reverberation of a non-depressive past and the anticipation of a future without having depression are fundamentally restricted, as one sticks to the present moment,

[5]A bias is "a proclivity to take one direction over another which under same conditions will lead to accuracy or realism, but under other conditions will lead to inaccuracy"; while "distortion" implies something invariably wrong (cf. Power, 1991; Power and Dalgleish, 2008, p. 247).

which forms an isolated and disconnected experience of presence (for detailed analysis of altered temporality in psychopathology, see, e.g., cf. Bech, 1975; Ciompi, 1988; Mundt et al., 1998; Fuchs, 2001, 2005a, 2013; Habermas et al., 2008). As depression is often a long-term condition, the "depressive" evaluative patterns can become part of the habitual evaluative repertoire by means of their structural rigidity, which then shapes the *depressive habitus*[6]. With respect to the self-perpetuating evaluative dynamics by which the depressive situation enfolds as a long-term condition, a chronic disease management model for depression is needed (cf. Andrews, 2001). At least, the high co-morbidity of depressive symptoms with long-term conditions, e.g., diabetes (cf. Anderson et al., 2001), heart diseases, anxiety disorders (e.g., Paschalides et al., 2004), and physical conditions with inflammatory processes (cf. Harrison et al., 2009), point toward the relevance of perceiving the depressive situation in lights of long-term impairment. This is reflected in its conceptualization as a specific type of existential situation that challenges individuals to develop long-time oriented coping strategies (cf. e.g., McEvoy and Barnes, 2007; Smit et al., 2007; Naylor et al., 2012) to counterbalance the structural changes of evaluation by which it manifests as a harmful condition of existential narrowness.

(DISTORTIONS OF) PRACTICAL SENSE IN DEPRESSION

It is with respect to the openness of one's existential situation that one is able to re-evaluate and to readjust in the light of new experiences and practice of sense-making.

"I can(not)"

Normally, we register when things might (have) go(ne) wrong, and when particular modes of meaningful relatedness become inherently problematic. It is in virtue of *practical sense*, that we realize the possibilities and restrictions, thus the opportunities and limits for reassessment and readjustment in our life. The notion of *practical* sense reminds us of that according to which the world and self are perceived not solely in terms of an abstract "I think that," but in terms of an "I can" as Maurice Merleau-Ponty emphasizes in his *Phenomenology of Perception* (Merleau-Ponty, 1962, Part I, Chapter 3, §19, p. 159). Martin Heidegger (cf. Heidegger, 1962, 114, §18) expresses the idea that the world is perceived as a place in which things appear to individuals not only as "present-at-hand" (*German: Vorhandenheit*), but is normally experienced as a place of (normative) affordances, according to which things appear to them as "ready-to-hand" (*German: Zuhandenheit*). Practical sense apparently influences not only *what* we do in a specific situation, but significantly contributes to *how* we are existentially situated in the world, i.e., how we enact in and through processes of pre-reflective and reflective evaluation, by which a particular meaningful relation between self, others and the world is instantiated (cf. Jacobs, 2013a). As such, it provides us also with modes for readjustment, for coping and adaptation by implementing a "world of possibilities." This is also integral in Husserl's (1960) and Merleau-Ponty's (1962) concepts of *horizon*, where the horizontal structure of experience particularly points to how the body sets up the world, and how this is implicated in someone's particular experience of the world. Although many alterations of practical sense cannot be labeled pathological *per se*, existential situations of the pathological type very often include significant distortions of practical sense.

Evaluative incoherence in depression

In psychopathological conditions, like depression, such experiences of being vitally connected to the world and others through the body, which normally is a transparent medium for evaluative self- and world-disclosure, can become severely distorted (cf. Svenaeus, 2000; Fuchs, 2005b,c) as the following quote from Solomon illustrates:

"I found everything excruciatingly difficult, and so, for example, the prospect of lifting the telephone receiver seemed to me like bench pressing four hundred pounds." (Solomon, 2001, p. 85)

This is one way to account for a distortion of practical sense in depression. The question is, whether one can address on the bodily corporal level a fragmentation of the experiential unity in evaluative self- and world-disclosing processes with this example: I believe that the experience of "difficulty" in this example, indeed, reflects a type of experiencing evaluative incoherence. The appraisal is not solely provided by reflective assessment, but given in virtue of pre-reflective evaluative appraisals. Incoherence does play a role, insofar as one perceives the actual bodily corporal condition ("I cannot") always against the backdrop of the condition (the bodily corporal "I can") prior to the onset of one's depression. The incorporated sense of being able to behave and to act as one normally does, becomes fragile, i.e., corporal-bodily possibilities cannot be actualized in such specific situations of being unable to even lift the telephone receiver.

"Evaluative incoherence" also captures more severe cases of estrangement, alienation, and even de-realization/depersonalization-processes in depression. The experiential unity is fragmented in such moments of experiencing oneself no longer the center of one's own perception or losing trust in these. Inasmuch as perceiving oneself, others and the world in a particular way already entails processes of evaluation, Thompson's example of altered self-awareness accounts for this distortion of the experiential unity, too:

"I began to lose faith in my own perceptions. It was as if I were standing in front of a mirror which was gradually getting distorted; eventually, what I saw bore little relationship to reality, but the change had been so slow that I had no idea where the distortion began." (Thompson, 1996, pp. 125–126)

Another option to specify distortions of practical sense is to point to the scenario in which the "I cannot" stems from being overwhelmed by the sheer concomitance of evaluative experience occurring in a specific situation, and/or from being torn by evaluative conflict. The more complex a situation is, the more likely it is also that conflicting evaluative content (desire to kill oneself

[6]With Bourdieu's theory, the bodily disposition and specific gestures, postures and body-language of depressive persons can be systematized and decoded as reflecting the historicity and actuality of a depressed individual's whole bio-psycho-social status. These aspects of "depressive habitus" exemplarily have been detailed by the German psychiatrist Hubertus Tellenbach, who addresses the habitual dimension of depression – inclusive its characteristic *hexis* – with his famous "*Typus Melancholicus*" (cf. Tellenbach, 1961; see also Jaspers, 1913, 141–144; §5; Fuchs, 2006).

vs. desire to take care for one's children) is present, and sometimes needs to be additionally assessed against the backdrop of corresponding different evaluative types (a belief, that leaving the children is selfish vs. the feeling that nothing makes sense any longer). On an experiential level, these evaluations demand their structural integration into a consistent evaluative pattern that allows for evaluative coherence.

Structural inconsistencies normally can be solved, e.g., in self-reflexive processes, or simply resolve themselves due to the (phenomenal-intentional) strength of a particular evaluative type/evaluative pattern overriding the other(s), and thus, initiates the respective experiential and practical mode of meaningful relatedness. We often also stay in the particular evaluative state of ambivalence, which clearly presents a different type of meaningful relatedness as when one is stuck in the process of constant rumination and indecisiveness, as it is seen as symptomatic for depression (cf. DSM-IV-TR; American Psychiatric Association, 2000, p. 349; Watkins and Teasdale, 2001). The particular symptomatic experiences can be explained in terms of distortions in one's self-reflexive evaluative processes. These become surface, for instance, in a depressive person's inability to affirm or distance oneself from certain thoughts, desires, or feelings and to adopt an objectifying stance that normally would give one some time to recognize what one really thinks, feels, desires in a specific situation. Even if one associates constant rumination with such a self-reflexive stance, the ability to rank preferences, i.e., to create a hierarchy of motives, according to which that what is of importance, normally becomes clear to someone – seems impaired in depression, as the symptom of depressive indecisiveness suggests.

Being capable to decide is often perceived as a structural requirement for effective deliberation, and thus significantly contributes to experience of not being fundamentally determined and existentially exposed. Consequently, one could assume that if these structural divergences in evaluation – which also presents themselves as divergences of import – remain unresolved, not only the integrative unity in evaluative experience is put at risk (with Harry Frankfurt we can even claim a distortion of the synchronic unity of the *self*; cf. Frankfurt, 1988, 2006, p. 19), but in tendency also an agent's autonomy. In emphasizing on such moments of structural evaluative inconsistency, and corresponding modes of incoherent evaluative experience, an alteration of practical sense in depression is addressed, in which the "I cannot" shows in a distortion of particular self-reflexive processes.

It can be objected, however, that there are many other ways to specify an "I cannot" in depression, and that these examples, moreover, show that it is misleading to tie evaluative incapacity too close to specific clinical symptoms. This can be outlined by a contrary case of depressive *decisiveness*. Imagine, for instance, the situation in which the desire to kick the ladder on which one has stood already for an hour with a rope around one's neck, is neither outplayed by the desire to be a good mom, nor by feelings of love and sorrow for the children one would leave behind by committing suicide, and does not results in the decision to climb down the ladder to prepare breakfast for them, but leads one to "wholeheartedly" kick the ladder and hang oneself. The evaluative incoherence assumed as pressing in the situation, as such, is definitely solved. Respectively, it seems to represent rather a classic case of "I can" in depression, thus, rules out the description of evaluative incapacity in virtue of the clinical symptoms provided above, too.

To my defense, it can be assumed that effective suicide is frequently committed in phases of recovery (cf. Schweizer et al., 1988; Mittal et al., 2009), thus, when it is likely that someone has restored some psychic and physic resources, that may allow one to effectively deliberate, in contrast to those phases when one cannot even perform the simplest tasks of daily life. Moreover, one may doubt, whether this example really can account for an intact practical sense, albeit an "I can" is involved. The example shows that a depressed individual may be able to decide, but is not able to *care* for oneself, for other people, and for the things that have been close to one's heart, at least in such ways that committing suicide remains the "unthinkable" option.

The example further illustrates that the "I cannot" generally allows not only for many ways to account for evaluative incoherence and particular types of incapacity, but to generally state evaluative incoherence and (in)capacity in depression as a matter of *degree*. It points toward the in-between of the two poles of global incapacity (e.g., absolute indecisiveness) vs. fully intact capacity (e.g., "wholeheartedly" committing suicide). I do not argue, indeed, that depressives *generally* face global evaluative incapacity, but that they apparently show a diminished *coping flexibly* with such stresses and strains on the evaluative structure by which the challenging normative aspects of complex evaluative situations become transparent.

THE NORMATIVE DIMENSION OF THE DEPRESSIVE SITUATION

By relying on the notion of practical sense, one can further elaborate, how individuals calibrate their individual experiences of significance (*Sinn*) with those objective patterns of meaning (*Bedeutung*) that define what actually "makes sense" in specific contexts. As such, practical sense illustrate show subjective evaluative patterns for self- and world-disclosure become interrelated with the objective evaluative patterns of meaningful (social) practice. To be more precise: the *mediating function* of practical sense provides one not only with a sense for one's own individual evaluative situation in terms of an "I can" (I have addressed elsewhere with a sense of agency cf. Jacobs, 2012), but also with an understanding of it *in relation* to those patterns of meaning generated in processes of inter-subjective practice (cf. Jacobs, 2013b). Individuals are able to recognize the respective patterns of meaning generated in and through social interaction, and, simultaneously, *register how they fit into that.* They experience how their individual meaningful practice matches or mismatches actual contextual (conventional, moral, social, etc.) requirements and standards of inter-subjective practice (e.g., what it *means* to be a good mom, and what sense it makes to commit suicide). It is with respect to practical sense that one counterbalances one's evaluative experience and practice against the pre-descriptive (normative) sphere of meaning in the respective fields of social practice. With respect to modes of (re-)adjustment this implies that we do not only ask what we actually *can* do, but what *should* be all things considered the best way to do. Practical sense therein refers to more than what is captured with an "I

can," as particularly the *normative dimension of evaluative encounters* with the world in terms of an "I should" becomes addressed with it[7] .

One should note that meaningful relatedness does not have to automatically merge into the experience of a mere match of individual evaluation (e.g., moral judgment) with objective evaluative patterns (e.g., moral rules). There are several other modes of meaningful relatedness that rather rely on *distinction* (as Bourdieu, 1979 emphasizes) by which one can stress (habitual) evaluative divergence and difference as essential for one's self-understanding and one's world-orientation, too. Depressed individuals certainly experience themselves as distinct, but it would often be cynical to account for these experiences as modes of distinctive practice that normally are powerful tools for positioning oneself in the fields of social practice. Patricia Dunker reflects this in her novel *Hallucinating Foucault*:

"And that is the loneliness of seeing a different world from that of the people around you. Their lives remain remote from yours. You can see the gulf and they can't. You live among them. They walk the earth. You walk on glass. They reassure themselves with conformity, with carefully constructed resemblances. You are masked, aware of your absolute difference." (Dunker, 1996, p. 110).

This points to a specific subclass of normative conflict in depression, in which the imperatives of an "I should" (in terms of incorporated social, cultural, moral, etc. structures) stand in conflicting relation with particular experiences of an "I cannot" – as the case example of suicide has exemplarily shown – that forms a fundamental source of suffering. This is addressed in many prominent depression narratives (e.g., Plath, 1963, p. 102, 137; Styron, 1990, p. 38; Thompson, 1996, p. 47, 57), and described also in the following passage by Solomon:

"[...] I would sometimes start to cry again, weeping not only because of what I could not do, but because *the fact that I could not do it seemed so idiotic to me.* All over the world people were taking showers. Why, of why, could I not be one of them? And then I would reflect that those people also had families and jobs and bank accounts and passports and dinner plans and problems, real problems, cancer and hunger and the death of their children and isolating loneliness and failure; and I had so few problems by comparison, except that I couldn't turn over again. [...]Always at the back of my mind there was a voice, calm and clear, that said, *don't be so maudlin; don't do anything melodramatic.* […] get dressed, and *do whatever it is that you're supposed to do.* I heard that voice all the time, that voice like my mother's. There was a sadness and a terrible loneliness as I contemplated what was lost." (Solomon, 2001, pp. 52–53; emphasis added K. J.).

Depressed individuals experiences themselves not only as deprived of the resources to deal flexibly with the stresses and strains on their evaluative structure arising out of an "I cannot"; they simultaneously know – or at least, remember – that they normally could or even should have performed in a specific way that once was experienced as meaningful, as a quote from Duke and Hochman illustrates:

"Those periods when I stayed in bed, behind closed doors for all those weeks, I felt dirty, smelly. Part of me thought *I should* get up and wash myself, and then I would dismiss that because I couldn't get up. My thoughts would vary from blaming others to wishing for the absolutely unattainable peace of mind. And also thoughts of: Why is this happening to me? *Why am I like this? I'm a terrible person.*" (Duke and Hochman, 1992, Chapter 7, paragraph 19; emphasis added K. J)

Consequently, the distortion of practical sense points to the problematic evaluative self-relation that does not allow for the experiential and practical modes of transcending the depressive situation in light of an existential openness; it rather harmfully reminds one that experiencing oneself, others and the world as meaningful points of reference, has lost most of its practical significance.

CONCLUSION

I have examined the depressive situation as a specific type of existential situation and emphasized the procedural dynamics of the pre-reflective and reflective spheres of evaluation, narrativity as a structuring principle for the experiential unity in evaluative processes, processes of evaluative disintegration and experiences of evaluative incoherence, and how these appear as distortions of practical sense in depression. First person accounts of depression from the memoirs genre served to illustrate these contexts. These have evidenced changes of meaningful relatedness in depression in pointing to recurrent experiential patterns of evaluative incoherence and incapacity, and moreover to the normative challenges associated with these. It seems that some of the phenomena I have discussed should be given more attention in diagnostic-clinical theory and practice. Although these may not share the grounds of operationalization, and respective validity and reliability as claimed for the official criteria listed in the diagnostic manuals, they may be of diagnostic value in addition to the current criteria. It is, moreover, another issue to show how such phenomenological descriptions of evaluative experience can be emphasized more in the context of future empirical research that aims to single out the neuropsychological correlates of the complex dynamics of pre-reflective and reflective evaluative self- and world-disclosing processes, and how these can substantiate some considerations of characteristic distortions in depression that have been discussed.

Although my analysis is restricted in scope, I believe that I have provided some reasons to accept that a holistic perspective on (changes in) evaluative processes in depression may contribute to a better understanding of its characteristic modes of self- and world-disclosure.

Future research will focus on an even more detailed analysis of salient changes of meaningful relatedness in psychopathology, especially with respect to the bodily corporal dynamics as constitutive for processes of normative self- and world-disclosure. A related methodological topic is to continue relating phenomenological considerations closer to empirical research and

[7]The notion of practical sense therein contributes to an understanding of social, particularly: *moral* practice. A lack of moral sense (moral incapacity) can be addressed as a subtype of distortion of practical sense to experience oneself as meaningfully (morally) related to others (cf. Jacobs, 2012, 2013a).

normative considerations about such specific concepts as agency, capacity, and autonomy, which are central to the notion of existential situation.

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Narrative or self-feeling? A historical note on the biological foundation of the "depressive situation"

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In her paper "The depressive situation" Jacobs (2013) stresses the role of normative evaluative and narrative processes in what creates the lived experience of depression. These "evaluative dynamics" are seen to emerge out of an "interplay of pre-reflective and reflective processes," which are claimed as being "significantly altered in depression." Even if acknowledging a pre-reflective level of the processes of evaluative self- and world-disclosure, Jacobs argues that this "cannot be reduced to or equated with a mere affective (or: "felt") dimension." Jacobs' philosophical analysis ties in a way on the cognitive interpretation of depression that has prevailed since the 60s especially due to the work of Aron Beck and the development of cognitive psychotherapy for depression (Beck, 1975, 1979; Clark et al., 1999). However, Jacobs well admits that affectivity provides a mode of an evaluative process.

Interestingly - and in contrast to Jacob's theory -, this *affective*, felt, dimension had once been considered as absolutely crucial for the "depressive situation." But this was long before the cognitive turn in psychology overwhelmed psychiatry.

So, my comment just wants to remind of the role of affectivity, or more precisely what was called "self-feeling," in the very early theories of depression and mental disorder that were formulated at the beginnings of psychiatry as a medical discipline.

SELF-FEELING AS Gemeingefühl

The English term "*self-feeling*" was introduced into psychiatry by Alexander Crichton in his "Inquiry into the Nature and Origin of Mental Derangement" as a translation of the German "physiological" notion "'Selbst-gefühl' and 'Gemeingefühl'" (Crichton, 1798, p. 113). "*Physiological*," because in Johann Christian Reil's theory of "Gemeingefühl" as the somatic foundation of self-consciousness, mood, temper, volition and behavior - "corresponding exactly to the feelings that we call the temperament of persons" - self-feeling was thought of as emerging primarily only from the physiological arrangement of the nervous system (Reil, 1794/1817, pp. 83–84).

SELF-FEELING AS Selbstgefühl

The philosophical notion "Selbstgefühl" had already been introduced by Johann Bernhard Basedow in 1764 with reference to Locke as the translation of "inner sense" (Drüe, 1994) and was embraced with enthusiasm in philosophical discussions in the 1770 about self-consciousness and its relation to some sort of inner sense – crystallizing within a single notion the romantic idea of the *primary importance of feeling* (Frank, 2002). The *affective structure of self-feeling* in contrast to pure somatic perceptual or, on the contrary, the cognitive meaning of self-feeling as inner sense was repeatedly stressed.

Self-feeling was thought as the most basic and primordial sense of subjectivity, as the direct experience of existence: a feeling of being, an existential feeling. In this line, the term "Selbstgefühl" had also been chosen as the translation of the French "*sentiment de soi-même*," a feeling inseparable of the feeling of existence, "*sens intime de l'existence*" or "*sentiment de notre propre existence*" as it was called also by Rousseau and the Encyclopaedists (Frank, 2002, p. 79f). Remarkably, both these notions ("feelings of being" and "existential feeling") are today used by phenomenologist Matthew Ratcliffe especially in reference to a phenomenological analysis of depression (see Ratcliffe, 2008; Slaby and Stephan, 2008).

Moreover, romantic philosophers stressed the relation of "Selbstgefühl" to the latin "*sensus sui ipsius*" that, in the stoic tradition, denotes a self-feeling that every sentient living being has by nature as an integral affective component of its striving for self-preservation. In its very striving, an animal displays a background orientation towards its own life as something worth preserving (Frank, 2002, p. 28).

SELF-FEELING, PHYSIOLOGY, AND MENTAL DISORDER

Even if there was a vivid debate between philosophers, romantic physicians and psychiatrists concerning the correct theoretical explication and especially the *biological* basis of self-feeling, it was seen mostly as layered in itself, Gemeingefühl being the lowest most primitive form (Burdach, 1828, p. 166). In its higher levels, self-feeling was to include such background longlasting feelings as mood and temper. In this line, the 1845 textbook "Principles of Medical Psychology" - written by the romantic psychiatrist Ernst Freiherr von Feuchtersleben who coined in this context the term "psycho-somatic" - explains:

> "Self-feeling unifies sensation and representation. In self-feeling, subjectivity permeates the organic body; it is what we mean when saying "I" and it provides the ground for all other feelings and emotions. Within self-feeling, the Gemeingefühl takes on a human character. The pleasantness of the latter is transformed into cheerfulness, displeasure into sadness, the changing and interplay of these states is called mood and so it comes that human weal and woe exist trough this channel of inner life." (von Feuchtersleben, 1845, pp. 137–138).

SELF-FEELING AND DEPRESSION

Introduced by romantic psychiatry, the idea of self-feelings being diminished in depression had been central to early German and French psychiatry.

Already in the first edition of his psychiatric textbook, "The Pathology and Therapy of Mental Diseases," Wilhelm Griesinger noted that "especially melancholic's" complain of a "kind of anaesthesia: I see, I hear, I feel, they say, but the object does not reach me; I cannot receive the sensation; it seems to me as if there was a wall between me and the external world"(Griesinger, 1845, p. 67). Griesinger thought this form of emotional anaesthesia to be due to an "anomaly of Gemeingefühl" (Griesinger, 1845, p. 65).

In early German and French psychiatry, changes in self-feeling were seen as the core of melancholia or lypémanie, the conceptual precursors of depression. Reporting about patients that "complained almost in the same terms of a lack of sensations, [. . .] to them it was a total lack of feelings, as if they were dead," Albert Zeller had explained their symptoms as a disorder "self-awareness," of "Gemeingefühl" or "self-feeling" (Zeller, 1838, pp. 522–525).

Friedrich Schäfer classified similar observations as a subtype of melancholia, naming this subtype *Melancholia anaesthetica*: "when these patients complain about their suffering, they relate it explicitly to a sort of emptiness, hollowness in their head, or in the pit of their stomach; of a discomfort of not reaching the surroundings with their inner selves. They see and hear everything, but without experiencing any representation or feeling of their inner stirrings, of their sensory vividness" (Schäfer, 1880, p. 242).

Published in the same year as Feuchtersleben's textbook, also Gotthilf Heinrich Schubert's "Diseases and Disorders of the Human Soul" explained, that it would be "the self-feeling of the melancholic that is tarnished and impaired" – a distortion that robs the melancholic of every energy to entertain any wantings, and this while having full insight into his own misery;" a state that Schubert called "paralyzing melancholia" (Schubert, 1845, p. 303).

In France, Jean-Étienne Esquirol blew long in the very same horn: "An abyss, they say, separates them from the external world, I hear, I see, I touch, say many lypemaniacs, but I am not as I formerly was. Objects do not come to me, they do not identify themselves with my being; a thick cloud, a veil changes the hue and aspect of objects" (Esquirol, 1838, p. 414).

Taking a description of a similar state in Amiel's "Journal Intime," Ludovic Dugas coined the term "*depersonalization*" in 1898, initiating a rich debate about the correct phenomenological description, classification and biological underpinnings of these symptoms of emotional numbing, anomalous body experience, anomalous subjective recall and alienation from one's surroundings (Dugas and Moutier, 1911; Sierra and Berrios, 1997).

In the light of the complaints so often uttered by depressed patients that they "feel nothing," that they are no more than "a corpse," or even to "be dead," it was Jules Cotard who especially focused on these symptoms of anomalous corporal experience - ranging from somatosensory distortions, feelings of disembodiment up to the loss of body ownership feelings - in his 1880 lecture "On hypochondriacal delusions in a severe form of anxious melancholia," later on called "délire de negation" – délire however being a complex of emotional, volitional and intellectual symptoms and not a delusion in the sense of a false idea or thought or disordered thinking (Cotard, 1880, 1882).

Findings of modern neuropsychiatric research also speak for a *biological foundation of these symptoms*, probably involving a down regulation of the amygdale because of stress induced hyperactivity in the orbito-frontal cortex (Sierra and Berrios, 1998). Whatever the complete and correct biological explanation of the impairment of self-feeling seen in the emotional numbing and the related inability to color experience with feelings, these affective changes in depressive illness seem to pertain to a very basic affective dimension of self-feeling, as many autobiographic patient narratives and memoirs illustrate in drastic terms (see, e.g., Styron, 1990; Kuiper, 1995; Thompson, 1996; Solomon, 2001; Brampton, 2008; see also Ratcliffe, 2008).

The utter strangeness and distance from everyday experience of these symptoms encountered in severe melancholic depression is what can present a real challenge in treating depression. Such a complete loss of emotional colouring – a colouring taken for granted in our everyday lives – is almost impossible to imagine. Moreover, it seems not to be a result of a narrative or cognitive evaluation. Consequently, it cannot, so to say, be discussed away. Therefore, these severe depressive symptoms might remain quite resistant to psychotherapy, especially cognitive psychotherapy, but show most often relievable by somatic therapies as medication or electro-convulsive therapy (APA, 2001, 2010).

CONCLUSION

Romantic psychiatry knew of a notion of "self-feeling" as referring to a pre-reflective *affective* form of self-awareness that is the foundation of a person's mood and temper and with these of her motivational, evaluative and practical perspective on the world (vgl. Slaby, 2012). In contrast to its remote ramifications with terms such as "self-confidence" or "self-esteem," "self-feeling" also encompassed the opposite of self-confidence, i.e., states of low self-esteem, self-depreciation, submissiveness and the like and was thought to originate in a very basic somatic organismic dimension: a quite basic feeling of vitality or "vital tone" of a person's existence. A feeling so basic and seen as granted that only loss of or change in it - as in depression - might remind of its existence.

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Different conceptions of mental illness: consequences for the association with patients†

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Whenever partial knowledge is considered absolute and turned into ideological and dogmatic conceptions, the risk increases that the conditions for the people involved might become dangerous. This will be illustrated by casuistic examples of consequences of one-sided psychiatric conceptions such as social, biological, and psychological ideas about the treatment and care of the mentally ill. Present perspectives of an integrative model, i.e., an advanced bio-psycho-social conception about evidence-based characteristics on the social, psychological, and molecular-genetic level, require that all of these dimensions should be considered in order to personalize and thereby improve the care and treatment of the mentally ill.

Keywords: conceptions of mental illness, absolutization of partial conceptions, social space of the family, instrumentalization of the ill, social hygiene, preference diagnosis, normative content of human experience, bio-psycho-social model

The development of psychiatry has been accompanied by the debate about various conceptions of mental illness and the prevailing of one or the other of them at different times. Different consequences on attitudes in treating and caring for mentally ill patients of always one prevailing conception has been the stronger the more the conception focused only on a partial aspect of mental illness. This will be illustrated by casuistic examples

1. for the relationship of conception to practice
2. for the consequences of (at least of one-sided) conceptions on individual patients
3. Finally in contrast to such absolutization of partial conceptions ("Verabsolutierung von Partialerkenntnissen" (Jaspers, 1913) an integrative concept will be suggested.

CONCEPTION AND PRACTICE

The German epigonal placarding of the controversy between Heinroth[1] (and Ideler[2]) on the one hand and Jacobi[3] as well as Nasse[4] on the other hand as one between "psychicists" and "somaticists" in the first half of the nineteenth century disregards the fact that these representatives of different conceptions of mental illness in practice had more in common than was separating them (Kutzer, 2003). Both reasoned rationally with the conception of excitability developed by the Scottish physician John Brown ("Brownianism") – his thesis of excitability dominated medicine at that time – and planned their treatment with mechanical coercion measures as "contrastimulation" (Schott and Tölle, 2006).

My opinion is that the practice of treatment and care of the mentally ill depends less upon a disease conception but more upon the experience, attitude, and personality of the psychiatrist – at least in his individual development.

However, this does not mean that there is no influence from the commonly accepted conceptions of the time. Thus the psychicist-versus somaticist-controversy may also indicate how much the reception of conceptions depends upon the connotations of the respective epoch. Today both terms are used in an almost opposite meaning: whereas psychicists such as Heinroth – at that time in the tradition of the Romantic – ascribed mental illness to the emotions of guilt about a sinful and failed life and reasoned with this a treatment with mechanical ("somatic") coercion measures as a pedagogic therapy that currently is considered inhumane; but today the psychicists are misunderstood as representatives of a morally positively seen psychological-psychotherapeutic medicine. Conversely, at that time "somaticists" were psychiatrists who – such as Griesinger[5] – ascribed mental illness to brain diseases and thereby were seen as more modern and humane, whereas

[1] Heinroth, Johann August Christian (1773–1843), founder of the worldwide first Chair of Psychiatry at the University of Leipzig (1811).
[2] Ideler, Karl (1795–1860), head of the "lunatic asylum" at the Charité in Berlin.
[3] Jacobi, Maximilian (1775–1858), founder of the mental hospital in Siegburg, near Bonn.
[4] Nasse, Friedrich (1778–1851), Bonn.
[5] Griesinger, Wilhelm (1817–1868), chair of psychiatry at the Berlin Charité 1865–1868.

nowadays "biological" psychiatrists are criticized as biological reductionists. However in the first half of the nineteenth century the introduction of a somatic conception of mental illness was a great step forward in the direction of appreciation of the mentally ill as ill persons [therefore it is no surprise that Griesinger also supported psychotherapy (Tölle, 2002)]. Today further important aspects of these conceptions are recognized: the risks of passivity and dependence of the medical-somatic disease conception, which protects the ill, and guilt in the disease conception of the psychicists, which also indicates self-responsibility.

UNCONDITIONALITY OF PARTIAL CONCEPTIONS

Even if psychiatric disease conceptions are attenuated or changed by medical experience, they can develop considerable effects, particularly if they do not grasp the complexity of mental illness but only a partial aspect of it and if this is then accentuated dogmatically. This is the case especially with persons outside of psychiatry who know the world of the acting medical persons only indirectly or only by hearsay. This will be made clear by three concepts or clusters of concepts: that of social psychiatry, that of biological psychiatry, and that of psychological medicine respectively psychotherapy.

SOCIAL PSYCHIATRY

In the 1960s social conditions and consequences of mental illness increasingly came to the fore with young psychiatrists. In the UK – as the cult movie "Family Life" suggested – the social space of the family was seen as pathogenic; mental illness was understood as a reaction to a morbid society; or mental illness was even asserted to be a fiction of psychiatrists, most valuably by the Hungarian-American psychiatrist Szasz (1961).

In Italy the unbearable conditions of accommodation or custody of the mentally ill in large psychiatric hospitals, such as in Görz and Triest in northern Italy, caused the psychiatrist Basaglia (1968) to "liberate" these mentally ill by "negation," i.e., to urge the closure of these large hospitals and, thanks to successful political exertion of influence, to realize this with the law number 180 in 1978.

This forcible and radical reform in Italy led to the disadvantage of many severely mentally ill persons and their helplessly overburdened families, who had to take in their otherwise not cared-for ill family members, and not before, with the development of community mental health centers, the basic idea of extramural-rehabilitative support of the mentally ill gained acceptance (Pycha et al., 2011). Still, the older history of social psychiatry shows that such ideological excess of a right basic idea leads to instrumentalization of the ill and finally to inhumanity.

The term "social psychiatry" appeared in the beginning of the twentieth century (Ilberg, 1904) in the context of terms such as "social pathology" or "social hygiene" as a rational reasoning for governmental efforts to control the social conditions and consequences of mental illness (e.g., syphilis, alcoholism, "asocial" psychopathy, vagabondage) by social, particularly even eugenic measures (Grotjahn, 1912; Rüdin, 1931; Priebe and Finzen, 2002; Schmiedebach and Priebe, 2003). During the economic misery after World War I these aims of social psychiatry were radicalized by the eugenic and thereby biological ideas of "racial hygiene" all the way to "euthanasia" (Schmiedebach and Priebe, 2003).

This process of convergence, even merging social psychiatry into "racial hygiene" caused other, much older forms of philanthropically or economically motivated forms such as "family care" or "open care" as social support systems for the mentally ill outside the asylums to fade into the background and narrowed them to modes of social control of the mentally ill. This development became terribly clear with the "reform" psychiatrists Paul Nitsche and Valentin Faltlhauser, who stipulated in the 1920s to bring the mentally ill out from hospital custody and to support them extramurally (Nitsche, 1931) but then in the 1930s, in the context of increasing ideologization in the interest of the collective (the "people") advocated the social control of the mentally ill and finally murdered them during the war.

After World War I Faltlhauser became a close associate of Gustav Kolb. With his conception of "open care" Kolb initiated an internationally recognized psychiatric reform. The conception of "open care" was based upon outpatient care and a social support network for the chronically mentally ill. As a senior staff member in the psychiatric hospital of Erlangen Faltlhauser also took over the position of a care physician ("Fürsorgearzt"). Finally he was one of the leading reform psychiatrists and in 1929 he became the director of the Kaufbeuren psychiatric hospital where he also established "open care." Together with Kolb and Hans Roemer[6] he published "Die offene Fürsorge in der Psychiatrie und ihren Grenzgebieten" ("Open Care in Psychiatry and its Related Areas") in 1927. Even in 1932, in his textbook of psychiatric care, he recommended the treatment of the chronically ill and rejected euthanasia measures. However, Faltlhauser pursued from the beginning the elimination of so-called "psychopaths":

> "...One of the most difficult questions of the treatment of psychopaths in open care is the question of marriage of psychopaths. It is not too much to assert that 80% of psychopaths marry a psychopath. It is the obligation of social care to prevent such an intended marriage as far as possible . . . (Because even) tireless information (is useless), perhaps the suggestion of incapacitation might be successful" (Roemer et al., 1927).

In contrast the current conception of social psychiatry, developed after World War II, is indeed also extramural but most notably oriented to the individual by helping the chronically mentally ill in a graded system of institutional aids to lead a more or less self-determined life in society[7].

My opinion is that partial conceptions of mental illness might indeed convey transiently less recognized aspects to the public awareness. However, the more selective they are, the more they let other aspects be forgotten, and the more they become dogmatic,

[6]Hans Roemer was a convinced representative of the eugenic prevention of mental illnesses (including forced sterilization) but – as opposed to Faltlhauser – rejected euthanasia by a clear memorandum and retired ahead of time 1940 as director of the psychiatric hospital Illenau when he saw that he was unable to stop the deportation of his patients (Roelcke, 1993, 2012).

[7]This indicates also a change of the meaning of terms with the times going, i.e., the term of social psychiatry comprises a cluster of fairly different conceptions. Nevertheless, all conceptions are at risk to become absolutized.

the more they can become dangerous for the individual ill person in practice.

This will be illustrated by three examples from the practice:

1. A young assistant in the psychiatric hospital, convinced of social psychiatry, refused to take over a patient with an acutely delirious state from surgery because he "was somatically ill." (At that time this seemed for me to be a special form of brainless psychiatry).
2. At the height of the cult movie "Family Life" young colleagues implicitly addressed reproaches to the parents, mainly those of patients with schizophrenia, of bearing the blame for the manifestation of the disease – although this of course elevated contratherapeutically the emotional level of tension in the family.
3. A student with schizophrenia, decompensated during her university examinations, developed a postpsychotic residual state that was not accepted by her young therapist. The therapist intensively urged the patient to participate in an active rehabilitation program, which the patient tried to avoid. Several weeks later the patient committed suicide outside the hospital. Presumably she felt overburdened by the program.

BIOLOGICAL PSYCHIATRY

The impressive improvement of the treatment of mentally ill that was made possible by the development of psychotropic drugs 60 years ago led the practice of drug treatment and research to the neurochemistry of the brain and the development of new drugs. For this focus on the brain and thereby on the biological foundations of mental illness the term "biological" psychiatry has become established.

However currently this term seems to be fading and to be substituted by the term "neuroscientific" after the possibility of gaining knowledge by the various neuroimaging measures developed during the last 30 years broadened research on the neuronal determinants of psychic functions and diseases so that psychiatry currently is neuroscientifically oriented. From a psychiatric viewpoint this research is disease-oriented brain research and its counterpart in practice is among others a differentiated drug treatment as well as neuropsychologically based methods of behavioral therapy.

Along with this objectifying, quantifying, disease-oriented view there is the risk that the patient's feeling of illness will fade into the background and be recognized only insufficiently, i.e., that the patient's experience of changes of his inner world as well as of his capacity to acting, his processing of his disease, his awareness of disease-conditioned disturbances of his relationship to his environment will pass from view. These different perspectives of disease versus feeling ill of the mentally ill person will be illustrated by my own experience:

- In the 1950s in a large outpatient clinic for people with epilepsy I tried to relieve patients from their seizures. My emphasis was on the disease. Side effects of the necessary drug treatment had to be accepted by the patient. With increasing experience my view widened from the disease to the feeling of being ill on the part of the patient. Today the patient is not only informed about the side effects of the treatment but, as appropriate, it will be decided together with the patient for which therapy objective he is willing to accept which burdens [this development was recently named "preference diagnosis" (Mulley et al., 2012)].

My opinion: The objectifying narrowing view of the disease leads to the disadvantage of an empathetic assessment of the patient's feeling of being ill. This becomes comprehensible when biomedical research publications almost exclusively speak about research *on* patients, whereas it should be termed research *with* patients, because the individual subject should be invited to participate in a research intervention but it should not be researched on him as an object.

PSYCHOTHERAPY

During the past decades the concept of psychoanalytic psychotherapy has been joined or even opposed by many other conceptions of psychotherapy. This has led in practice – due to "the closeness of this field of the art of healing to a space free of sanctions" (Ritschl, 1989) – to the increase of private modifications of conceptions and finally – perhaps according to the contemporary "postmodern" credo of some philosophers that "everything goes" (Feyerabend, 1983) – in a few cases resulted in deadly quackery. Perhaps as a counter-reaction some therapists tried to assert rigorously the conception of the method that they performed, i.e., to keep it "pure." Also the standardization of therapy manuals has been promoted.

My opinion is that not only the training in a specific method of psychotherapy and the indication for a specific state of mental disease determine the choice of a certain treatment in an actual case but also the normative content of the human experience of the therapist.

This can be seen in the establishment of therapy objectives – not only in psychodynamic but also in other psychotherapies such as, e.g., in deconditioning (sometimes even manipulating) techniques of behavioral therapy. "Is it about adaptation, optimal adaptation to the social environment, such as if the meaning of human life is classification or relationship to others? Or is the objective the maximal evolvement of the patient's potential such as if the criteria of a healthy existence are only inside the single individual?" (Ritschl, 1989).

However if psychotherapy – in this case the psychoanalytic kind as recently published by the philosopher Paul Biegler (2011) – is declared dogmatically as the ethical imperative in order to support the autonomy of depressive patients, then danger threatens. This became apparent when a patient with depression sued his psychotherapist for withholding him antidepressant drugs (Klerman, 1990), or a patient with schizophrenia asked in court for compensation because for years his psychotherapist had refused to treat him with drugs.

AN INTEGRATIVE CONCEPTION

Against the narrowing of conceptions that depict only partial aspects of mental illness and whose ideological radicalization during the past century had disastrous consequences for the mentally ill, most psychiatrists today follow a bio-psycho-social concept. This concept is reasoned in the experience of psychiatrists who

see in their everyday practice how much the mental state of their patients is influenced by interaction with their social environment as well as by earlier impressions in the microsocial space of the family. These latter ones may be of developmental psychological or of biological-genetical nature.

In the 1960s the psychiatrist Hans-Joachim Bochnik introduced a graphical scheme in his Frankfurt University Hospital – the "Bochnik Triangle" – in which the grade of expression of somatic, psychic, and social influence on the current state of disease was to be mirrored (Bochnik et al., 1967). Thus the assistants had to turn their attention systematically toward all of these determining dimensions of being mentally ill. The conception became internationally well-known as the bio-psycho-social model by the Science-publication of Engel (1977). However, it has been criticized for being arbitrary and vague with regard to causal explanations of mental and behavioral disorders, and no rule exists for weighing the relevance of the various conceptions (Ghaemi, 2009). Therefore, the arbitrariness of the model should be reduced by focusing on scientifically proven concepts, to be tested for empirical evidence in the individual case, and taken as provisional in a longitudinal perspective (Brendel, 2003). Nevertheless, it may be helpful in two directions: at first, it should be used as a didactic tool to direct the psychiatrist toward a systematic exploration of the patient's intern and extern context because the knowledge gained by this procedure may be helpful to guide the patient in overcoming his being ill; such preliminary trials to assess the complex texture of the disease at the clinical macro-level can deepened today at the micro-level: at second, the model encourages the psychiatrist to open his mind for real interdisciplinary considerations of the causal interchange between social, psychic, and neurobiological determinants of mental disorders which is hoped to be developed in the future as some stimulating findings indicate.

Thus, e.g., the brain researcher Florian Holsboer recently reported an example for such a gene-environment interaction. Holsboer et al could confirm the hypothesis that defined variations of the gene for the protein FKBP5, which modulates the function of the corticoid receptor, the most important receptor of the hormonal stress system, makes the bearer particularly sensitive for trauma expositions, which initiate a depression (Zimmermann et al., 2011).

This example, as one of to-date numerous others (Caspi et al., 2002; Haddad and Meyer-Lindenberg, 2012), substantiates the hope of recognizing genetic risk profiles for interactions with specific environmental somatic, psychic, and social factors for individuals and thereby to approach the objective of individualized medicine.

However, if this individualization is focused on its somatic base, i.e., to be content with the question which drug is the right one for which patient, and if by that loses sight of the psychic and social context, the chance will be lost to move from individualizing to personalizing medicine, medicine that recognizes the patient as a person in all of his relationships and does justice for him.

My final opinion concludes that disease conceptions are effective as background coordinates for the practicing psychiatrist. They help him to organize the complex diversity of phenomena: they are instrumental in nature. Even partial conceptions can help to provide particular awareness for a less considered but important aspect. However they become dangerous if they are not understood only as instruments to be used only transiently but are made ideologically absolute under the influence of dominating ideas in societal context, if they increasingly shut out important parts of reality, and if they instrumentalize the ill person – whether for scientific, or for political, or for personal purposes.

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GENERAL COMMENTARY
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The biopsychosocial model between biologism and arbitrariness. A Commentary to H. Helmchen

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A commentary on

Different conceptions of mental illness: consequences for the association with patients
by Helmchen, H. (2013). Front. Psychol. 4:269. doi: 10.3389/fpsyg.2013.00269

In his article "Different conceptions of mental illness: consequences for the association with patients" Helmchen rightly cautions against any kind of dogmatism in psychiatry, regardless of whether it is a social, a psychological or a biological one. Instead, he favors the biopsychosocial model as a remedy for "the narrowing of conceptions that depict only partial aspects of mental illness" (Helmchen, 2013, p. 3). The main criticism of this model is traditionally that it "borders on anarchy" because one can emphasize the "bio" if one wishes, or the "psycho" [. . .], or the "social." There is "no rationale why one heads in one direction or the other" (Ghaemi, 2009, p. 3). Against this alleged arbitrariness and vagueness of the integrative model Helmchen recommends basing it "on scientifically proven concepts" (Helmchen, 2013, p. 4). Yet, it is not quite clear in which relation the three elements of the integrative model should stand and what its proposed grounding on scientifically proven concepts amounts to. I assume that the biopsychosocial model either has to be based on *biological* facts or else it will remain arbitrary. But if it is based on biological facts—even if not exclusively—it will probably be charged with "biologism" in just the same way as current accounts of biological psychiatry.

It is certainly true that a dogmatic overemphasis of the physiological side of the disorder-coin is ill-advised and in all likelihood to the disadvantage of the patient. Whether biological theories of the mind are in fact utterly brain-focused is, however, a point of contention. Admittedly, there are indeed voices that urge the concept of mental illness to be replaced by an account of brain disease (Bickle, 2006; Akil et al., 2010; Holsboer, 2010; White et al., 2012). But the vast majority of biological psychiatrists *does* try to *understand* the patient's personal situation. Even a hardboiled reductionist cannot avoid asking the respective patient about what she "feels." The reason is simply that it is up to now impossible to read off the brain whether someone feels depressed or not, whether she has delusions or not. The causes of a mental illness on the one hand and the symptoms on the other are to be found on different levels. This points to the distinction between "explaining" and "understanding" Jaspers is so often cited with and which even a biological psychiatrist cannot—and will not—ignore. In his *General Psychopathology* Jaspers explains:

> "The units of phenomenology (e.g., hallucinations, modes of perception, etc.) *are explained by bodily events.* Complex meaningful connections in their turn are considered as units (e.g., a manic syndrome plus all its contents can be regarded as the *effect of a cerebral process* or of some emotional trauma such as the death of an intimate.") (Jaspers, 1963, p. 305, my italics).

Biological psychiatry is sometimes regarded as nothing but an ideology (Berger, 2001; McLaren, 2010; at least implicitly also Cohen, 1993). On a closer look, things are not that simple. In actual fact, when Helmchen defends the integrative model as a "didactic tool" (Helmchen, 2013, p. 4), this comes quite near even to Bickle who declares within his "ruthless reductionism" that "[h]euristically, higher level investigations and explanations are essential to neuroscience's development" (Bickle, 2006, p. 428). Similarly, Insel and Quirion who demand "that mental disorders be understood and treated as brain disorders" emphasize "the need for a sophisticated understanding of interpersonal relationships along with the use of evidence-based, nonpharmacological treatments" (Insel and Quirion, 2005, p. 2223). Last but not least, Kandel—who, too, is usually regarded a radical reductionist—explains that it "would be unfortunate, even tragic, if the rich insights that have come from psychoanalysis were to be lost in the rapprochement between psychiatry and the biological sciences" (Kandel, 1998, p. 467).

What is, then, the difference between the biological and the biopsychosocial model as Helmchen conceives it? According to the first, mental illness as a phenomenon can be understood on the level of the patient's experiences and be *explained* biologically. According to the latter, the psychological and social levels are essential for an adequate understanding of mental illness, but both should to be based on scientific proven concepts. The much criticized biological psychiatry seems not to be very different from the integrative, scientifically based model.

A critic may object that the searched-for foundation of the biopsychosocial model of mental illness does not at all need to be a biological one. What about Psychology? Isn't it a science, too, with proven concepts? The answer to this objection is—in a nutshell—2-fold. Either psychology is a science or it is not. If it is, it is unavoidable to bring it into accordance

with biology, which undoubtedly is a science as well. In cases of theoretical conflict we would need a criterion to decide which concept (or theory, or law) should be given priority. The crucial question, then, is whether the "bio," the "psycho," or the "social" should be the maestro in the orchestra of concepts.

If, on the other hand, psychology is not a science, the psychologist will simply not be able to provide the "*scientifically* proven concepts" which are so necessary to mitigate the lurking arbitrariness of the biopsychosocial model of mental illness. It is one thing to use a conception as an auxiliary means for an adequate understanding of something and quite another to make use of it as a scientific foundation.

To sum up, the biopsychosocial model of mental illness is valuable as a reminder that there is more to mental illness than brain functions. Seen as a theory, it will either be based on biology and meet similar trouble as the so called biologism in psychiatry, or else it will indeed be vague and border on anarchy.

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"Early Psychosis" as a mirror of biologist controversies in post-war German, Anglo-Saxon, and Soviet Psychiatry†

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The English term "early psychosis" was coined in the 1930s to refer to feelings of irritability, loss of concentration, hypochondriac ideas, moodiness, and lassitude that were seen to precede the onset of clear-cut hallucinations and delusions. The history of thinking about "early psychosis" under names such as "latent," "masked," "mild," "simple" or "sluggish" schizophrenia before World War II and afterwards on the different sides of the Wall and the Iron Curtain reveals "early psychosis" as a mirror of quite aged international biologist controversies that are still alive today and to the same extent as they are misunderstood, are influential in their implications in today's psychiatry.

Keywords: latent schizophrenia, sluggish schizophrenia, prodrome, vulnerability, early psychosis, subjective symptoms, basic symptoms

The fifth revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM) for the first time includes a category named "*attenuated psychosis syndrome*" as a condition for further studies (Yung et al., 2012). What had been proposed at the beginning of the revisions, however, was the introduction of "*psychosis risk syndrome*" as a new diagnosis to describe a condition with a recent onset of modest, psychotic-like symptoms with clinically relevant distress that would indicate a significantly increased risk of conversion to schizophrenia. Vigorous debates among international psychiatrists finally came to the conclusion that it might be premature to recommend a new category primarily based on future "risk" (Yung et al., 2010).

The departing point of these debates seemed to be a dissent about the meaning of "risk for schizophrenia." It is not only the way that risk criteria differed within a "near Babylonian speech confusion" about terms as "*prodrome,*" "*early psychosis,*" "*at risk mental state,*" "*high and ultrahigh risk*" (Schultze-Lutter et al., 2011, 2012), there was—and probably still is—also a confusion about the significance of "risk for schizophrenia" tout court. Although the DSM does not claim to pinpoint disease entities, the proposed formulation of a new diagnosis, "*psychosis risk syndrome*," did seem to implicate the existence of a disease or illness. But is a "risk for a disease" already a disease? Yes, it is, was claimed by proponents advocating the introduction of a new psychosis spectrum disorder in DSM-5 under the words "Probably at-risk, but certainly ill" (Ruhrmann et al., 2010); no, it isn't, was claimed by others. Interestingly, this up-to-date controversy that is still going on for the eleventh revision of the International Classification of Diseases (ICD) appears like a reflection of bygone biologist controversies in Post-War German, Anglo-Saxon and Soviet Psychiatry.

The English term "*early psychosis*" entered the stage of classical psychiatry in 1938, first of all with reference to "*early diagnosis*." This new interest in diagnosing schizophrenia early was revolutionary in a time that had thought of schizophrenia as a disease process that per definition would lead into premature dementia: dementia praecox. The treatments by hypoglycaemia and convulsions that had been introduced since 1934 by Manfred Sakel and Ladislas Meduna caused to totter the concept of incurability and "brought into the foreground the necessity for early diagnosis," as the most promising ameliorations were obtained in "early cases" (Mayer-Gross, 1938). Even conceptually quite a gap, then psychiatrists needed just a little step from the idea of *early detection* of a disease to the idea that the disease itself might have an *early phase* or even be a specific *early form* of a chronic disease: *early schizophrenia* or more general, *early psychosis* (Cameron, 1938a,b).

EARLY SCHIZOPHRENIA OR *PRODROMES* IN CLASSICAL EUROPEAN PSYCHIATRY

Subtle changes in mood and personality that gained significance retrospectively had of course always been part of the case descriptions in the classic textbooks on dementia praecox and schizophrenia. In Emil Kraepelin's description of dementia praecox in 1893, a potpourri of *initial* symptoms, especially of somatic kind, was given:

> Usually the psychosis begins with symptoms of general malaise and uneasiness, headaches, ear noises, dizziness, disagreeable feelings in different parts of the body, insomnia and poor appetite. The sick persons become shy, withdrawn into themselves, downcast, anxious, stop working, express vague concerns especially with hypochondriac contents (Kraepelin, 1893, 439).

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Concerning the talk about the "*early*" or "*initial*" symptoms, Eugen Bleuler felt he needed to add some words in order to prevent misunderstandings concerning the meaning of "*early symptoms*":

> When speaking of initial symptoms of schizophrenia we have to restrain us to the first symptoms that were noticed; too often we just don't know the symptoms that really appeared first.

In the corresponding footnote he went on to explain:

> We do not speak of "prodromes." We might differentiate prodromes of a seizure and inter-current signs from the full-blown seizure, if we like—prodromes of a disease, however, I am not able to imagine. What are named in this way are the first symptoms that we are not able to interpret in the right way (Bleuler, 1911, 206).

Speaking of "*prodromes*" was, however, quite common in European Psychiatry. Years before Bleuler proposed his concept of "*schizophrenia*" as a substitute for Kraepelin's "*dementia praecox*," notions such as "*depressive prodromes*" or "*prodromal pseudoneurasthenia*" had already been discussed in the continental psychiatric literature (Pascal, 1906, 1907).

No matter what words were used, these quotations clearly show that the feelings of irritability, loss of concentration, hypochondriac ideas, headaches, moodiness, and lassitude that were seen to precede the onset of clear-cut hallucinations and delusions since the earliest descriptions of dementia praecox or schizophrenia were not conceptualized as "risk" signs for the occurrence of a disease, but they were seen as already manifesting the disease *process:* However, "what this schizophrenic process consists in, we don't know," admitted Bleuler in 1911, even if there were clear findings of mild brain atrophy and specific histological changes in severe cases. Bleuler continued:

> The question if there might be a specific brain disposition to schizophrenia and how it would manifest has still not been addressed at all (Bleuler, 1911, 376f).

BLEULER'S *LATENT SCHIZOPHRENIA*

Bleuler's favorite explication of the pathomechanism of schizophrenia was the idea of an infection or autoimmune process, which may manifest in a chronic or acute manner and may even stay *latent* over a longer period (Bleuler, 1911, 376f). Not surprisingly, *latent schizophrenia* was considered a very widespread and underdiagnosed phase of schizophrenia with fuzzy boundaries especially to *schizophrenia simplex* at first extensively described with patient examples by Otto Diem in 1903 (Diem, 1903). By separating this form of schizophrenia from *hebephrenia*, as opposed to Kraepelin, Bleuler gave *schizophrenia simplex* and *latent schizophrenia* the central exemplary position in his theory of "schizophrenia," demonstrating his advocated dichotomy between *fundamental* symptoms (e.g., cognitive or emotional blunting) and *accessory* symptoms (e.g., hallucinations or delusions) (Bleuler, 1911, 194). Consequently, seemingly uncharacteristic symptoms such as increased distraction, forgetfulness, reduced emotional reactivity or anhedony and avolition characterized Bleulerian core schizophrenia and were therefore no risk and no prodrome: "A latent schizophrenia already is a psychosis" (Bleuler, 1917, 29).

The problem of drawing the line between character and disease was answered with resolute words:

> As it is clear that many cases of schizophrenia go back into youth and as many cases impress as simply intensification of the existing character, it seems probable to me that these autistic abnormalities in character are the first symptoms themselves and not only an expression of the disposition (Bleuler, 1911, 206).

Nine years later, however, in the 3rd edition of his textbook, Bleuler used Kretschmer's term "*schizoid*" for the first time in order to admit the unresolved question of the qualitative boundaries or only quantitative differences between constitution or predisposition and disease:

> As from which level of anomaly on a person should be classified solely as a "schizoid" psychopath or else as schizophrenic and mentally ill, is still not possible to define at all (Bleuler, 1920, 325).

To what extent these personality peculiarities already "are the young disease or solely expression of the predisposition," was questioned by Bleuler especially from the point of view of genetics: of course, one must differentiate between hereditary and phenomenological visible features of schizophrenia, "Erbschizose" and "Sichtschizose," because the hereditary features are linked to the visible ones "by a long causal chain complicated probably by the influence of some inner and external factors" (Bleuler, 1917, 31). Also "accompanying psychic predispositions, that *per se* have nothing to do with the gene of the disease, might contribute"; Bleuler here thinks of "a certain sensitivity that does not only appertain to future schizophrenics" (Bleuler, 1917, 32).

After the collapse of Nazism into "euthanasia" and World War II, West-German Psychiatry turned its back on the genetic theory of schizophrenia, much more than Bleuler—in spite of his criticism on the methodology of Ernst Rüdin's studies—would have advocated.

THE END OF *CLASSICAL BIOLOGICAL* PSYCHIATRY

Already Kurt Schneider recommended his pragmatic symptomatologic classification oriented on his first (e.g., auditory hallucinations and delusions of control) and second rank symptoms, because he had capitulated in face of the indecisive results of the biologic research in schizophrenia. Bleuler's schizophrenia concept was simply but silently put aside.

With the retirement of Schneider from the chief position of the psychiatric university clinic in Heidelberg in 1955, "classical psychiatry" was said by Walter von Baeyer, his successor, "to have come to its end; the future was for existential analysis ("Daseinsanalyse") in the sense of Heidegger, Husserl and Binswanger" (Huber, 2009, 70). In other places of European Psychiatry, the influence of psychoanalysis had already departed

large parts of thinking about "early psychosis" from Bleuler's classical biological views.

Even if early schizophrenia was still in the spotlight, there was a "sort of panic" in West-German Psychiatry a year before the Wall was build: there was an anxious suspicion that everything concerning schizophrenia research had been seen and done in a wrong way (Kraemer, 1960). Almost everything of the biologic view on schizophrenia and the diagnostic methods used, was questioned as being wrong. Accordingly, in the 60s, West-German Psychiatry started to see paranoid schizophrenia and early schizophrenia in an anthropologic light and to explain them by the individual situation in the life of the concerned person: a personal *"failure on the road of life"* (Zutt and Kuhlenkampff, 1958). Similarly, as in the classic psychiatric schools situated in the west of the new Iron Curtain in Europe, the "schizophrenic person" gained center stage (Wyrsch, 1949). "Schizophrenic" characteristics were explained in the light of Heidegger's existential philosophy: "*eccentricity, crankiness, mannerism as three forms of failed existence*" (Binswanger, 1956, "Drei Formen missglückten Daseins. Verstiegenheit, Verschrobenheit, Manieriertheit"). Symptoms of beginning schizophrenia were reformulated in Heidegger's language as *"disclosure, dissolution and overwhelming as forms of loss of the existential position in life"* (Kulenkampff, 1955, "Entbergung, Entgrenzung und Überwältigung—als Weisen des Standverlustes"). At the institutional level, the separation of the departments for neurology and psychiatry in the university clinics was pressed ahead and the (West-)German Council of Science and Humanities insisted in 1960 on the implementation of professorships for psychotherapy.

Klaus Conrad, who had published a phenomenological analysis of the steps of symptom progression at the onset of schizophrenia entitled "*The Beginning Schizophrenia*" (Conrad, 1958) in 1958, criticized the diagnostic practice of the time: what traditionally had been classified as "beginning schizophrenic phase" and already had been challenged by Kretschmer's "*schizoid*"-concept into "a sensitive delusion of reference on the base of a schizoid constitution," that would nowadays be seen, "in Frankfurt as a consequence of a deranged existential order of being, as a form of existential failure in the pursuit of life" (Conrad, 1959, 489). As a matter of fact and in contrast to Karl Leonhard who migrated to the GDR and took up the chair of the Charité-Nervenklinik in East-Berlin in 1957, Jürg Zutt and Caspar Kulenkampff abandoned the classic biological Frankfurt-Kleist-Wernicke school. But also the Heidelberg school was increasingly marked by the wish to explain the psychopathology out of individual and family psychodynamics, as is evident in the academic writing of senior physicians of the Heidelberg clinic, for example Heinz Häfner's "Existential Analytical Investigations in the Structure and Course of Psychopaths" (Häfner, 1961), or Karl-Peter Kisker's study results *"Comparative Situation Analysis of Beginning Schizophrenias and Reactive Maldevelopment in Adolescents"* (Kisker and Strötzel, 1961/62).

Not by chance, *"adolescent crisis"* or *"maturation crisis"* became the main differential diagnoses of beginning schizophrenia (Kulenkampff, 1964; Feldmann, 1967). These concepts together with the idea of "*existential failure*" reflect a way of thinking about "early psychosis" that American psychiatry had already chosen before World War II.

MENTAL HYGIENE AND THE "*SCHIZOPHRENIC REACTION*" IN THE DSM

Contrary to the situation in Europe, American Psychiatry generally developed independently from neurology and was decisively shaped by its founder Adolph Meyer and his psychobiological school. In opposition to the classical pre-war European view that granted psychological factors not much more than the role of unveiling the *latent biological* basic disorder, Meyer's school explained all mental diseases as "*psychological reaction types*" (Muncie, 1935). The early American favor for early detection and prevention grew exactly out of this psychological perspective: under the assumption that all mental diseases can be explained by psychological, environmental causes, it was just a logical reasoning that they might be impeded or nipped in the bud if their causes would be detected early enough and neutralized. Meyer's Mental Hygiene Movement was based on this argument (Kalinowsky, 1955).

The emigration of European psychoanalysts to America during National Socialism led to the integration of Freud's theory of intrapsychic conflict into the environmentally oriented Mental Hygiene Movement: a broadly defined psychosocial model was born that conceptualized even schizophrenia as reducible to one basic psychosocial process: Karl Menninger's *"failure of the suffering individual to adapt to his or her environment"* (Wilson, 1993, 400). Only the intensity of the trauma determined if the reaction would be of a neurotic or of a psychotic kind. Symptoms were seen in the psychodynamic light of "meaning." As a result, the frontiers between character eccentricities and schizophrenia vanished on psychological grounds. Schizophrenia was just a more severe psychological maladjustment than other personality or neurotic abnormalities; it was no longer a genetic disease but rather a psychosocial reaction, as expressed in the revision of the Army nomenclature under the leadership of Menninger, the first DSM published in 1951, and its revealing term: "*schizophrenic reaction*."

The psychodynamic or even psychoanalytic interpretation of *early* schizophrenia was already evident in Harry Stack Sullivan's lecture entitled "*The onset of schizophrenia,*" held on the occasion of the joint meeting of the American Psychiatric Association (APA) and the American Psychopathological Association in 1926 (Sullivan, 1927), as it is in the famous article on the *"Diagnostic evaluation of early schizophrenia"* written by Phillip Polatin and Paul Hoch in 1947 (Polatin and Hoch, 1947). The introduction of the term "*ambulatory schizophrenia*" by Gregory Zilboorg in 1941, *"pseudoneurotic schizophrenia"* by Hoch and Polatin in 1949 and the interpretation of "*Borderline States*' by Robert Knight in 1953, continued to foster this psychodynamic view on *early* and *mild* psychosis (Zilboorg, 1941; Hoch and Polatin, 1949; Knight, 1953).

Granted, three of the first articles published in English on early schizophrenia still had a classical medical model of the condition, but the articles were written by a German psychiatrist who had immigrated to London (Mayer-Gross, 1938) and by a Scottish psychiatrist who trained under the successor of Bleuler at the famous Burghoelzi Clinic in Switzerland during publication year (Cameron, 1938a,b). Even if classical views were still published in Anglo-Saxon psychiatry, their impact on thinking about "early psychosis" was almost non-existent in the years that

followed World War II—just as the results of the clinical study "The Genetics of Schizophrenia" of another German refugee from National Socialism (Kallmann, 1938).

During the 1960s, the view of mental disorders as non-biological psychosocial problems became the source of anti-psychiatric arguments: "if conceived of psychosocially, psychiatric illness is not the province of medicine because psychiatric problems are not truly medical, but social, political, and legal" (Wilson, 1993, 402); mental illness was a myth and psychiatric labels arbitrary designations (Szaz, 1961). The revision of the DSM, published in 1968 by the APA as *DSM-II*, consequently dropped the term "*reaction*" even if psychodynamic views largely prevailed besides a re-appropriation of classical concepts (American Psychiatric Association, 1968). Orienting itself on the 8th revision of the ICD that listed—in classical Bleulerian tradition—as subtypes of schizophrenia "*Schizophrenia simplex*" and "*latent schizophrenia*" (World Health Organisation, 1965), the APA also consented on a "*simple type*" and a "*latent type*" of schizophrenia. In explaining "*latent schizophrenia*," however, it was added that—among "incipient" and "pre-psychotic"—"pseudo-neurotic, pseudo-psychopathic, or borderline-schizophrenia are categorized here"—which clearly were of psychodynamic origin (American Psychiatric Association, 1968).

SLUGGISH SCHIZOPHRENIA IN SOVIET PSYCHIATRY

Soviet Psychiatry strictly rejected western anthropological interpretations of mental illness denunciating these views "as a sign of a severe crisis in capitalistic countries' psychiatry" (Sternberg, 1964).

Characteristic for Soviet psychiatry was not only its clear biological orientation, but also especially its preoccupation with Bleuler's "latent schizophrenia." Clinical research started as early as 1924 at the *Moscow Institute for Neuropsychiatric Prophylaxis* and centered on the questions of "*mild*," "*attenuated*" or "*masked*" schizophrenia. However, Bleuler was criticized for using the word "*latent*" in a context where schizophrenia was already manifest, but in a mild, non-psychotic form, just as he himself had very well tried to explain, but was easily misread by the unclear signification of the word "*latent.*" As a consequence, *mild* or *sluggish* schizophrenia was assumed to consist of a sort of *attenuated organic*, perhaps toxic, *process with slow progression* (Kameneva, 1935). The director of the Institute for Neuropsychiatric Prophylaxis of the time, L. M. Rosenstein, himself pointed out that the elaboration of the concept "*sluggish schizophrenia*" was conditioned by the politically enforced restructuring of the medical psychiatric facilities with closure of private consultations and a concentration on polyclinic centers. "The moments that mostly determine the development of scientific categories of our discipline are the current historically-given forms of our psychiatric practice," wrote Rosenstein in his report about the new achievements concerning "early psychosis" since the foundation of the Soviet Union (SU) in 1922. The most recent form of psychiatric *practice*, "namely the set-up of psychiatric welfare units called 'dispensaries' " where "psychiatrists are facing a material, that usually counts as 'healthy' or 'nervous' and will have to do prophylactic work on it," is feeding back on the theoretical concepts (Rosenstein, 1933, 299f). The parallel of the *dispensaries* to the institutional development inside the American Mental Hygiene Movement is quite interesting due to the different if not opposing theoretical foundations. In Europe, attention had been paid to the mild forms of schizophrenia until the end of World War II after the classic description of "Heboidophrenia" by Karl Ludwig Kahlbaum and "Dementia simplex" by Diem, especially in the context of the growing acceptance of another psychiatric practice: *psychotherapy* (Kronfeld, 1928; Wyrsch, 1945).

The focus of clinical interest on bland, mild or sluggish schizophrenias was to shape the whole Soviet theory of schizophrenia that was seen as a life-long process of a genetically determined disease (Sternberg, 1973). Classification remained oriented on the course or progressive evolution of symptoms seen in the "unitary psychosis layer model" formulated by Andrej Sneshnewski (Piatnitski et al., 1998).

GERD HUBER AND GISELA GROSS AS ADVOCATES OF THE CLASSICAL VIEWS IN WEST GERMANY

Few West-German psychiatrists have been invited for lectures in the German Democratic Republic (GDR). Thanks to an invitation of the (East-)Berlin Society for Psychiatry and Neurology at the Humboldt-University, Gerd Huber was among them to present his pneumencephalographic studies in schizophrenia in the year 1958 (Dietrich, 1958). The university psychiatric clinic of Heidelberg with its growing focus on anthropological thinking had just generously allowed Huber to finish his compilation of pneumencephalographies taken at the onset of schizophrenia that he had started in 1950 in order to correlate psychopathological symptoms with localized brain atrophy (Huber, 1957a). The description of "*coenaesthetic schizophrenia*" that Huber published in the same year (Huber, 1957b), was sparely appreciated in West-German Psychiatry, but was received with emphatic approval in the SU and GDR. It is no surprise then that the clinical "differentiation of hypochondriac syndromes," was seen "as currently one of the most difficult and urging psychiatric problems" in Soviet psychiatry. Consequently, Huber's work was applauded as an important contribution to the organic base of "*hypochondriac schizophrenia*" as already described by the Russian psychiatrist G. A. Rothstein (Sternberg, 1964).

As the leading physician of the psychiatric outpatient department of the Heidelberg university psychiatry, Huber had been able to conduct his barely connived follow-up examinations until 1962 for the construction of his "Heidelberg Checklist of Basic Symptoms" (Huber, 1962) that is nowadays well known under the name "Bonn Scale for the Assessment of Basic Symptoms" (*BSAB*) and is used as "*an instrument for the assessment of schizophrenia proneness*" (Gross et al., 1987; Klosterkötter et al., 1997).

"Barely connived" was Huber's psychopathologic assessment of subjective complaints of patients with early schizophrenia because of the biological idea on which they were founded. The reason why Huber was interested in subjective experience and feelings was solely because he thought that they would shed light on the organic base of schizophrenia. The *subjective symptoms in early psychosis* were credited to lead directly to the *biologic* "*fundamental*"—"*primary*"—*or* "*basic*"—*symptoms.*

The subjective experience of subtle cognitive deficits and changed self-feeling were originally identified by Huber as "*pure defect*" in chronic schizophrenia after the psychotic symptoms had disappeared, but then recognized in the prodromal phase. The notion of "*basic symptoms*" was used to make clear that these subtle feelings are the core symptoms, the "most primary symptoms" of schizophrenia in the sense of their direct organic origin (Huber, 1966). Even though some of the contents of paranoid ideation in full-blown schizophrenia might be explicable by the individual personal situation of the affected person, what is seen in early psychosis is the direct expression of the organic origin of schizophrenia according to Huber and not analyzable in the frame of existential psychiatry. Likewise, Klaus Conrad thought it was possible to extract an analysis of the different stages of "beginning schizophrenia" out of the examination of uniformed soldiers realized during World War II: the question was not about individual conflicts and situations but about *the neuropsychological laws of symptom progression* at the onset of schizophrenia (Conrad, 1958). Evidence for the neurobiological determination of the different stages of the changing experience in early schizophrenia would also have been searched for by Conrad with biological means, just as he already had tried to find the genetics of epilepsy or schizoid constitution, if he had not died in the year of the construction of the wall before assuming the directory of the Max Planck Institute for Psychiatry in Munich.

Huber, his lifelong co-worker Gisela Gross and sympathizing psychologists nevertheless arrived at pinpointing "basic" symptoms even in "*the failure state of latent schizophrenia*" (Blankenburg, 1968), a denomination that might associate an anthropological psychodynamic account in Menninger's tradition as "failure to adapt to personal life challenges." On the contrary, the "*juvenile-asthenic failure-syndromes,*" as Huber called "*the failure states of early psychosis,*" were traced back to an organic base (Glatzel and Huber, 1968). This way, a seemingly uncharacteristic symptomatology was conceptualized as "abortive, latent or masked schizophrenia" (Gross et al., 1982). The number of colleagues who sympathized with this view in West Germany might be counted on one hand relying on the BRD-psychiatrists who followed Huber's invitation to the *Weißenauer Symposien*. The first symposium, which still took place at the psychiatric hospital "Weißenau" in 1971 (at this time under the directory of Huber before he finally found refuge in Bonn and the Weißenauer Symposion with him), was not accidentally dedicated to the etiology of schizophrenia, and defined a clear biologic direction of future research (Huber, 1971). The extent to which this alignment was rejected as outdated and obsolete in the 70s in West-Germany is mirrored in the blatant opposition of the audience that Huber encountered during his lecture on schizophrenia on the occasion of his application for the directory of the Heidelberg psychiatric university clinic in 1972: the audience wove a banner with the words: "Huber, evil excrescence of bourgeois psychiatry" (Huber, 1996, 237).

THE ROLE OF PSYCHOLOGY

Perhaps surprisingly, it was the practice of psychotherapy of schizophrenia that finally led to a revival of the medical model of "early psychosis" thanks to a newly flourishing branch of psychology called "experimental psychology."

Clinical psychologists with an originally psychoanalytic training began to recognize that cognitive deficits of patients with schizophrenia impeded psychotherapy on large grounds. This psychotherapeutic approach finally paved the way to a clinical research in psychology that tried to understand the mechanisms of the observed cognitive deficits in schizophrenia by applying the techniques of experimental psychology (Chapman et al., 1959). One of the very first programs of this kind was situated in Glasgow and headed by Arthur McGie, the Principal Psychologist at Royal DundeeLiff Hospital and honorary professor at the Department of Psychiatry at St Andrews University. As early as 1961, McGhie and a young psychiatrist, named James Chapman, published their observations on specific "*disorders of attention and perception in early schizophrenia*" (McGhie and Chapman, 1961). Chapman gained his MD with a thesis entitled "*On the early diagnosis of schizophrenia*" in 1964 and his summary publication of his results in 1966 as "*The early symptoms of schizophrenia*" became the starting point for other psychologists all over the world to reconsider early psychosis on empirical and finally biological grounds as shown by the example of the German psychologist Lilo Süllwold and her *Frankfurt Complaint Questionnaire* that is the instrument most widely used in Europe for assessing subjective experience in schizophrenia (Chapman, 1964, 1966; Süllwold, 1977).

Initially employed for a research program about the family psychodynamics of pre-schizophrenic adolescents at the psychiatric university clinic in Heidelberg, she started to collect complaints of subjective cognitive deficits in these young patients. Already in her first presentations on the occasion of the *Weißenauer Symposien* in 1971 and 1973, Süllwold explicitly combined her phenomenological approach—for which she cites McGhie and Chapman—with a biological interpretation of the observed malfunctioning (Süllwold, 1971, 1973). The gradually developing *Frankfurt Complaint Questionnaire* aimed to enable a reliable early differential diagnosis of pre-psychotic schizophrenia in contrast to neurotic troubles (Süllwold, 1973), even if Süllwold, just as McGhie und Chapman, was not just interested in early diagnosis, but finally also in a reapplication of the findings for cognitive behavioral psychotherapy (Chapman and McGhie, 1963).

Interestingly, the Anglo-Saxon results of primary attention and perceptual deficits in schizophrenia matched with the Soviet experimental schizophrenia research of Poljakow (Poljakow, 1971) for example. In the first publication of her *Frankfurt Complaint Questionnaire*, Süllwold referred to Poljakow the same way she had already pointed out in her presentation of the very beginnings of her complaint list, that the experimental research on schizophrenia conducted by Anglo-Saxon clinical psychology, eventually accomplishes Kraeplin's demand and so tied in with the tradition of classic psychiatry (Süllwold, 1971, 37; Süllwold, 1977).

Actually, there was a remarkable intertwining on the subject of experimental psychological research on the perceptual and cognitive deficits in schizophrenia across the Iron Curtain. Frank Fish for example, a Scottish psychiatrist, summarized the newly developed neuropsychological test methods for his colleagues in

West-Germany (Fish, 1966) and was invited, on the other side of the Wall, by the East-German psychiatric journal, to present his own neuropsychological testing results in schizophrenia (Fish, 1965). Equally, papers about experimental psychology in their significance for the biological theory of schizophrenia and their basic symptoms were welcome in the East even if written by west-psychologists (Plaum, 1978). The transfer was clearly not achieved by psychology as such, but by psychology as a servant of biological psychiatry.

Especially the question of *subjective symptoms of beginning schizophrenia*, that Conrad had initially called to mind after World War II, as well as their neuropsychological, neurobiological explanation by Huber and Gross (Gross, 1969) and Süllwold (Süllwold, 1977) in West-Germany, McGhie and Chapman from the UK (Chapman, 1966), Dudek from Canada (Dudek, 1969), and Freedman and Chapman, USA (Freedman and Chapman, 1973), started to form a bridge over the Iron Curtain. Opposing the American psychiatric tradition of Menninger with its psychodynamic view on schizophrenia, Fish outlined his neurophysiologic theory via Conrad's phase-model of beginning schizophrenia (Fish, 1961).

The mission of psychology in this context (McGhie, Süllwold, Chapman and Freedman all of them were psychologists) was couched in the clearest possible terms by the American psychologist Paul Meehl in his lecture addressed to the American Psychological Association in 1962: "in the near future" psychology with its new experimental techniques will help "to establish that schizophrenia, while its content is learned, is fundamentally a neurological disease of genetic origin" (Meehl, 1962).

Meehl's taking side with genetics and his concept of "*schizotaxia*" as genetic foundation of the "*schizotype*" character, the last being only the compensated form of schizophrenia, as in clinically compensated cardiac or kidney disease, did not appeal much to the large parts of psychiatry and psychology that still held on to psychodynamics up to the end of the 70s (Meehl, 1989). The American psychiatrist Joseph Zubin together with the psychologist Bonnie Spring were to have greater success in bringing together the warring parties by integrating all available psychological, biological and social aspects into a recycled concept of "*vulnerability*" (Zubin and Spring, 1977). By no longer defining "vulnerability" as "*causa interna*" but as "the empirical *probability* that an individual will experience an episode" of schizophrenia, Zubin and Spring admitted any possibilities of how this inclination comes about: it may be of genetic origin, it may be caused by acquired etiological factors such as perinatal complications, substance abuse but also "just" by family stress. However, "vulnerability" is generally seen as meaning more than "probability" or "risk" because a causal claim is implicitly made concerning the enumerated factors.

LATENT SCHIZOPHRENIA AND *PRODROMAL SYMPTOMS* AFTER 1980

The overabundant labeling of schizophrenia due to psychodynamic presuppositions in American Psychiatry and due to its concept of "soft" or "sluggish schizophrenia" in Soviet Psychiatry had meanwhile come to light in 1973 with the publication of the first results of the *International Pilot Study of Schizophrenia* that was conducted by the World Health Organization since end of the 60s (World Health Organisation, 1973). With the desire to enhance diagnostic reliability and thus re-open possibilities for meaningful research, the APA decided on a 3rd revision of the DSM that was to be a non-theoretical purely descriptive manual with emphasis on the assessment of easily observable symptoms for objective measurement (American Psychiatric Association, 1980). Consequently, "*simple*" and "*latent*" *schizophrenia* disappeared; diagnosis of schizophrenia completely oriented itself toward Schneiderian first as well as second rank symptoms. On the other hand, non-psychotic, schizophrenia-like disorders were classified as "*schizoid*" or "*schizotype personality disorders.*"

However, the symptoms of simple and latent schizophrenia also found refuge under another label: the list of *prodromal* symptoms that enumerated eight mostly behavioral, observable, so-called "*negative symptoms*": "1, social isolation; 2, marked impairment in role functioning; 3, markedly peculiar behavior; 4, marked impairment in personal hygiene and grooming; 5, blunted, flat, or inappropriate affect; 6, digressive, vague, overelaborate, circumstantial, or metaphorical speech; 7, odd or bizarre ideation, or magical thinking and 8, unusual perceptual experiences." That list was added with "9, loss of energy" in the DSM-III-R in 1987.

In the SU, the classic concept of "*early psychosis*" in the sense of "mild" or "latent" schizophrenia lived on without any challenge due to the application of a self made classification system for mental disorders completely independent of DSM and ICD. Due to this system, developed at the Moscow Psychiatric Institute by Sneshnewski, a wide concept of "schizophrenia" remained in place that also encompassed the non-psychotic forms. The study of these "mild" forms of schizophrenia had remained a core theme of Soviet psychiatric research until end of the 80s. Many subtypes of mild schizophrenia have been differentiated, among them "*simplex-schizophrenia,*" "*hypochondriac schizophrenia*," "*hysteriform schizophrenia*," forms with predominant *depersonalization* or affective symptoms, "*anancastic schizophrenia*" or "*psychopathic like schizophrenia in childhood*" (Sneshnewski, 1977; Piatnitski et al., 1998).

These biological concepts of schizophrenia united psychiatrists across the Iron Curtain in such a way that enabled international symposia in the SU, as the "Biological and Genetical Aspects of Schizophrenia" symposium in 1973 that was jointly organized by the World Psychiatric Association and the Moscow Academy for Medical Sciences.

Nevertheless, in the course of the political misuse of psychiatry in the SU, the Soviet concept of "*sluggish schizophrenia*" was criticized concerning its possible misuse for political reasons (Merskey and Shafran, 1986).

Shortly after the fall of the Wall and before the end of the SU, two symposia took place in 1990 mirroring the lasting controversial position of the "early psychosis" concept: the presentation of the first prospective study on early schizophrenia on the occasion of the 8th Weißenauer Symposion in March 1990 and the Symposium "*Symptoms of schizophrenia that are not criteria of DSM*" at Annual Meeting of the APA in New York, May 1990.

The first prospective study on early diagnosis of schizophrenia has been initiated by Huber and Gross in 1970, was founded by the West-German Ministry for Research and Technology and was later on continued by Joachim Klosterkötter under the name of "*CologneEarlyRecognition-Study*" *(CER)* (Gross et al., 1992; Klosterkötter et al., 2001). At the 8th Weißenauer Symposion, the discussion that followed the two lectures presenting the very first results of the "*Basic-symptom oriented diagnostic of schizophrenic vulnerability*" (Gross et al., 1990; Klosterkötter et al., 1990) became a crossfire: due to the fact that the basic symptoms that served the description of the "prodrome" now sailed under the flag of Zubin's "vulnerability," the question arose if vulnerability really always already is a pathology and sign of a disease. If "vulnerability" equaled "prodrome," wouldn't this mean that the prodrome—and with it the initial phase of schizophrenia, would be present from birth on, if one credited genetics or perinatal trauma with a role in vulnerability? Accordingly, would the term "vulnerability" equal "*compensated*" or "*latent schizophrenia*"? Would "vulnerability for schizophrenia" already *be* schizophrenia? The oscillation of the conceptualization of the basic symptoms between *state* or *trait* markers was of course not entirely innocent for this ambiguity. Moreover, anticipating the objection that the basic symptoms that were used for the diagnosis of schizophrenic vulnerability had not proved to be specific for schizophrenia, Gisela Gross frankly declared that there would not exist any specific psychopathological phenomena at all in psychiatry—and thus made a comment in the direction of an unspecific vulnerability in the sense of a strong "unitary psychosis" model of mental disorder comparable to the Russian "layer-model," yet continued by arguing that the basic symptoms would not exist in personality or neurotic disorders. Thus she corrected herself to a sort of weak "unitary psychosis" model of affective and schizophrenia disorders. However, as a matter of fact, the work of the Bonn School on early diagnosis has been understood as if there would be a schizophrenia specific cognitive vulnerability that could be identified by the subtle psychopathological examination via the "Bonn Scale" and would enable early detection and early treatment (Klosterkötter et al., 2001). In any case, current formulations as "Diagnosing schizophrenia in the initial prodromal phase" make just too clear that "prodrome" is seen in a classical Bleulerian perspective as the initial state of schizophrenia and not as "risk/vulnerability" for schizophrenia (Klosterkötter et al., 2001).

At the APA symposium in 1990, Huber and Gross argued for their classic view on early psychosis, basic symptoms and prodromes. Nevertheless, the list of prodromal symptoms was dropped for the DSM-IV in 1994: without any alternative.

Even if the ICD-10 still knows of *schizophrenia simplex* (World Health Organisation, 1992), no criteria are given neither in the DSM-IV nor the ICD-10 to diagnose "prodromes" of schizophrenia or "latent schizophrenia". Under the strong promotion of the professional descendants of Huber and Gross, however, the DSM-5 has now introduced "*attenuated psychotic syndrome*" as a research category, which may well be seen—just as the originally proposed "*psychosis risk syndrome*"—as standing in the tradition of "*early psychosis*" or "*latent schizophrenia*" that the article has recalled.

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Comment on Lara Rzesnitzek (2013) "Early Psychosis" as a mirror of biologist controversies in post-war German, Anglo-Saxon, and Soviet Psychiatry

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A mental illness with severe disturbances of subjective experiences and behavior with a progressive course, due to the onset of cognitive deterioration during the second and third decade of life, was described more than a century ago as dementia praecox (Kraepelin, 1896). In 1911 the diversity of existing marked symptoms led to the suggestion of a group of mental disorders, summarized as "Dementia praecox or group of schizophrenias" (Bleuler, 1911). Under the term schizophrenia various core symptoms of the diagnosis and/or of the disorder were defined, among others mainly: basic and secondary symptoms (Bleuler, 1911), first and second rank symptoms (Schneider, 1950), positive and negative symptoms (Andreasen, 1982). Sometimes the significance of the affective and intentional symptoms prevailed, at other times the cognitive disturbances were seen as the central phenomena, thus, e.g., in reframing schizophrenia as a "cognitive illness" (Kahn and Keefe, 2013).

For more than 100 years the enigma of schizophrenia has been under debate (Häfner, 2005). Particularly the nature of "basic" (Bleuler, 1911) or "negative" symptoms (Andreasen, 1982) is unclear: are these psychopathological phenomena—special cognitive disturbances, blunted affect sometimes difficult to differentiate from depressive disturbances (Häfner et al., 2013), and intentional disturbances—core symptoms of the disease or risk factors or consequences of the disease—or a mixture of all of them?

Lara Rzesnitzek (2013) in her informative and readable review recalls the early discussion on the nosological status of "early psychosis": are its seemingly unspecific but in its entirety rather specific psychopathological phenomena before the manifestation of unequivocally psychotic symptoms dispositional and stable risk factors or initial symptoms of a gradually developing schizophrenia? Today's psychiatrists may wonder about the categorical black-or-white thinking of former conceptualists due to the currently dominating, more multiconditional concept, i.e., a specified bio-psycho-social model: developmental interactions between genetically conveyed sensitivity toward a distinct social context (and perhaps perinatal brain lesions as well) may form a disposition of vulnerability for critical life events, e.g., hormonal changes or social stress during adolescence (Zubin and Spring, 1977; Häfner, 2002; Haddad and Meyer-Lindenberg, 2012). "Early psychosis" today is subject to empirical long-term investigations on transition rates from bland symptoms to full blown psychoses, on contextual conditions and consequences, such as the Mannheim ABC-Study (Häfner et al., 2013), and on treatment.

In view of this I will comment on some ethical implications of "early psychosis," irrespectively whether the symptoms indicate risk factors with predictive value or a beginning psychosis, because symptoms will be treated only if they intensify over time into functional or social handicaps. Due to the blandness of symptoms and their sluggish manifestation the diagnosis of "early schizophrenia" is difficult. This implies a particular *responsibility of the diagnostician* with regard to various aspects:

1. The diagnosis of the mental illness "psychosis" or even "schizophrenia" may stigmatize the concerned person, e.g., it may put a strain on the atmosphere in the family—and even may lead to a self-fulfilling prophecy in the sense of a disturbed mental development of the person involved.
(In connection with the potential of psychiatric diagnoses to stigmatize their bearers it should be mentioned that in 2002 the Japanese Society of Psychiatry and Neurology substituted the term schizophrenia ("split-brain disorder") with the neutral term "integration disorder," in order to avoid a negative stigmatizing effect with the result of lowering the threshold for the contact of concerned persons with professionals (Sato, 2006). Together with this renaming Japanese psychiatrists also changed the etiological concept of schizophrenia from Kraepelin's biological disease concept to the vulnerability-stress model (Zubin and Spring, 1977) and thereby found it easier to explain the disorder to patients).
2. The uncertainty of diagnosis is open to other than medical influence, e.g., political influence, as was the case with dissidents in the former USSR who were silenced by a psychiatric diagnosis, particularly that of "sluggish schizophrenia," in order to keep them away from the public in special psychiatric hospitals (Bloch and Reddawy, 1984; van Voren, 2010). However, not only misuse of psychiatric diagnoses has happened, but also their use in protecting patients, e.g., less stigmatizing terms for schizophrenia were used in the 1930s in National Socialistic Germany in order to protect patients from forced sterilization, which was demanded by law.

The diagnostic uncertainty of "early psychosis" and thereby its prognostic invalidity also calls for *the responsibility of the therapist*. A major discussion deals with the problem of preventive treatment (Klosterkotter et al., 2001). The chance of preventing a full blown psychosis must be contrasted with the risk of side effects of drug treatment in a person who never would have become psychotic without treatment, i.e., the risk of side effects of unnecessary treatment. However, the benefit-risk-estimation (Helmchen, in press) in such cases is difficult insofar as the psychiatrist:

1. must deal with a large degree of uncertainty of predictive criteria of "early psychosis,"
2. must consider the risk of stigmatization by a premature or unnecessary diagnosis for the concerned person, and
3. must explain understandably the probabilities of transition from "early psychosis" to full blown psychosis and of its prevention by treatment.

Corresponding to these demands the promotion of the concept of existential philosophy by anthropological psychiatrists was helpful, because it opened up an understanding of the subjective experiences of the (pre-)psychotic individual and fostered the recognition of the person and efforts to understand comprehensively the individual patient: the better the knowledge of a person in his/her contexts the better he/she can be informed appropriately.

However, Rzesnitzek's description exaggerates the role of the anthropological concept in West German psychiatry of the 1950s and 1960s, because it did not dominate the entire West German psychiatry but mainly the Frankfurt school of Jürg Zutt and Caspar Kulenkampff and, more or less, the southwest region of Germany. Furthermore, at the same time the very successful drug treatment of people with psychoses stimulated a new interest in neurochemistry, brain functions, and biological aspects of psychosis, and a network of young psychiatrists established long-term investigations on the course and treatment of schizophrenia. Thus, it was not a complete change from the biologically oriented nomothetic approach to a hermeneutic-idiographic concept, but rather the latter was an important addition to the former.

Two additional remarks may be helpful:

1. Today, terms such as the "schizophrenic person" or even "the schizophrenic" are no longer used, because they identify the mental illness schizophrenia with its bearer and thereby extend the negative stigma of the term schizophrenia to the concerned patient. Therefore, terms such as a "person with schizophrenia" comparable to "a person with a bone fracture" are preferred.
2. It might be misunderstood to translate the German term "Schub" as "phase" because the German term "Phase" was restricted to episodes of affective disorders. According to the dominating concept of the Kraepelinian dichotomy of "endogenous psychoses" at that time episodes of psychotic disorders were differentiated terminologically as "Schub" for schizophrenia and "Phase" for manic-depressive disorders. This terminology implied a course of affective disorders with completely remitting episodes, i.e., "Phasen," but a progressive course of schizophrenia with a remaining residual on a lower level after an episode, i.e., "Schub" in the sense of taking a step downward. However, this terminology is no longer used, due to the fact that episodes of pure affective disorders may end with a remaining residual, and unequivocal episodes of schizophrenia may remit completely.

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Clinical knowledge, health policies and social identities. Commentary on Lara Rzesnitzek (2013) "Early psychosis as a mirror of biologist controversies in post war German, Anglo-Saxon and Soviet psychiatry"

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In her wide ranging and thoughtful article Rzesnitzek (2013), recalls the long, conflicting and at times convoluted history of attempts to describe and delineate the beginnings of schizophrenia. As Rzesnitzek shows us, this history is at once scientific, clinical, and political. It involved practitioners from all over the Western world as well as from what used to be called the Eastern world. The reader is struck by the range of the debates, the number of conflicting positions as well as the size of the research effort involved in these debates. In a way, the debates over the beginnings of schizophrenia were merely an abridged version of those over the nature of the disorder itself. In fact, while Rzesnitzek herself does not clearly make this point, her article demonstrates quite convincingly that the debates over the beginnings of schizophrenia *were* debates over the very definition of the disorder. What was seen as the manifestation of an early form of schizophrenia or what was rather understood as a predisposing condition clearly reflected divergent visions of the nature and symptoms of the disorder. As a sociologist and social historian, I would add that these visions should in turn be associated with the settings and practical conditions in which psychiatrists saw patients.

What also necessarily strikes the reader is the open-ended nature of these debates. There clearly were—and probably still are—too many uncertainties to overcome to reach an agreement over criteria for defining the early phases of schizophrenia. Given these uncertainties, one may and probably should wonder what stimulated the interest of several generations of psychiatrists in developing tools and criteria for the early diagnosis of schizophrenia. Rzesnitzek does not give an answer to this question, although her article suggests the fascination that may have been created among researchers by questions surrounding the nature of what a recent book has called "the sublime object of psychiatry" (Woods, 2011). In the remainder of this commentary, I prefer to reflect on some of the consequences of the debates for the people concerned. I will specifically comment on two dimensions of the story of early psychosis, which Rzesnitzek does not explore at length.

The first concerns the policy and political dimensions of the story. Seen from Germany, this may not be an important perspective. Nazism, with its programs of sterilization and euthanasia, brought into disrepute both a generation of psychiatrists and ways of thinking about mental disorders, which probably prevented any further efforts at developing preventive practices and policies in the field of mental health in Germany after World War II. However, in many countries and in various ways, both mental hygiene and eugenicist movements remained strong players in the psychiatric field until well after 1945 (Kevles, 1985; Grob, 1991; Rose, 2001; Bashford and Levine, 2010). The idea of mental health as a resource to be preserved and of mental health professionals as contributors to the public good have been major aspects of psychiatric thinking in most Western countries after World War II. In many countries, these ideas have translated into programs in primary prevention, especially with children (Jones, 1999; Stewart, 2013). On a darker side, sterilization programs continued in the US as well as in Northern Europe until at least the late 1970s (Broberg and Roll-Hansen, 2005; Largent, 2008). To what extent researchers in the field of early diagnosis or genetic psychiatry aimed at contributing to these policies is not clear, just as it is not clear what sort of prevention practices have been developed from their research.

Today's practitioners in the field of early intervention have developed a strong awareness of the policy implications of their work, and a segment of the field has even developed a commitment to developing policies that may help to better screen, diagnose, and treat schizophrenia in its early phase. The International Declaration on Youth Mental Health launched in 2012 by a group of youth psychiatrist is the last and most spectacular action in this direction (Coughlan et al., 2013). Yet these actions rely on a concept of mental health policy which differs in major ways from earlier proposals. While the mental hygiene movement was built upon the idea that the psychiatrist was the only expert in defining and enforcing prevention practices, today's mental health movement insists on the necessary participation of the people concerned in their own treatment and care. In a way,

concern for mental health affects all of us, and may be a way of life for the most vulnerable. The psychiatric profession is only one actor in the drama of mental health, and often doesn't play the most important role.

This leads to my second commentary, which concerns transformations in the experience of developing schizophrenia as it relates to the changing experiences of being young and becoming adult. The history of schizophrenia as a "coming-of-age" disorder remains largely to be written. Yet, as Rzesnitzek reminds us, age is an obvious component of this disorder. The label for the most iconic form of schizophrenia, "hebephrenia," was historically proposed by German psychiatrists Kahlbaum and Hecker to label a disorder typically characterized as affecting young people (Kraam and Phillips, 2012). The term was not kept in DSM III, but the definition of schizophrenia in this manual entailed as a criterion an age of onset before 45—a criterion, however, that was removed in subsequent editions (American Psychiatric Association, 1980). More recently, the concept of an "at-risk mental state" targets young people between 16 and 30 (McGorry et al., 2003). And in fact, current practices and policies of early intervention are built upon the premise that young adulthood is an age of maximal vulnerability to mental health disorders.

There is nothing here to surprise a sociologist. As a large body of scholarship in sociology and psychology has now shown, the age between 20 and 30 has emerged as a new life phase characterized as an age of uncertainties, both existential and economic (Booth et al., 1999; Arnett, 2001; Van de Velde, 2008; Booth, 2012). At the same time, it is clear that for most young people these uncertainties, however distressing they might be, will never translate into a problem as dramatic as a major psychiatric disorder. The characterization of early psychosis as a condition affecting young people should be understood in this context. In fact, many mental health professionals see a continuum between minor mental health problems that may develop as a consequence of the existential turmoil of young adulthood, and major psychiatric disorders—or, at least, they are not able to differentiate between a minor mental health problem and the initial signs of what may turn out to be a major psychiatric disorder (see for instance Patrick McGorry's model of clinical staging: McGorry et al., 2007). In turn, it is probable that early intervention practices and policies will affect our vision of young adulthood—including young people's visions of themselves. In North America, several social movements initiated and led by young people are now trying to make a case for youth mental health on academic campuses, and a new generation of youth mental health activists has emerged who are beginning to play an important role in advocating early intervention. In this regard, resilience to early psychosis is perhaps becoming a way for a new generation of young people to build their identity.

Both these discussions point to the fact that medical entities have a social life that extends well beyond the jurisdiction of medicine. As medicine legitimates the existence of a phenomenon as a medical entity, it also leaves open the possibility for many other actors to use this definition for their own purpose—whether these actors are politicians, administrators, activists or patients. This is why discussions on the dangers of psychiatric labeling and on ethical safeguards to help psychiatrists anticipate and prevent the consequences of their judgment may not necessarily always be effective. What patients and society at large do with medical concepts usually goes beyond what medical men and women—including ethicists—imagine. However conscious of the implications of their judgments medical practitioners may be, what becomes of these judgments will certainly be far beyond their reach. Philosopher Ian Hacking has proposed the concept of "looping effect" to describe the transformations which people labeled with a medical diagnosis may in turn create within these diagnostic labels once they have adopted them as their own (Hacking, 1995, 1999). Historian Charles Rosenberg also wrote about the "tyranny of diagnosis" to point to both the necessity of diagnosis in medical practice and the burden of its often unwanted and unexpected consequences (Rosenberg, 2002). This may be even more complicated for the medical profession in situations such as early intervention where the medical status of a category remains disputed, although this category nevertheless has a life of its own outside medicine.

A final striking dimension of the story told by Rzesnitzek is the existence of local variations in the conceptualization and use of a diagnostic category such as early psychosis. This aspect would probably have been even stronger if Rzesnitzek had had the chance to describe actual research and clinical practices that have developed around this category in different countries at different periods. Indeed, such variations not only reflect different clinical traditions, but also different approaches to the practical issue of treating people and different approaches to psychiatric research. The very German history told by Rzesnitzek has a lot to do with the specificity of psychiatric research in that country and its organization around the psychiatric clinic as an academic institution. In contrast, the funding of US psychiatric research by the American Congress may have made it much more sensitive to pressures from the social world. However, from my two previous series of remarks, one could also infer that these variations also owe much to the diverse ways in which the people concerned act in relation to their labels. This layer of complexity adds to those already present in Rzesnitzek's article.

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