

Are Mental Illnesses Diseases of the Brain?

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This chapter offers a systemic and ecological account as an opposing view to the naturalist idea that mental illnesses can be reduced to dysfunctions of the brain. Mental illness is regarded, on the one hand, as inseparable from the living organism and on the other, as inseparable from the patient's lifeworld or social environment. In order to grasp mental disorders in their context, the notion of monolinear causation has to be replaced by the notion of *circular causality*. In this view mental illnesses are marked by a disruption of *vertical circular causality*, that is, the interplay between lower-level processes and higher faculties of the organism. This primarily affects a mentally ill person's relation to themselves which continually co-determines the course of the illness. On the other hand, mental illnesses are characterized by a disruption of *horizontal circular causality*, in other words of social relationships and the ability to respond adequately to the demands and expectations of others. This leads to negative feedback loops in socio-functional cycles that influence the course of the illness from the very beginning. Both kinds of circular causal processes are tied to mediation by the brain, but cannot exclusively be located within it. For this reason reduction of mental illnesses to diseases of the brain is in principle not possible.

The basic research program of the neurosciences consists in naturalizing consciousness, subjectivity, and also intersubjectivity—in other words explaining them in neurobiological terms. Even though this program is far from being realized, the impression is created that subjective experience can be imaged in the brain and in this way, as it were, materialized. This has far reaching effects on our image of the human being in general. The use of “brain language” is increasingly permeating our self-conception. In the wake of a popularized neurobiology, we are beginning to regard ourselves not as persons having wishes, motives, or reasons, but as agents of our genes, hormones, and neurones. Consequently, our problems and sufferings are often no longer considered existential tasks that we must face, but results of malfunctioning neuronal circuits and hormonal metabolism.

Critical Neuroscience: A Handbook of the Social and Cultural Contexts of Neuroscience, First Edition.

Edited by Suparna Choudhury and Jan Slaby.

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Biological psychiatry for its part aims to find the cause of mental illness in deviant functioning of the brain, according to the dictum commonly ascribed to Griesinger: "Mental illnesses are diseases of the brain."¹ The—as yet—poor attempts towards the end of the nineteenth century by Theodor Meynert (1884), for instance, to subsume mental illnesses under the "diseases of the forebrain" were derided by Jaspers (1913/1973, p. 16) at the time as "brain mythologies." Today, however, it seems only a matter of time until specific genetic and neurophysiological correlates of all mental illnesses are found and allow us to causally trace them back to neuronal substrates. If anxiety disorders, depression, and schizophrenia are actually brain disorders, psychiatry finally becomes a branch of neurology and the psychiatrist a brain specialist.² Against such a background, there is a risk that therapeutic interventions in psychiatric practice will increasingly be oriented towards brain-centered procedures—pharmacological or directly stimulating modes of influencing brain functions—at the loss of psychotherapeutic or systemic approaches that consider the patients in their biographical and environmental context.

In what follows, I want to provide an opposing systemic-ecological view of mental illnesses. It is based on the assumption that, from birth on, the brain is embedded in interrelations between the person and the environment and is best seen as an organ of mediation and transformation for biological, mental, and social processes that are bound up in circular interplay. In this interplay, subjectivity—a person's experience and their relation to themselves—plays a central role, no less than the person's social interactions with others. For this reason, I claim that mental illnesses are not just brain diseases in the sense in which, for instance, we can trace back an angina pectoris to a coronary heart disease. The patient's altered subjective experience and disturbed relation to others are not mere epiphenomena of an effective organic process; much rather, they are essential elements of the illness itself. However, in order to grasp mental disorders in their subjective and intersubjective context, we first need to consider the notion of *causality* in living systems. Only by challenging the one-way causation that leads from the brain to the mind will we advance an ecological view of mental disorders and, through this, a person-oriented psychiatry.

Circular Causality of Living Systems

In order to embed the brain in the relations of organism and environment, I want to introduce, in what follows, the notion of *circular causality* as a property of living systems (Fuchs, 2009; Haken, 1993). It characterizes the systemic processes of interplay and feedback that were also foundational for Jakob von Uexküll's model of the *functional cycle* (1920/1973) and Viktor von Weizsäcker's theory of the *Gestalt cycle* (1940/1986; see also Fuchs, 2008, p. 121 et seq.). Both concepts refer to the

¹ Note that Griesinger himself in no way held a purely biological view. He was concerned with opposing a contemporary view according to which mental illnesses could not only be located in the brain, but in the entire body (see Schott and Tölle, 2006).

² See Insel & Quirion (2005): "The recognition that mental disorders are brain disorders suggests that psychiatrists of the future will need to be educated as brain scientists."

inseparable interconnection of perception and movement: what an organism senses is a function of how it moves, and how it moves is a function of what it senses. Thus, the touching hand anticipates and selects what it feels by its movements, whereas the shape of the object reciprocally guides the hand's touch. Through this, organism and environment co-constitute each other. Similar concepts have been developed more recently in enactivist theories of perception and cognition, as put forward by Varela, Thompson, and Rosch (1991), O'Regan and Noë (2001), Thompson (2007), and others. The feedback cycles between an organism and its biological as well as social environment may be termed *horizontal circular causality*. Examples are the aforementioned cycles of perception and action, but also the interactive processes in social systems, as they are, for instance, analyzed in family systems therapy (see also Fuchs & De Jaegher, 2009).

But there are also circular relations *within* the organism, namely between the whole and its parts, or between lower and higher systemic levels. I characterize them as *vertical circular causality*. Thus, a living being may be regarded as a system that continuously reproduces the components of which it consists (organs, cells), while these components reciprocally sustain and regenerate the system as a whole.³ The whole is the condition of its parts, but is in turn realized by them. Such a structure, for instance, characterizes the relations between genes and the organism: the genetic structure of an individual cell nucleus controls the necessary production of specialized cellular organs and functions ("upward" causality). Conversely, the configurations and functions of the entire organism determine which genes are even given relevance for the development and regulation of a certain individual cell ("downward" causality).

This type of causality is often regarded as problematic or obscure, for two main reasons. First, since the whole consists of the parts itself, cause and effect cannot be assigned here to separate agents acting externally on each other. Second, the causal effect of higher systemic levels seems to presuppose unknown physical forces, thus either contradicting the laws of physics or falling prey to Occam's razor (see Craver & Bechtel, 2007 for a criticism). However, there is no need to restrict the notion of causality to *efficient* causality, according to the paradigm of billiard balls acting on each other. Macro-structures can well have *formative* causal influences on the micro-elements by which they are structurally realized. This formative causality does not imply the emergence of novel natural forces that are at odds with the laws of physics. Rather, macro-structures, by their particular form and configuration, are able to "select" certain properties of their components, and "block" others (Campbell, 1974; Moreno & Umerez, 2000). Moreover, the components may also acquire new, emergent properties. For example, iron molecules integrated into haemoglobin become able to reversibly bind oxygen, which is an extremely improbable state in anorganic nature. No physical "miracle" is required to accomplish this, but only a higher order structure (in this case haemoglobin) which "enslaves" its own constitutive elements (Haken, 1993) and involves them in specific patterns of behavior.

³ Accordingly, Varela has defined an autopoietic system, or the minimal living organization, as "one that continuously produces the components that specify it, while at the same time realising it (the system) as a concrete unity in space and time, which makes the network of production of components possible" (Varela, 1997, p. 75).

Similarly, mental processes may have a formative impact on the physical behavior of living beings without being reducible to the physical events by which they are realized. When I am speaking, the muscles of my tongue and larynx show organized movement patterns. Their immediate efficient cause is the neuronally triggered release of acetylcholine at the motor endplates causing the muscle fibres to contract. However, it is equally adequate to say that my tongue and larynx move the way they do "because I am speaking these sentences." The over-arching, formative or organizing cause of the muscle actions is my speaking ("downward"), which in turn is realized by a series of combined physiological mechanisms ("upward"). The cause of my speaking, however, is neither my tongue nor my brain (though both are necessary to realize it)—it is me. Thus, in any conscious performance (speaking, writing, running, or thinking, for example), the living being itself acts as a downward formative cause, or in other words, the achievement in question is realized by vertical causality.

Accordingly, vertical causality also characterizes the functions of the brain. Pain stimuli from the periphery, for instance, through central processing in the brain, lead to pain experience ("upward"); conversely only the overall situation of attention and affectivity determines whether an impulse is "admitted" as a painful experience or whether it is suppressed by descending, inhibitory tracts ("downward"), as may be the case in a state of intense affective excitement. To give another example, an emotional state can, on the one hand be treated pharmacologically, by influencing the transmitter metabolism in the brain (upward). On the other hand, this can also be achieved psychotherapeutically, by changing the subjective perception of one's personal situation (downward). In this sense, anxiety can be influenced by sedatives as well as by a calming talk. As such, subjectivity represents a high or integral systemic level of the organism that feeds back into lower-level physiological processes. The brain functions as a *transformer* for this vertical circular causality, by converting higher- and lower-level influences on the organism and "translating" them into the other levels in the hierarchy (see Fuchs, 2009, 2008, p. 158 et seq.).

Mental Illness as Circular Process

Having introduced these terminological clarifications, let us now try to characterize mental illnesses as circular processes. I begin with vertical circular causality.

Vertical circular causality

Other than in the case of somatic conditions, in mental illnesses the patient does not succeed in attributing the condition to their body; for the condition primarily affects their experience of themselves. Put differently, the subjective side of the illness does not consist merely in a secondary reaction to physiological dysfunctions. To a certain extent, it always involves a self-alienation or a "splitting" of the self. Something "in me" confronts me, defies my control, or dominates me while I desperately try to take control again, be it a panic attack, a depressive mood, a compulsion, or audible thoughts. Impulses or functions that have so far been integrated, take on a life of their own or become particularized and defy control. Mental illness hereby affects the

person centrally, namely in their experience of themselves and in their autonomy. What is more, the altered experience of and relation to themselves, as such, is an effective factor in the further course of the illness. It follows that, independent of its origin, *vertical circular causality* always plays a decisive role in the illness.

Take the example of a depressive disorder. In whatever way different causal conditions—be they genetic, neurobiological, biographical, or interpersonal—interact in the particular case, as soon as the depression becomes manifest, it is per se an *illness of the person*. The disorder is accompanied by a fundamental change in bodily experience (inhibition, restriction, anxiety, heaviness, and loss of motivation); hardly any other illness has a comparable effect on a person's bodily subjectivity. But it also gives rise to negative perceptions and evaluations of oneself (self-reproach, feelings of guilt) and typical, depressive patterns of thought—negative assessments of their situation by the patients. These negative self-assessments, as self-fulfilling prophecies, increase the likelihood of further failures and contribute to the depression. Similar vicious circles are well known in anxiety disorders. They have the following pattern: the occurrence of physiological features of stress (activation of the sympathetic nervous system, increased pulse rate, and so forth) leads in turn to the perception of the physiological symptoms as “threatening,” catastrophic cognitions and evaluations, increased physiological stress and so on. The subjectivity of experience, as a relation to oneself, thus becomes an important component affecting the course of the illness.

Every psychopathological experience is characterized by a personal meaning that the patient attributes to it, and a certain stance that they take towards it—suffering passively, giving in, acting out, fighting against it, or detaching oneself from it. This position taking is a relevant clinical feature in itself. Of course, these subjective modes of experience and behavior are enabled by neuronal processes, otherwise they could not be effective within the organism. The brain here functions as a transformational organ that converts peripheral and central, lower- and higher-level components of the previously mentioned “vicious circles” into one another. However, the phenomena of subjectively ascribing meaning, assessing a situation, and relating to oneself cannot be equated with processes in the neuronal substrate, as these lack acts of meaning making or intentionality. That all thought is realized in neuronal activity does not make it the case that it is identical with brain processes. Intentional content and directedness is inseparable from a subject's relation to the world. If neuronal processes function as “carriers” of intentional acts, they can do so only as part of an overarching life process that includes the organism as a whole and its environment. In this way, mental processes are enabled or realized by neuronal processes, but are not localizable in the brain.

In a similar vein, it is not possible to reduce mental illness to circumscribed neurobiological dysfunctions—no matter how reliably correlated dysfunctions of the substrate can be identified. For, on the one hand, the subjective experience of the illness in its specific quality—its “what-it-is-like-ness” and its intentional contents—is not reducible to physiological descriptions. No imaging of brain activities can provide a psychiatrist with an understanding of what it is like to be depressive, to experience a panic attack or to hear voices. In fact, imaging methods themselves do not even provide criteria for what counts either as a pathological or as an ordinary physiological process—this can only be known from clinical practice, that is, from the patient's

experience and behavior. Moreover, no description of the biological markers of anxiety or depression, however detailed, will tell us whether the patient in question is worried about a failure in the past, a threatening loss of his or her job, a public speech the patient has to give, or a current illness of his or her child. Obviously the biological data will be of very limited value as long as it remains isolated from its experiential context.

On the other hand, an even more crucial reason for this irreducibility is given by the patient's relation to himself, which is continually involved in the illness process, influences it positively or negatively, and, as such, bars us from seeing mental illness as purely biological. The perception and assessment of one's own condition are genuinely personal phenomena that also limit the transferability of animal models to particular components of the illness. They give rise to a unique, specifically human kind of vertical circular causality, namely the feedback from subjective perceptions and evaluations into more fundamental processes of the illness. Not least, the possibility of suicide—which only humans have—bears witness to the fact that the relation to oneself can significantly influence the course of the illness, though, in this case, fatally.⁴

Horizontal circular causality

Just as mental illnesses cannot be detached from the person and be ascribed exclusively to the neuronal substrate, it is also not possible to see them as purely individual dysfunctions; in other words as detached from their *interpersonal aspects*. Irrespective of their causes, mental illnesses are always disturbances of the patient's interactions and relationships. They are accompanied by various impairments of the freedom to flexibly and autonomously respond to situations, offers, and demands of the social environment. As such, one can characterize them as impairments of a person's *responsivity* (Fuchs, 2007): certain abilities of the patient to shape social relationships according to their needs are either inhibited due to the illness or have not been developed in the first place. Thus, a significant part of psychopathology cannot be assessed in isolated patients, let alone their brains, but only as interactional dysfunctions.

As soon as social responsivity is impaired, feedback effects necessarily occur in the socio-functional cycles and, from the very beginning, influence or even determine the progression of the illness. The gestalt cycle of social perception and action is impaired or interrupted; the patients lose the usual resonance of their environment. Therefore, one can also characterize mental illnesses as communicative dysfunctions in the broadest sense. Symptoms of the illness evoke these dysfunctions, but they, in turn, are sustained, promoted, or even generated by the communicative impairments.

In the case of depression, for instance, a loss of emotional resonance occurs; that is a severe dysfunction of the responsivity to and exchange with the environment (Fuchs, 2000, 2001). This dysfunction in turn intensifies the patient's depressive self-perception. However, it also has an effect on their social system. Family and friends

⁴ This is not to portray suicide as a freely chosen action, for it is almost always based on a severe cognitive and emotional narrowing of situative perception. Nonetheless, it presupposes an assessment of the situation by the patient and cannot just be seen as a manifestation of a neurobiochemical dysfunction.

usually react at first by giving support, but over time with an increasing sense of helplessness and feeling of guilt as well as with latent or open annoyance. Their mostly inconsistent behavior and the patient's depression amplify one another in a vicious circle (Ruf, 2005, p. 178). The crucial influence of partnership interaction on depression has repeatedly been confirmed (Backenstrass et al., 2007; Barbato & D'Avanzo, 2006; Mundt, Kronmüller, Backenstrass, Reck, & Fiedler, 1998). Further factors aggravating the illness are negative consequences in the workplace, the feared or actual stigmatization of the patient, but also a possible secondary gain. All of these influences are certainly not generated by the brain, but are continuously processed and transformed into altered dispositions of experience and behavior.

Circular Causality in Pathogenesis

Let us take a look at the aetiology of mental illnesses, once again, in the case of depression. Here, too, we find the above-mentioned dysfunctions in vertical and horizontal functional cycles. A look at the epidemiology of the condition is sufficient to show the inadequacy of purely biological explanations, for, in recent years, a significant increase in depressive disorders can be observed in highly industrialized societies, which most certainly cannot be traced back to genetic or neurobiological causes, but is rather due to social and cultural causes.⁵ The fact that the brain functions as the biological "final common path" for the various influences does not make the resulting illnesses brain diseases.

Nevertheless, epidemiological observations aside, we can also easily clarify the role of subjective and intersubjective processes in the aetiology of the illness. The manifestation of a depression is usually preceded by a personal situation that is perceived as a severe loss or threat by the person under the assumption that they do not have the resources for coping with it ("learned helplessness;" Seligman, 1975). Subjective perception and evaluation is, therefore, the decisive triggering factor. At the neuronal level, mediated by linking of prefrontal and limbic centers, and with significant involvement of the amygdala, this is accompanied by physiological stress which consequently leads to massive dysfunctions of the organismic functional cycles. This primarily affects the CRH-ACTH-cortisol, respectively the sympathetic nervous system as well as the serotonin-transmitter regulation in the limbic system. The self-perception of this altered organismic state, as a negative feedback loop, intensifies the physiological symptoms of stress. As a result, the organismic reaction becomes detached from its integration in superordinate feedback cycles and eludes

⁵ According to studies in the US, the frequency of depressive disorders increased tenfold between 1945 and 1990; certainly, altered diagnostic habits had a considerable share in this (Cross-National Collaborative Group 1992; Weissman & Klerman, 1978). Data from other countries, however, points in a similar direction. Even the incidence of schizophrenia is not independent of cultural influences, as has often been assumed. Studies in several European countries, among these the United Kingdom and the Netherlands, have shown that the frequency of schizophrenia among immigrants in the new environment is 4–10 times higher than in the native population as well as in the original population in the country of origin (Cantor-Graae, 2007; Fearon et al., 2006).

the person's control. Negative horizontal feedback loops connecting to the social environment then influence the further course as described above.

It thus emerges that, for the development of depressive disorders, subjective experience in no way merely plays an epiphenomenal role. Rather, the illness originates in a specific perception of the situation, in an "individual act of meaning-ascription" that is not, as an intentional relation to the environment, reducible to neuronal processes. Depression results from a perceived loss of meaning and social resonance, not from a lack of serotonin. Moreover, it is not the objective features of the situation, but their subjective evaluation as insurmountable, which is decisive for the depressive reaction. Consequently, biographically acquired dispositions such as lack of self-worth or self-efficacy become highly influential factors in pathogenesis. Only secondarily do the physiological reactions take on a life of their own as a sustained regulatory dysfunction affecting the entire organism. Granted, in later stages depressive episodes may result from minor events or even from somatic triggers. But even then the organismic dysfunction always remains circularly connected to the patient's subjective perceptions as well as to their illness-related behavior in interpersonal relations.

Though the weighting of the factors involved differs in other disorders, we can generalize the paradigm of depression insofar as we always find in mental illnesses a *complex interplay of circular processes* both at the vertical, organismic level and at the horizontal, interpersonal level. In each of these internal and external circularities, the brain functions as an organ of transformation or mediation—as the carrier of the biological component of pathogenesis. Its structure is, however, continually shaped and modified in turn by psychosocial interactions. In this way subjective experience, as a significant component of the interaction of environment and organism, exerts a structuring influence on the neuronal substrate—an insight that is of no little relevance for psychotherapeutic practice.

Asserting this general basic structure does not imply that all mental illnesses need to be considered in the same way. It is by all means necessary to distinguish whether an illness is to be traced back to a comprehensible relation between a personal learning history and experience of the environment (as in the case of anxiety disorders), to a neurosystemic dysfunction affecting the constitution of the self (as in the case of schizophrenia), or to a macroscopically identifiable lesion of the brain (as in the case of an apoplectic stroke). Depending on the illness, psychosocial and biological aspects have to be weighted differently. Intentional and psychosocial explanations remain indispensable for neurotic disorders that are derived from dysfunctional patterns of perception, behavior, and relationships (Henningssen & Kirmayer, 2000). Even if dysfunctions of neuronal systems are involved here as well, these are usually epiphenomena that necessitate pharmacological treatment only in the event that they become independent and chronic. Neurophysiological approaches are generally more relevant for those disorders that can be seen as defects in ordinary functioning. But even psychiatric or neurological defects are always connected to adaptive coping processes that are accessible to intentional modes of understanding and treatment—and this even applies to the formation of delusions (Kern, Glynn, Horan, & Marder, 2009; Solms, 2004).

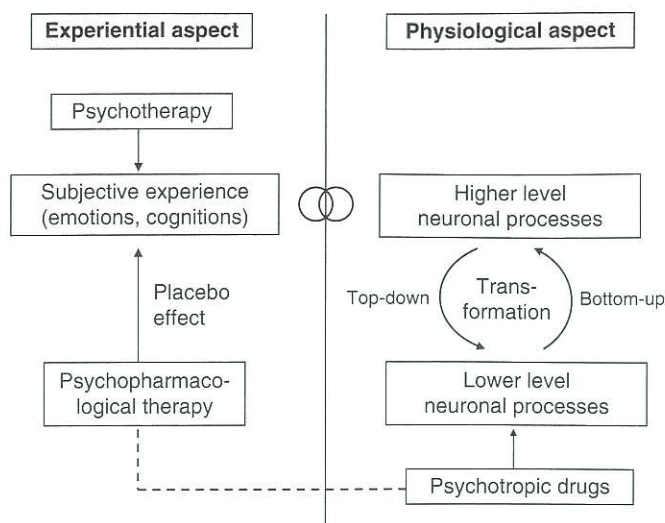


Figure 16.1 Effects of psychotherapy and drug therapy as seen from an experiential aspect (left) and from a physiological aspect (right). The two circles in the middle (∞) signify concomitant or concordant changes within both aspects; there is no “efficient causality” between them. Thus, the effect of psychotherapy on the brain is mediated by concomitant higher level neuronal processes being transformed into changes on lower levels. Conversely, the physiological effect of psychotropic drugs is transformed into higher level changes that realize altered subjective experiences (for example, decreased anxiety). However, drugs appear on the left side as well, because they are also efficient within the dimension of experience and meaning, this being known as placebo effect.

Circular Causality in Therapy

Finally, an ecological conception of mental illness also suggests a pluralistic understanding of treatment. The dualistic distinction between somatic therapies acting on the brain and psychological therapies having elusive, purely subjective effects is no longer tenable. The circular interactions of self, body, brain, and environment may be approached at various levels or turning points, since any mode of treatment will be transformed by the brain and hereby contribute to a holistic effect. Psychosocial influences on the level of meaning and intentionality are transformed into altered patterns of neuronal activity on lower levels, and vice versa: pharmacological effects are transformed into changes of brain activity at higher levels, resulting in altered affective or cognitive experience (see Figure 16.1). This means that any therapeutic intervention is of a physiological as well as of a psychological nature.

Psychotherapy addresses the patient as an experiencing, self-conscious, and self-relating subject. Yet its long-term impact is mediated by its effect on brain functions, as has been shown in a number of neuroimaging studies. Psychotherapy produced lasting effects mainly on prefrontal and frontal brain metabolism (for an overview, see Beauregard, 2007; Fuchs, 2004). Thus, in Positron Emission Tomography (PET)

studies of depressive patients, Brody and colleagues (2001) and Martin and colleagues (2001) found significant decreases in prefrontal lobe activity following treatment with Interpersonal Psychotherapy (IPT). These and other comparable studies strongly support the view that the subjective nature and the intentional content of mental processes (thoughts, feelings, beliefs, expectations, and volitions) significantly influence the various levels of brain functioning (molecular, cellular, and neural circuits) as well as brain plasticity. The transformation runs "top-down" that is, it starts from subjective experience that is realized by (though not localized in) higher level neuronal processes (mainly in cortical networks), and results, on the lower level, in altered synaptic transmission, altered gene expression, and rewiring of neuronal networks.

On the other hand, effects of psychotropic drugs start from influencing the transmitter metabolism at lower levels, mainly in subcortical regions, and are transformed "bottom-up" into higher level processes, resulting in a modification of subjective experience. In a particularly interesting PET study of depressive patients, Goldapple and colleagues (2004) found differential target areas of successful Cognitive Behavioral Therapy (CBT) versus pharmacotherapy: CBT primarily produced changes in the medial frontal and cingulate cortex, whereas drug treatment changed metabolism in limbic-subcortical regions (brainstem, insula, subgenual cingulate). This fits the idea of CBT interventions focussing mainly on modifying dysfunctional cognitions, and leading to an alleviation of vegetative symptoms and inhibition, while pharmacotherapy rather takes the opposite course.

However, direct subjective effects of pharmacological treatment must not be overlooked: each drug administration also operates on the intersubjective level of shared meaning and emotional relationship between doctor and patient, commonly known as placebo effect (see left side of Figure 16.1). The resulting changes in brain metabolism have also been demonstrated by neuroimaging: in an fMRI study on major depression, Mayberg and colleagues (2002) again found mainly cortical effects of placebo treatment, as against more subcortical-limbic and brainstem effects of antidepressant drugs.

This underlines that there is no separation, but rather a circular interaction of psychological and biological processes, and accordingly, no "merely biological" or "merely psychological" treatment. This interaction, however, cannot be expressed in terms like "the mind acting on the body" or "the brain producing the mind." Instead, the brain acts as a mediator and transformer which may be addressed through input on different hierarchical levels and which converts it in both directions: neuro-biochemical changes become mood changes on the subjective level, but subjectivity in turn influences the plasticity, structuring, and functioning of the brain. Vertical circular causality allows for both approaches equally.

This illustrates that both ways of treatment may also interact synergically. On the one hand, beyond a certain point, the neurobiological and endocrine dysfunctions involved in depression, for example, may be too advanced to be accessible to interventions on the psychological level. Pharmacological ("bottom-up") treatment may then enable the patient to re-engage in his relationships and, therefore, will indirectly further his or her social well-being. On the other hand, psychotherapy can help the individual to reframe their beliefs, for example, so that they align with the actual

nature of events to which they are directed (Glannon, 2008). This can alter the patient's misperception of events or social situations as well as his or her corresponding behavior in a beneficial way. Moreover, as we have seen, psychotherapy not only changes the patients' implicit relational patterns, attitudes, and behavior, but also the functions and structures of their brains. Mental states are not epiphenomenal to brain states but can have a causal influence on them. In view of the limited effectiveness of medication, especially in chronic illness, it would be wrong to neglect these "top-down" options of treatment.

Drug therapies targeting neuronal pathways and transmitter systems treat only one dimension of mental disorders (Glannon, 2008). Moreover, a mere biological view still tends to isolate the individual patient and to make his illness seem separated from its interconnections with his environment. However, the intentional and qualitative aspects of beliefs and emotions cannot be explained in terms of physical processes in the brain. Nor can we forego (inter)subjective experience if we want to change the patient's maladaptive cognitive, emotional, and behavioral dispositions that have led to his or her illness and may lead to a relapse in the future. Only conscious, embodied experience is able to correct the corresponding dysfunctional patterns of neural activity. And only repeated interactions with the environment—in other words, processes of interpersonal learning—can stabilize new attractors of perception and behavior in the brain. Since the neural structures that underly our personal dispositions are shaped by embodied experience, there will probably never be a way to create new views of the self and the world by brain manipulation directly. Any psychotherapeutic and social approach to psychiatry is thus based on a holistic, ecological view of life.

Conclusion

The brain is not the sole producer of the mind but a relational organ that mediates the interaction between the organism and its complementary environment (Fuchs, 2008). Our mental states are the emergent products of circular causation consisting of neurophysiological, environmental, and social influences continuously interacting with each other in a series of feed-forward and feedback loops (Fuchs, 2004, 2009; Glannon, 2008). Disordered states of mind result when these circular processes are disturbed in some way. I have distinguished two dimensions which characterize this disturbance:

- On the one hand, mental illnesses can be characterized as *dysfunctions in vertical feedback cycles*. The central integration of partial functions or impulses fails; the latter take on a life of their own and elude the person's control, for instance, in the form of neurotic symptoms, compulsions, panic attacks, disorders of impulse-control, self-disorders, hallucinations, and so forth. These particularized processes, in turn, affect the person's relation to themselves. They lead to various attempts at coping and reintegration, but also to secondary reactions and symptoms ("fear of fear", self-reproaches, for example) that make the illness worse; in other words, they are a significant component in its progression.
- On the other hand, mental illnesses can also be seen as *dysfunctions in horizontal feedback circuits*, for they are connected to more or less severe impairments of

responsivity and interactions with the social environment. In relationships to significant others, negative feedback loops and vicious circles occur that sustain or further intensify the symptoms. These feedback loops are tied to (inter)subjective perception and evaluation, to the patient's and their relatives' experience and behavior. Though they certainly influence brain functioning, they may not be described on the brain level alone.

Having mentioned both aspects, it follows that a reductionist description and explanation of mental illness based on neurophysiological facts alone does not do justice to its actual complexity. No mental illness can be diagnosed, described, or explained without taking account of the patient's subjectivity and their interpersonal relationships. Mental illnesses are always illnesses of the person and their relationships to other persons. The brain, with its functions, is centrally implicated in them, but a narrowly neurobiological perspective is never sufficient to describe and explain all facets of the illness. The final disorder is the product of a cascade of subjective, neuronal, social, and environmental influences continuously interacting with each other. Within these circular interactions the brain acts as a mediating, transforming, and also amplifying organ, but not as "the monolinear cause."

While advances in neurobiology have contributed to overcoming dualistic models of mental illness, one would be throwing out the baby with the bathwater if one wanted to trace all forms of mental illness back to brain processes in an undifferentiated manner. Neurophysiologically (by means of imaging technologies) determinable anomalies in themselves are not more than correlative in character. No such findings could be identified as pathological at all without being related to subjective suffering and intersubjective disturbances. They only become aetiologically relevant if they are embedded in the overarching circular processes that include the organism-environment system as well as the patient's interpersonal relationships.

In the case of obsessive-compulsive disorder (OCD), for instance, hyperactivity of the caudate nucleus provides no indication as to the cause of the disorder. Local activations of the brain's metabolism only correlatively reflect the function that is being activated; they are only a partial component of the illness. Depressive and anxiety disorders are not solely caused by the amygdala, just as OCD is not solely caused by the caudate nucleus, even if these brain regions are implicated in the illnesses. To the extent that neurophysiological changes are to be found, these are correlates, adaptive processes, or biological scars that have emerged in the context of repeated perceptions of situations as dangerous or threatening. Even if neurosystemic developmental impairments in schizophrenia or amygdaloid hyperactivity in posttraumatic stress disorder clearly act as restricting factors, such dysfunctions never become monolinear causes.⁶

Integrally viewing mental illnesses as relational dysfunctions, however, is also a precondition for treating them adequately. The complexity of the circular processes is not best captured either by an opposition between or a mere summation of various

⁶ Monolinear causation may only be attributed to brain lesions (for example, apoplexy, brain tumor) that result in a failure of functions. However, even failures of this nature are followed by manifold processes of adaptation and coping that imply circular interactions of person, brain, and environment.

therapeutic approaches. What is called for is rather a bi- or polyperspectival approach. Here various, especially somatic and psychotherapeutical approaches, can be combined to influence circular causalities. However, psychosocial descriptions and interventions will remain indispensable, for a purely neurobiological explanation or treatment of mental illnesses is not in principle possible. What psychiatry needs is a systemic or ecological view of the brain in order to better understand the interplay of biological, psychological, and socio-cultural processes and to do justice to the complexity of its subject matter. This is not the brain in isolation, but the embodied human being living in relationships.

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