Nature, Diagnosis and Classification of Mental Disorders

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What is a mental disorder? This question must be addressed by any diagnostic and classification system in psychiatry. In this chapter, a brief systematic overview of theories of mental disorders is given and an integrative working definition is suggested. Furthermore, an overview of the prevalence of mental disorders in Europe is given and the current situation for psychiatric care in Germany is presented. Subsequently, some problems of the current classification systems (ICD and DSM), which are based on clinical criteria alone, are discussed. Finally, three more recent approaches to the understanding of mental disorders are presented, which follow an integrative approach, i.e. take clinical, neuroscientific and psychosocial aspects into account.

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Learning Objectives

After reading this chapter, the reader should be able to explain how mental disorders can be defined, how they are diagnosed and classified, how common they are, and which recent integrative approaches to mental disorders are currently being discussed.

9.1 What Is a Mental Disorder?

9.1.1 Background and Historical Context

Psychiatry deals with mental disorders such as schizophrenia, depression, anxiety disorders, obsessive-compulsive disorders or post-traumatic stress disorders. In contrast, neurology deals with diseases such as stroke, brain tumors, multiple sclerosis, peripheral nerve damage and muscle diseases. However, there is also overlap between these two medical specialties, such as dementia, which is addressed by both. While it is relatively easy to give examples of mental disorders, it is much more difficult to say what a mental disorder actually *is*. Problems of demarcation arise on two sides. First, where does the psychological normal end and the pathological begin? Where, for example, is the boundary between shyness and social phobia, between grief and depression, or between extraordinary experiences like hearing voices and schizophrenia? Secondly, where is the boundary between neurology and psychiatry, when both are apparently based on dysfunctions of the brain? In addition to these problems of demarcation, there is the specific issue that psychiatry has repeatedly been accused throughout its history—and in certain cases quite rightly—that the term "disease", "illness" or "disorder" serves only to pathologize deviation from social standards and thus serves to discipline society. It was not until 1990, for example, that the World Health Organization (WHO) removed homosexuality from the list of mental disorders. In Germany, homosexual acts were still considered a criminal offence until 1969, and the corresponding paragraph §175 was deleted from the Criminal Code only in 1994. Political dissidents in the Soviet Union were sometimes labeled as mentally disturbed and internalised in hospitals for "treatment" if they did obey to the insights into the truths of Marxist-Leninist doctrines. And in the Third Reich, based on racial hygiene doctrins, the persecution and murder of mentally ill patients was systematically organized within the T3 action.

Considering this background, it is not surprising that strong opposition to clinical psychiatry arose in the 1960s, which must also be regarded against the background that at that time many mentally ill patients were housed in large, isolated state hospitals under conditions that are unacceptable from today's standards and were treated inadequately, if at all. The anti-psychiatric movement emerged, which postulated that mental disorders did not exist, but were only socially constructed based on deviant behavior and thus ultimately an expression of social problems. In Western Germany, the Psychiatry Enquete Commision in 1975 drafted a

report on the state of psychiatry. As a result, many of the state hospitals were dissolved, Departments of Psychiatry were created in normal hospitals, i.e. psychiatry was integrated into medicine, and many things improved.

These psychiatry-specific developments coincided with a boom of the neurosciences, a better understanding of the nervous system, the introduction of effective psychotropic drugs in the 1960s and 1970s, and a professionalization of psychotherapy with the development of new, effective treatment methods. Knowledge about mental disorders has increased considerably since then, although not to the same extent as, for example, in neurologgy.

In addition to its therapeutic mandate to treat disorders and reduce suffering, psychiatry today still has a public-legal function. When patients, due to a mental disorder, have a lack or strong impairment in their capacity for insight or in controlling their actions and at the same time therefore are a danger for themselves or others, they can be compulsory admitted and/or treated against their will in a psychiatric hospital, a procedure strictly regulated by specific laws. This may be the case for example while being in a delirium due to alcohol withdrawal, or being in a psychotic delusional state of mind, e.g. in schizophrenia, or being suicidal in a severe depressive episode. After the first effective psychotropic drugs were introduced to the market, the pharmaceutical industry gained a great deal of influence in the field of psychiatry. As we know today, drug effects were exaggerated, results presented far too positively, side effects were played down, and frequently all of this was done with criminal intent (cf. for example Hasler 2013). Against this background, it is not surprising that widespread mistrust in medication had developed as well as public reservations about the shere possibility of compulsory admission and treatment. Despite all of these negative aspects, psychiatric care in Germany has improved dramatically in recent decades. There is often very good inpatient care, a dense network for outpatients, excellent funding compared to many other countries, complementary facilities and care structures, a more modest use of drugs has developed with using lower doses, and a wealth of evidence-based psychotherapies for almost all mental disorders is now available. Nevertheless, one must be aware of this historical context when looking at classification and diagnosis in order to be able to properly understand some underlying controversies in the field of psychiatry.

9.1.2 Construction of a General Concept of Disease and Disorder from a Philosophy of Science Point of View

The question of what disease and health *are* arises not only in psychiatry, but in medicine in general. The medical ethicist Peter Hucklenbroich from Münster, Germany, provides a sound construction of the general concept of disease in biomedicine from a philosophy of science point of view (Hucklenbroich 2012). First, he distinguishes four levels of the concept of disease, disorder or pathology (in German: Krankheit). The first is personal and related to the life-world of a person (person X is sick). On the second level, a distinction can be made between healthy and pathological life processes (process X is pathological). On the third level, reference is made to a normal model of the human organism (X is pathologically altered), on which the pathodisciplines are based (pathophysiology, pathobiochemistry and psychopathology). Only at the fourth level disease entities and categories are postulated (X is a disease). These entities denote either individual diseases (influenza, myocardial infarction, femoral neck fracture, alcohol withdrawal delirium) or categories (e.g., cystic kidney

disease, tachyarrhythmias). This distinction of levels is helpful in better understanding some of the discussions in the field. For example, it is popular to claim that there are no diseases at all, only sick people. This sounds good, but mixes up the first and fourth level and does not change the need to look for correlations, mechanisms and causes at level two and three.

Life processes to which the four criteria apply can be said to be pathological (in German: krank): They are conditions, processes or procedures

1. of or within individuals,
2. that can be attributed to the organism, not the environment,
3. which exist and develop independently of the will and knowledge of the individual organism, and
4. to which there exists at least one non-pathological alternative course.

But what exactly is pathological and what is not? Here it is important to distinguish between positive and negative disease criteria (■ Table 9.1).

Clearly, this list of criteria is very general and includes some critical formulations that have been discussed again and again in the history of psychiatry, especially the fourth and fifth positive disease criteria. The first negative criterion also has been discussed again and again in view of the possibilities of modern medicine. But at least this set of criteria provides a blueprint for assessing the relevance of the biomedical model of disease in psychiatry.

Another helpful distinction is to distinguish secondary and tertiary pathological features from primary pathological ones. Secondary pathological features occur as a result of primary pathological processes and do not otherwise occur in the organism, e.g. fever, redness or swelling, scarring. Accordingly, in psychiatry, some symptoms may be only secondary pathological. Tertiary pathological features are not pathological in the first place, but can be considered pathological because they are a causal result of other pathological processes. An example would be pathological short stature compared to people who are simply short within the normal variance. In psychiatry, this may apply, for example, to certain forms of social behavior that may be very similar to normal variation but have different causes, for example "normal variants" (shyness) and social withdrawal (in the case of pronounced social phobia or in the context of schizophrenia).

Finally, the concept of disease entitities postulates that there are individual diseases that can be distinguished from each other. The system of disease entitities is referred to as "nosology", whereas the doctrine of disease causes is called "etiology". In medicine, five dimensions of diseases are typically distinguished: the nature of the initial cause, the nature of the subjects potentially affected, the nature of the effect of the cause on those who are affected by the disease, a specific pathogenesis and pathophysiology, and a time characteristic of the course and signs of the disease. This can be well spelled

■ **Table 9.1** Positive and negative disease criteria. (According to Hucklenbroich 2012 with changes)

Five positive disease criteria	Two negative disease criteria (= not sick)
1. Lethality 2. Pain, discomfort, suffering 3. Disposition for 1 or 2 4. Inability to reproduce 5. Inability to live together	1. Universal occurrence and inevitability, e.g. sex, intrauterine and ontogenetic phases, pregnancy, menopause, age, natural death 2. Knowingly and willingly self-induced behaviour (provided self-determination is not impaired), such as value judgements, risky behaviour, abstinence, deliberate lying, reflected suicide ("German: Freitod")

out by the example of a respiratory infection or the radius fracture *loco classico* (cf. in detail Hucklenbroich 2012). As a final aspect, it should be pointed out that, once established, treatment methods can of course also be used to treat non-pathological conditions in the above sense, such as age-related complaints, unavoidable pain conditions (childbirth, teething pain), prophylaxis or protection against social disadvantages due to physical stigmas, on request (cosmetic surgery) or for life and family planning (contraception, induction of childbirth, sterilization). Applied to psychiatry, this means that one can of course also "treat" life problems, which are not diseases or disorders, with psychotherapeutic techniques.

9.1.3 Current Definitions of Mental Disorders

From the Biomedical to the Biopsychosocial Model

In the early days of psychiatry, the leading model was the biomedical one. Mental diseases (disorders), as the then 28-year-old Wilhelm Griesinger put it in his textbook "Die Pathologie und Therapie der psychischen Krankheiten, für Aerzte und Studirende" (The Pathology and Therapy of Mental Diseases, for Physicians and Students) as early as 1845, are brain diseases. This claim did not stand in contrast to his own, quite progressive social psychiatric approach. Subsequently, psychiatrist tried to find causes for mental disorders according to the respective state of knowledge and the available methods of their time by looking for infectious causes (prime example: Treponema pallidum as causative agent of progressive paralysis), histological changes of the brain (heyday of neuroanatomy at the beginning of the twentieth century), genetic factors (hereditary theory), neurotransmitter dysfunction in the brain (discovery of psychotropic drugs) or in anatomical and functional connectivity changes of the brain (with the emergence of functional neuroimaging some decades ago). Interestingly, one of the main proponents of anti-psychiatry, Thomas Szasz, also follows the biomedical model of disease. In accordance with this model, he argues that mental illnesses would be "real" illnesses if one could identify a clear neuropathology, as in neurology. But since this is not the case, or so he argues, mental illness is a "myth" (Szasz 1961) that falsely categorizes common life problems as illnesses. In his 1961 book, however, he does not take on schizophrenia or depression, but discusses hysteria, as popularized by Charcot around 1900 (among other things, Freud attended Charcots clinical demonstrations), as a prime example. Today, the clinical picture of hysteria is categorized as a "dissociative disorder" in modern classification systems, and conceptualized as a mainly psychogenic disorder, and plays only a very marginal role in psychiatry. However, there are recent studies on dissociative disorders that attempt to identify the neurobiological mechanisms involved in the etiopathogenesis of "hysteria" (Boeckle et al. 2016; Schönfeldt-Lecuona et al. 2004). Nowadays, it seems self-evident to us that psychogenic diseases must also be manifest in the brain in some or the other way (cf. the preface in Walter 2005).

Furthermore, today we take it for granted that, in addition to genetic predispositions and neurobiological factors, life experiences, psychological processing and social factors also play a role in the development of mental illnesses and disorders. This was not always so clear, because the narrow biomedical model had no place for social, psychological and behavioural mechanisms. Their relevance was effectively postulated only by the historian of psychiatry George L. Engel in his biopsychosocial model, now cited in virtually all psychiatry textbooks (Engel

1977). It is closely related to the vulnerability-stress or stress-diathesis model. These models state that we are all endowed with a greater or lesser degree of vulnerability which, under the influence of 'stressors' on our experience and behaviour, can result in us becoming ill. Or to put it even more simply. Mental illness is always a combination of predisposition and environmental influences. However, this statement is so general that it is almost trivially true. Moreover, it says nothing about what a mental illness is, but rather something about its etiology, that is, how it comes about.

Definitions of Mental Disorders

How do the two major classification systems of psychiatry, the ICD (International Classification of Diseases) of the WHO (Dilling et al. 2015) and the DSM (Diagnostic and Statistical Manual of Mental Disorders) of the American Psychiatric Association (APA 2013) define mental disorders? The ICD classification of diseases evolved from the need to categorize diseases for death statistics. It is now of central administrative, and therefore financial and statistical, relevance in billing with health insurers in both Germany (ICD-10) and the USA (ICD 9). The fifth chapter of ICD-10 covers "Mental and behavioural disorders". The title itself suggests that it is apparently not always clear what exactly is mental, and that it is sometimes easier to simply classify behaviour. The DSM, also called the "bible" of psychiatry, is a manual that is much more comprehensive than the ICD-10 and contains detailed scientific explanations of the individual clinical conditions. It only attained a far-reaching significance in its third version of 1980 (DSM-I: 1952, DSM-II: 1968) On page 20 the following definition of a mental disorder can be found:

> A mental disorder is a syndrome characterized by a clinically relevant disturbance in an individual's cognition, emotion regulation, or behavior that indicates dysfunction in the psychological, biological, or developmental processes underlying mental functioning. Mental disorders are usually associated with significant distress or disability in social, occupational, or other important activities. An expected or culturally recognized reaction to a common stressor or loss, such as the death of a loved one, is not a mental disorder. Socially deviant behavior (e.g., political, religious, or sexual) or conflict that exists primarily between the individual and society is not a mental disorder unless the deviance or conflict results from dysfunction in the individual as described above. DSM-III, 1980.

This definition is not as bad as its reputation. Similar to Hucklenbroich, it contains both positive and negative criteria. However, it is very broad, so that it is not surprising that the demarcation of non-pathological psychological problems from disordes is not always easy.

A rather narrow definition of the term *clinically relevant* mental disorder can be found in Heinz (2015). He distinguishes three aspects associated with different concepts of "disorder":

- the medical aspect, i.e. in the broadest sense pathophysiological, objectifiable functional disorders (*disease*),
- the subjective feeling of being *ill* (*illness*), i.e. the aspect of suffering
- finally, impairments in the way of life or social participation (*sickness*).

Only when all three aspects are present, it is suggested, can there be a relevant mental illness. This definition is compatible with Wakefield's (1992) classical theory of illness, which focuses on the concept of "harmful dysfunction," although Wakefield takes a more evolutionary approach. For Heinz, only dysfunctions that affect mental functions relevant to surviving should be classified as diseases, such as dysfunctions of alertness, orientation, reasoning ability, memory, delusion, or the loss of affective vibratory capacity. The advantage of such a narrow definition is that it covers all severe mental disorders such as dementia, delirium, paranoid-hallucinatory schizophrenia or severe depression; however, many other disorders, in particular personality disorders, can no longer be regarded as diseases without further ado.

The meaning of dysfunction as well as the consideration of the clinical relevance or severity of a disease makes it clear that normative aspects play a role in the classification of a dysfunction as a disease. At this point, it should be noted that the concept of "normality" can occur in at least three meanings. Often, it is used in a prescriptive sense, i.e. as a setting or social norm. However, there are also norms and normality in a statistical sense (cf. the biostatistical theory of disease by Boorse 2011) and thirdly in a biological sense (function for the organism, so-called Cummins-functions) i.e. evolutionary normality, i.e. with reference to the history of the coming into existence of a function, also called *proper function*.

A very useful definition of mental illness has been provided by the philosopher Georg Graham. According to him, a mental disorder is "a disability, dysfunction, or impairment in one or more basic mental or psychological *faculties* (in the original: "*mental faculties or psychological capacities*") of a person that has harmful or potentially harmful consequences for the person concerned." (Graham 2010, p. 28). What is important in his theory is that the affected person does not necessarily recognise or acknowledge the harmful consequences themselves, she cannot simply control the condition and the condition cannot be made to go away simply by using additional psychological resources, e.g. by simply 'pulling oneself together'. Graham also makes a distinction from typical neurological diseases such as Huntington's disease (genetic defect), Alzheimer's disease (neurodegenerative disease), or encephalitis (inflammation of the brain). Whereas in these cases the disease, which may well also include mental symptoms, is caused by a direct affection of the brain, i.e. through the "brute forces of the neurological", according to Graham in mental disorders the mental is always involved in the genesis of the disease via intentional or conscious processes.

Of course, the question arises here as to what is the nature of intentional or conscious processes—but we can put that question aside here as long as we assume that what is meant by this is not some ominous substance that cannot be grasped scientifically, but a particular kind of natural process that constitutes the mental and for which the brain plays a central and indispensable role.

In the box: Working Definition of Mental Disorder, a working definition for mental disorders is proposed that attempts to preserve the insights of the above theories and closely follows Hucklenbroich's general theory of illness.

Working Definition of Mental Disorder

A mental disorder is a (P1) mental dysfunction, i.e. a disability, disorder or impairment of one or more of a person's basic mental faculties that (P2) results in clinically relevant subjective distress or discomfort and thus (P3) impairs everyday skills of living in a clinically relevant way. It is (N1) not controllable by simple volition or reasonable effort, (N2) not an unavoidable universally occurring process (such as exhaustion, separation stress, or fear of pain), (N3) not an expectable and culturally recognized response to stressors or loss (such as grief), and (N4) not simply a deviation from social values, preferences, or behaviors (e.g., political, religious, or sexual) unless it is secondary caused by one or more independent mental dysfunctions.

This working definition contains three positive (P) and four negative criteria (N). The latter are mainly used to demarcate much discussed "simple" problem cases such as "reasonable fears" grief, homosexuality or political and religious beliefs. The definition does not contain a clear demarcation between disease or simple dysfunction. The simplest demarcation can probably be made by severity. It also does not contain a clear demarcation between a mild mental disorder and severe life problems. The reason for this is simple: there simply is no clear boundary, even though there are clear examples at the ends of the spectrum, i.e. of severe illness on the one hand and clear mental health on the other. Normative and societal factors play an elusive role in drawing the boundary, as will become clear from the discussion of pathological grief below. The exact demarcation is determined by too many theoretical and social factors that make it impossible to give clear boundaries in a definition.

9.2 How To Diagnose a Mental Disorder?

The clinical diagnosis of a mental disorder is made in a similar way as with other diseases and disorders, i.e. by taking a medical history, objective additional tests (laboratory values, brain imaging—in psychiatry, however, usually only to exclude "organic" causes of the complaints, such as neurological or medical diseases), the systematic assesment of the psychopathological status, the consideration of the family history and the observation of the clinical course. With the exception of Alzheimer's dementia, there is no single mental disorder for which objective additional diagnostic findings from the laboratory or imaging exist to objectively confirm or prove a clinical diagnosis. Many patients believe that neurotransmitter deficiencies can be measured, but this is not the case. The major diagnostic tool of the psychiatrist therefore is the psychopathological status, cross-sectionally as well as longitudinally.

Today, standardized procedures for assessing psychopathology exist (i.e., alertness, orientation, memory, formal and content-related thought disorder, affective symptoms, etc.), which will not be presented in detail here; see the respective chapters in this book. Instead, two central concepts for a theory of disease will be briefly discussed: Validity and Reliablity. The validity of a diagnosis means that what is diagnosed actually exists. The problem here, of course, is where the *ground truth* is, that is, how we know that a disease is present. In general medicine, the validity often only could be confirmed by an autopsy, i.e. the (histo-) pathological findings. Nowadays, outside of psychiatry, modern medicine has a variety of objective parameters or biomarkers, laboratory tests, biopsies, or imaging techniques. As already mentioned, these objective measures usually do not exist in psychiatry in

such a way that they could be used in clinical routine (or not yet, see below).

Therefore, since the publication of the DSM-III, great emphasis has instead been placed on reliability, i.e., the reliability of a diagnosis (independent of its validity)—in other words, whether two independent examiners arrive at the same diagnosis for the same patient. Whereas prior to the DSM-III, psychiatry was dominated by the triadic system (exogenous ("organic") disorders with known physical causes, endogenous ("internal") disorders with as yet unknown physical causes, and psychogenic disorders) based on a theoretical nosology, the DSM-III marked a clear shift to a descriptive approach. Experts sat together and formulated diagnostic criteria (see the individual disease chapters) so that a diagnosis could be made reliably by determining how many symptoms of a syndrome had to be present over what amount of time for a diagnosis to be made. This approach is descriptive in that it is neutral to the question of what gives rise to such a defined syndrome or what its causes are (exception: neurological and internal causes of an "organic" mental disorder). Thus, in the past, a distinction was made between endogenous depression (comes from within, without an external cause, must be treated with drugs), neurotic depression (has its cause in early conflicts, requires psychotherapy) and reactive depression (is caused by an external event such as death, divorce, job loss, or partner problems). Nowadays, depression is diagnosed only on the basis of number of symptoms and course, and the severity rather than the (assumed) cause is relevant for the type of treatment. We will discuss related problems below.

It was this approach that allowed for reliable diagnosis, epidemiological studies and comparisons between regions and countries, as we will discuss in the next section. It should be noted that a close co-evolution of DSM and ICD has occurred at least since the publication of DSM-III.

9.3 What Mental Disorders Are There and How Common Are They?

Due to the diagnostic criteria of the ICD-10, which have been trimmed for reliability, there are now very reliable studies on the frequency of mental disorders. Probably the most comprehensive and highest-quality study currently available for Europe was conducted in 2011 (Wittchen et al. 2011). In this study, systematic reviews, reanalysis of existing datasets, national surveys and consultations with experts were used to determine the frequency of diagnoses based on the ICD-10 within one year in 27 EU Member States (EU-27) as well as Switzerland, Norway and Iceland, together accounting for a good 500 million people. Accordingly, in 2010, more than one third of the European population met the diagnostic criteria for at least one of 27 mental disorders, more precisely 38.2% or roughly 165 million people. The absolute and percentage frequencies for the most important individual disorders according to ICD-10 are shown in ◘ Table 9.2.

Of great interest here is the question of whether mental disorders have increased in frequency, as it repeatedly has been claimed. In 2005, Wittchen and Jacobi (2005) published a study that used the same methodology. At that time, only 13 diseases were examined. Five years later, there was no increase in the frequency of diagnosis for these disorders (2005: 27.4%. 2010 27.1%). The increase in mental disorders often reported in the media usually is due to other factors, such as the number of sick leaves counted by health insurance companies due to mental disorders. These have indeed increased (Wittchen and Jacobi 2005). However, in practice the diagnostic criteria are generally not checked as strictly as in the surveys on which the Wittchen study is based. At best, therefore, it can be stated that the number of sick leaves has increased,

Table 9.2 Frequency of mental illnesses in Europe 2010 (EU-27 plus Switzerland, Iceland, Notwergen) from Wittchen et al. 2011. The three disorders printed in bold are core disorders of clinical psychiatry (schizophrenia, bipolar disorder, major depression). Disorders that were only included in the 2010 survey but not in 2005 (Wittchen and Jacobi 2005) are printed in italics

ICD-10	Category	Diagnosis	Frequency	Absolute number
F00-09	Organic, including symptomatic mental disorders	*Dementia*	1.2%	6.3 million
F10-19	Mental and behavioural disorders due to use of psychotropic substances	Alcohol Opioids Cannabis	**3.4%** 0.1–0.4% 0.3–1.8%	**14.6** million 1.0 million 1.4 million
F20-29	**Schizophrenia**, schizotypal and delusional disorders	**Psychotic disorders**	**1.2%**	**5.0 million**
F30-39	Affective disorders	**Depression** **Bipolar disorder**	**6.9%** **0.9%**	**30.3 million** **3.0 million**
F40-48	Neurotic, stress related and somatoform disorders	Panic disorder Agoraphobia Social phobias Generalised anxiety disorder, Obsseve-compulsive disorder, somatoform disorder, Post-traumatic stress disorder	1.8% 2.0% 2.3% 1.7–3.4% 0.7% 4.9% 1.1–2.9%	7.9 million 8.8 million 10.1 million 8.9 million 2.9 million 20.4 million 7.7 million
F50-59	Behavioural syndromes associated with physiological disturbances and physical factors	Anorexia Bulimia *Insomnia* *Hypersomnia* *Narcolepsy* *Sleep apnea*	0.2–0.5% 0.1–0.9% 3.5% (7%) 0.8% 0.02% 3.0%	0.8 million 0.7 million 14.6 (29.1) million 3.1 million 0.1 million 12.5 million
F60-69	Personality and behavioural disorders	*Borderline PD* *Dissocial PD*	0.7% 0.6%	2.3 million 2.0 million
F70-79	Mental retardation	*Mental retardation*	1.0%	4.2 million
F80-89	Developmental disorders	*Autism*	0.6%	0.6 million
F90-F98	Behavioural and emotional disorders with onset in childhood and adolescence	*ADHD* *Behavioral disorders*	5.0% 3.0%	3.3 million 2.1 million
F99-F99	Unspecified mental disorders	Remainder category		

but not the number of scientifically established diagnoses. This probably has to do both with the increased willingness to recognise and diagnose mental illness in the first place, and possibly also with the overly hasty attribution of a diagnosis.

However, the number of diagnoses alone says nothing about their clinical relevance, since all degrees of severity are combined in one category here. Gallinat et al. (2017) have presented the clinical reality in Germany much more realistically in terms of severity. According to these authors, 90% of all mental disorders are mild to moderate and only 10% of all mental disorders are severe, of which, surprisingly and depressingly, half are among adolescents between 13 and 17 years of age. The reality of care for these groups is as follows. The mild and moderate disorders are dominated by older people with depression, anxiety, stress and somatoform disorders. About 95% of them are in outpatient psychotherapy. Treatment is poorly managed, there are waiting times for psychotherapy of 3–9 months and thus a backlog, including those in need of an inpatient treatment. Patients in this group account for 90% of all days of incapacity to work, with direct costs (2014) of €8.3 billion and €13.1 billion in gross value added.

In contrast, the severe disorders are dominated by the schizophrenia spectrum, bipolar disorders, borderline personality disorder and psychotic depression. The risk factor migration plays a major role there. Only 3–5% of these patients are in outpatient psychotherapy, and they cause only 5–10% of all days of incapacity to work, since most of those affected are not employed or not able to work. Many of the patients are revolving door inpatients, meaning that they come back again and again or they live in therapeutic and long-term facilities or are housed in forensic institutions. This group accounts for 60% of all emergencies and 80% of all compulsory admissions. It shows high morbidity and mortality with an average life expectancy of only 55 years. The direct medical costs caused per case are high, averaging around €45,000 per year. This reality of care makes it clear, among other things, why there are such different views, even within professionals, on the reality of psychiatry and the incidence of mental illness, where everyone feels confirmed by their everyday experience in their own field of daily work.

9.4 Problems with the Classification of Mental Disorders

In the following, the problems with the classification of mental illnesses will be shortly explained. I will primarily refer to the DSM, since most of the literature refers to it, but the problems described apply equally to the ICD-10.

9.4.1 Heterogeneity

As explained, the current classifications are descriptive, i.e. a diagnosis is based on characteristic syndromes and is made when a number of specific symptoms are present for a certain period of time. For example, a diagnosis of depression according to DSM-5 is made when at least five of nine symptoms are present for most of the day for a period of at least 2 weeks, and one of the symptoms must be (1) or (2). At first, this sounds very plausible to any clinician. However, the question arises as to whether the diagnosis of depression is really a single clinical picture. Thus, it is possible for two individuals to be given the same diagnosis (depression) without having a single symptom in common. (◘ Table 9.3).

Theoretically, there are 227 unique symptom combinations that all lead to the same diagnosis; if one also takes into account that there can be too much or too little of sleep, appetite or psychomotor function, there are

Table 9.3 Possibilities for making a diagnosis of depression according to DSM-5 without a single overlapping symptom. (After Pawelzik, unpublished, with kind permission)

Mr. Miller	Mrs. Schmidt
(1) Depressed mood	(2) Loss of interest or pleasure
(3) Loss of appetite or weight	(3) Increase in appetite or weight gain
(4) Insomnia	(4) Hypersomnia
(5) Psychomotor agitation	(5) Psychomotor retardation
(7) Feelings of worthlessness or inappropriate guilt	(6) Loss of energy
(9) Thoughs of death or suicide	(8) Decreased concentration

even 945, and if one takes into account the sub-symptoms, there are even 16,400. Now, one could assume that this is just a theoretical consideeration, but that most depressions are very similar. This was examined empirically by Fried and Nesse (2015) using one of the largest treatment studies of depression (n = 3703), the so-called Star*D study. Using a symptom list (QIDS 16), 1030 unique symptom profiles emerged empirically with an average of only 3.6 individuals per profile. 501 symptom profiles (48.6%) existed in only one patient and 864 profiles (83.9%) included only 2–5 individuals. Thus, it is empirically apparent that there is a great deal of heterogeneity in depressive disorder. Time and again, it has been investigated whether there might be distinct subtypes that can be characterized on the basis of common symptom profiles, but all these attempts have so far not been supported by convincing evidence.

9.4.2 Demarcation Problems

For mental disorders, there are at least two demarcation problems. First, "normal" depression must be distinguished from so-called "organic" depression, e.g. depression after stroke, in Parkinson's disease or in medical diseases, e.g. thyroid disorders, or as side effects of medication (e.g. cortisone) or drugs (e.g. after esctasy consumption). This so-called exclusion diagnosis of other primary diseases that secondarily lead to a psychiatric syndrome is an obligatory part of every diagnostic process. It also includes the distinction between neurological and psychiatric disorders, which is obsolete for some disorders (dementia) but useful for others (▶ Sect. 9.1). Second, a much more difficult problem is the demarcation from normal psychological processes or life problems. A much discussed example is grief following the death of a significant other (Wakefield 2015). Thus, after the death of a life partner or even a child, it is not surprising to feel despair, to cry, to doubt the meaning of life, to feel no more pleasure, to have reduced drive, in short, to grieve. Looking at the symptoms alone, a diagnosis of depression can be made easily. But of course it is normal and natural to grieve after a significant other dies; indeed, not to grieve would be rather unnatural or even pathological. The authors of the DSM were well aware of this life problem. That is why in the DSM-IV (1994) the so-called *bereavement exclusion existed*. After a bereavement, it was only

allowed to diagnose depression only 2 months after the event at the earliest. With the introduction of the DSM-5, this *bereavement exclusion* was dropped. Why? The argument for this decision was that this exception would make it impossible for people who were grieving and in the process developed depression to be diagnosed, and thus impossible to receive treatment, since therapy would only be paid for by health insurance companies if a diagnosis was made. Another argument was that if one sticks with the *bereavement exclusion*, it does not seem plausible to define only death as an exception. After all, isn't it normal to have depressive symptoms when your partner leaves you, you lose your job, or your home is destroyed by a fire? So either the exception should be extended to include such other cases or it should be consistently dropped. Opponents of the abolition argued that the possibility of diagnosing depression only 2 weeks after a death would pathologise normal psychological processes and lead to an unjustified inflation of diagnoses.

This discussion also has a scientific part. First there have been attempts for a long time to establish so-called prolonged or complicated grief as an independent clinical condition (Wagner 2014). Second, empirical studies exist, e.g., by Wakefield, the theorist of mental disorder as *harmful dysfunction*, that there are uncomplicated depressions, i.e., conditions that although meeting the diagnostic criteria of depression cross-sectionally do not show an increased likelihood for future depressive episodes longitudinally, and thus should be considered to be benign depressions (Wakefield and Schmitz 2014). These are characterized as single episodes that resolve within 6 months, do not cause severe impairments, and are not associated with psychotic symptoms, suicidal ideation, psychomotor slowing, or feelings of worthlessness. Now, is this a "benign" depression or a "normal" psychological process? This question is difficult to answer or to decide by definition. Fortunately, however, a discussion of this issue can now be based on empirical data.

A third demarcation problem is to distinguish between between different types of mental disorders. According to DSM-5, mental disorders are defined categorically, i.e. there is a disorder or not. But this creates the problem of comorbidity. Often several disorders are present at the same time. For example, there is a close comorbidity of depression and anxiety disorders or of addictive disorders and depression. Is this really a case of the presence of two different disorders? Or is there not rather a connection between both disorders, or even a causal relationship? Someone who suffers from an addictive disorder could, for example, become secondarily ill with depression because he suffers from the consequences of his addictive disorder. An inverse relationship is also conceivable. For this reason, it was also considered in the DSM-5, especially for the personality disorders, to introduce a dimensional approach to mental disorders instead of a categorical approach. This means that a mentally ill person may present with symptoms in different dimensions that are more or less pronounced, instead of being diagnosed with different disorders. Such an approach has been already elaborated in the field of personality disorders, but has not yet gained wide acceptance.

9.4.3 The Problem of Biomarkers

The heterogeneity of purely symptomatically defined mental disorders has always been an argument for including neurobiological findings in the definition or diagnosis, as has now been achieved with cerebrospinal fluid diagnostics for the diagnosis of Alzheimer's disease, which shows high sensitivity and specificity. This is the promise of biological psychiatry. And indeed, it was a promise for the transition from DSM-IV to DSM-5. Yet neurobiologi-

cal criteria found virtually no entry into DSM-5 diagnoses. Why? The answer is simply that despite the wealth of neurobiological research and knowledge, there are virtually no clinically useful biomarkers—with a few exceptions, such as Alzheimer's disease. Take schizophrenia, for example: with such a severe and relatively uniform clinical picture worldwide, one would think that the chance of finding one or more reliable biomarkers should be quite good. Prata et al. (2014) investigated this empirically by performing a detailed analysis of all papers on biomarkers in psychosis (n = 3200). About half of the studies were related to diagnostic biomarkers, a quarter were reviews, and fewer than 200 papers were longitudinal studies. For the latter, the authors examined whether genetic, metabolic, or imaging markers were predictive of treatment outcome. They assessed the quality of the biomarkers based on quality (positive outcome, controlled trial, a priori definition of biomarker, sufficient statistical power, independent replication) and effect size with a maximum score of 8. The result?

> The only biomarker with a final score above 6 from the total of 362 predictive & monitoring biomarkers in the 114 studies was a pharmacogenetic biomarker that scored 7: the C allele of the 6672 G > C single nucleotide polymorphism (SNP) in the HLA-DQB1 region (Athanasiou et al. 2011) predicted risk for clozapine-induced agranulocytosis with an O.R. 16.8, was defined a priori, and its effect replicated in an independent sample. (Prata et al. 2014, p. 138).

In other words, the results of decades of biomarker research are very disappointing. Critics of biological psychiatry see this as an argument to stop doing this kind of research. Biologically oriented psychiatrists, on the other hand, argue that this merely shows that it is unlikely to find consistent biomarkers for purely clinically defined, heterogeneous syndromes as found in current classification systems (cf. on this ► Sect. 9.5.1).

9.4.4 Non-medical Interests

Another problem in the diagnosis of mental disorders is non-medical interests. On the one hand, this refers to financial conflicts of interest (cf. Hasler 2013) and the resulting distortions of nosology. Many co-authors of the DSM had consulting contracts with the pharmaceutical industry. The industry has an interest in defining, some say inventing, novel disorders in order to create new markets for drugs. Finally, there are individual, not financial, interests. For example, in the committees that drafted the DSM, some researchers fought for their own favorite disorder that was object of their own research activities for years or decades to be officially included in the DSM. Which, of course, is easier than in other medical disciplines when there are no objective biomarkers anyway. However, there are also stakeholders who have non-medical interests, e.g. because they fear losing financial advantages (e.g. through the omission of Asperger's syndrome from the DSM-5), or because they insist that a disorder has biological causes and not psychological ones (e.g. chronic fatigue syndrome), or simply because, from the perspective of those affected, the respective clinical syndrome naturally has a very high priority and, with ever more limited resources, lobbying for it is of high importance for them.

9.4.5 The Lock-in Syndrome

As already mentioned, the ICD forms the basis for the medical care system. Only with an official diagnosis do health insurance companies pay for therapies, you are eligible for an official notification of illness for the workplace, or have a chance of having an occupational illness recognised or receiving a pension. That is why, according to philosopher Rachel Cooper, it is almost impossible to fundamentally reform the DSM or the ICD (Cooper 2015). This is because any

change would have existential consequences for those in the existing care system and would destabilise a complex, constantly used and deeply embedded system. This, she argues, is similar to the QWERTY keyboard on a computer. Originally, the arrangement of the keys was due to the fact that the levers of a typewriter where supposed not to get stuck while typing. To establish a different arrangement of the keys, which would be more suited from a purely technical point of view on the computer, which has no more mechanical levers, is today however practically impossible, since a change of the system would be such a large expenditure that this will never happen: The system is in a "lock-in state". The only option would be a radical system change. In the computer realm, for example, this might be achieved by the development of voice input, i.e. speaking replacing manual input, i.e. typing. A similar radical change is proposed by the RDOC system (▶ Sect. 5.1).

9.4.6 The Mentalism Problem

Another fundamental problem that can only be outlined here is the mentalism problem (cf. Walter and Pawelzik 2018). It can be seen as a successor problem to the mind-body problem. As we saw in ▶ Chap 1, dualism has proved increasingly dispensable in the course of (Western) history. At the same time, the rejection of dualism by no means clarifies what the mental actually is, for instance in the biopsychosocial model. Hardly any of the theories of mental disorders address this question, but simply assume without further explanation that the mental or phenomenal experience is another level or aspect of reality. But what exactly is it? Is it simply identical with the neural, that is, with brain processes, as it would be natural to assume? A whole range of non-reductionist theories argue against this, e.g. by pointing out that brain states should not be ascribed properties on the personal level, that an "animate" organism also has a body, that mental states always develop in social interaction, are involved in a social and cultural context, are closely tied to language, at least in humans, and therefore mental processes must be regarded as complex, emergent phenomena. In the context of psychiatric controversies, however, three aspects of the mentalism problem are often mixed and not distinguished. The first aspect is the question of what exactly mental processes are. This is an *ontological* question about the nature of mental states. The second is the question of how we acquire knowledge about the nature of our experience and the content of normal (and pathological) mental processes. This is an *epistemiological* question. Thus, there is no contradiction in assuming that mental states are nothing more than brain states plus x (e.g., bodily states plus other external factors; on the externalism of the mental, see Walter 1997, 2018), but that we have access to the content of these mental states mainly and inevitably through subjective experience and socially grounded language. Such an assumption would explain why both, those who hold reductionist intuitions and those who point to the irreduciblity of personal experience feel justified. The third and ultimately most important aspect for psychiatry as a science is how mental dysfunction comes about. This question of the genesis of mental disorder and the causal factors relevant to it is neither identical with the ontological nor with the epistemological question alone. Although all three problems are related, they are conceptually independent of each other. For example, it is now generally assumed that a mix of causes, including genetic predisposition, neurobiological influences during the development of the organism, subjective experiences, stressors, and their processing, is relevant to most common mental disorders. Thus, when discussing mental disorders, we should always be careful to distinguish between ontological, epistemological, and causal issues, i.e.,

whether we are talking about the nature ("essence") of mental phenomena, how we can recognize them, and how they arise.

9.5 Recent Approaches

The difficulties with current classification systems and the lack of success of biological approaches to psychiatry have led to new proposals for changing and/or theorizing about the nature, study, and classification of mental disorders in the long run. Three such approaches will be presented here.

9.5.1 Research Domain Criteria (RDoc)

The RDoC initiative was launched in 2009, at the world's largest psychiatric research institute, the NIMH (National Institute of Mental Health) in Bethesda (Insel 2013; Kozak and Cuthbert 2016). Briefly, the issue was this (adapted from Walter 2017): Thomas Insel, then director of the NIMH, himself a researcher in the field of social neurobiology (functions of oxytocin and vasopressin), had always promoted neuroscientific research on mental disorders, from molecular biology to neuroimaging. With the introduction of the DSM-5 (2013), it was planned to also incorporate neurobiological findings into classification and diagnosis and to move from a categorical to a dimensional system. However, neither intention could be realized. This was for a variety of reasons, not least that the neurobiological findings were not robust enough to be incorporated into a clinically useful classification. This was unsatisfactory to many scientists, since much more was known about neural circuitry, including in humans, since the publication of the DSM-IV (1994), not least through non-invasive neuroimaging. For a long time, science-minded doctors had been dissatisfied with diagnosing mental illness only at the symptom level. But it is no wonder, says Thomas Insel, if biomarker research does not lead to success:

> » But it is critical to realize that we cannot succed if we use the 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 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 biomarkes because it does not detect a DSM category. We need to begin collecting genetic, imaging, physiologic, and cognitive data to see how all the data—not just the symptoms—form clusters and how these clusters relate to treatment response. (Insel 2013)

The RDoC system, which was made public in April 2013, therefore proposes that research into mental disorders should not be oriented towards (superficial) symptoms and syndromes, but should start from domains of neurocognitive functions based on the function of specific circuits, and map these at different levels (from gene to behaviour), in order to then use this database to classify the heterogeneous disorders into more specific disorders, independent of DSM criteria. Only constructs for which there was independent evidence of validity and for which knowledge about association with neural circuits were accepted as candidates for RDoC. The resulting constructs (currently 25) were sorted into six higher-level domains (Fig. 9.1) and also have subconstructs. Domains and constructs are not fixed and unmutable, but have been tentatively selected based on current knowledge. They can and should be refined, modified and extended by new empirical findings. For example, the sixth, sensorimotor domain has only recently been added,

NIMH Research Domain Criteria (RDoC)							
Functional domains (with neurocognitive constructs, without subconstructs)							
Negative Valence systems Acute threat (fear) Potential threat (fear) Persistent threat Loss Frustrating non-reward	**Positive Valence Systems** Reward responsivity Reward learning Reward evaluation	**Cognitive Systems** Attention Perception Declarative memory Language Cognitive control Working memory	**Social Processes** Affiliation and attachment Social communication Perception & understanding of the self Perception and understanding of others	**Arousal and Regulatory Systems** Arousal Circadian rhythms Sleep-wakefulness	**Sensorimotor Systems** Motor actions *Agency & ownership* (=agency & self-awareness) Habit (sensorimotor) innate motor patterns		
Units of analysis (levels)							
Gene	Molecule	Cells	Circuits	Physiology	Behavior	Self-reports	Paradigms

Fig. 9.1 The functional domains and neurocognitive constructs of the RDoC (as of June 30, 2019, retrieved from ▶ https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc/constructs/rdoc-matrix.shtml). Currently, there are six domains with a total of 25 constructs (see table) and 28 subconstructs (not listed here)

and a further domain "impulsivity" is being discussed.

All constructs can and should be systematically investigated at different levels (the RDoC authors prefer the term: units of analysis): from genes to molecules and cells to circuits, the physiological level (e.g. heart rate, cortisol level), observable behaviour and self-reports. To do this, different paradigms are used. Therefore, the RDoC matrix can be thought of as a two-dimensional table in which knowledge is collected. On the constantly updated RDoC homepage, one can look up what we currently know about each field in this table. Two other dimensions that are included in RDoC, though not in the matrix, and are relevant to all domains, are the developmental and the environmental dimension. This is because the functions mentioned above all emerge only in the course of an individual's development, and are shaped and modified by environmental influences (paradigmatic: epigenetic and learning effects).

The RDoC matrix provides a grid for the systematic study of mental disorders that is not fixed to previously defined disease categories. In the long term, the RDoC system is intended to contribute to a differentiated, and in some cases also novel, classification of mental disorders. Above all, however, it is intended to create the basis for better, tailor-made, ideally individualised therapy in the sense of *precision psychiatry*. Of course, the RDoC initiative has not been without criticism (cf. Walter 2017). What remains to be said is that it has had a significant impact on psychiatric research in recent years, as there is now a substantial body of work exploring and redesigning categories of mental disorders using this model. The RDOC approach can be succinctly summarized as "psychiatric research as applied cognitive neuroscience" (Walter 2017). A major extension of cognitive neuroscience is computational neuroscience, which has expanded into computational psychiatry within psychiatry (Friston et al. 2014; Heinz 2017, see for example ▶ Chap. 11 on schizophrenia in this book).

9.5.2 Network Theories of Mental Disorders

Another theory that has been discussed in recent years, not coincidentally, is the network theory of mental disorders (Borsboom 2017; Borsboom et al. 2019). It opposes the essentialist notion that mental health symptoms are "surface features" of an underlying pathological process, as in other diseases. Measles, for example, is clinically manifested by certain symptoms (rash, Koplik's

spots, fever), all of which are caused by a common cause, infection with the measles virus, and can thus be explained. The assumption that it is the same with depression, i.e. that the clinical symptoms of depression have a common cause (such as a lack of serotonin or a loss experience in childhood) is a misleading idea. Rather, the disease of depression consists (an ontological statement) of being a network of symptoms that are causally connected. For example, the symptoms of sleep disturbance, difficulty concentrating, ruminating, and self-worth problems are causally related. Those who sleep little are not rested the next morning, cannot concentrate, and have more time to ruminate, which can lead to thinking about their worthlessness. Conversely, a lot of ruminating can lead to insomnia. The pathological nature of depression is shown by the fact that the causal links between symptoms are so strong that a full-blown clinical picture is rapidly formed by an external event and causal interactions between symptoms, remains stable, and is difficult to become deactivated. A resilient network is characterised by the fact that although symptoms may develop, they quickly fade—due to the only weakly developed causal interconnections between the symptoms—and do not lead to a full-blown clinical picture of depression. A vulnerable network, on the other hand, only needs a trigger at some point, and then the symptoms spread by themselves, so to speak, and persist even if the external trigger is removed (Fig. 9.2). In network terminology, then, someone is mentally healthy with high resilience when the symptom network is weakly connected and there are few external stressors. With a weakly connected network and strong external stressors, there are increased symptoms, but the person is still healthy. A strongly connected network with few stressors indicates high vulnerability and with a strongly connected network with strong stressors, mental illness occurs.

Such a network approach differs from conventional theories of disorders in several aspects: For example, no common cause of mental illness is assumed; symptoms are given a crucial role. Symptoms are not merely counted independent of each other, but a causal nexus between them is postulated; a mental disorder is defined and described in terms of networks. Further, at

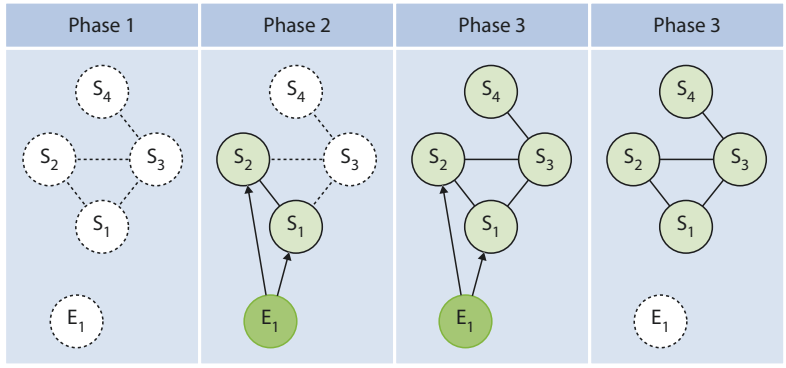

Fig. 9.2 Development of a mental disorder according to the network theory: In the first phase there are no symptoms (S), the network is "asleep" (phase 1). Under the influence of one (E1) or even several external events (stressors), individual symptoms are then activated (phase 2), which causes further symptoms to develop (phase 3). If the symptom network has strong connections, then recovery does not occur due to the omission of the external event. The external event has acted as a trigger and the network of symptoms keeps itself active and stuck in its active state. Similarly, it is conceivable that the activity of the network as a whole decreases as a result of therapy on individual symptoms. (After Borsboom 2017)

least according to the proponents of the network theory (Borsboom et al. 2019), neither disorders nor individual symptoms or their causal nexus can be attributed to causal processes in brain circuits, because the connections are too complex, because mental processes can be realized multiply, and because the focus on such details loses sight of what defines a mental disorder, namely the network properties of the symptoms, in the process. Thus, neurobiological reductionism is blocked. However, it remains a mystery how the different levels, such as those of neurobiology and mental symptoms and behavior, are connected. It is probably helpful to know that the founders of network theory are neither neurobiologically trained researchers nor clinical practitioners. Rather, they have their roots in psychiatric epidemiology and statistics, that is, in a field that deals with large, primarily clinical, data sets (symptom questionnaires) in large studies. It was mentioned at the outset that it is no coincidence that network theories are popular right now. Network theories are currently enjoying a lot of interest in various fields (social networks, neural networks, networks in physics). To date, however, there has been little contact or collaboration between epidemiological statisticians and neural network theories (Braun et al. 2018; Waller et al. 2018). Thereby, the attempt to bring together neurobiological and clinical levels using a common terminology and mathematical tools could be of high interest.

9.5.3 The New Mechanism

We have introduced two approaches that are in principle complementary to each other: While RDoC starts from basic neurocognitive processes and their brain circuits and neglects the level of symptoms, network theories focus on the symptom level and declare the underlying processes negligible. What both approaches have in common is that they want to move away from a purely descriptive approach and—in different ways—bring causal conditions into play. After all, identifying causal factors seems to be the fundamental hallmark of science, and both approaches emphasize this. In recent years, a new approach to understanding the brain has emerged in the field of philosophy of science in biology and neuroscience that is also of interest to psychiatry. This is the "new mechanism". For a long time, the philosophy of science was primarily concerned with theories in physics, in the form of quantifiable laws. One goal was to find as few as possible but fundamental general laws of nature to which other laws could be reduced. Another area of application for scientific theorists was the theory of evolution, since here, too, a general theory is available that claims to be able to explain the origin of life with a few general laws (mutation, selection, population dynamics). In the neurosciences, on the other hand, there is (as yet) no generally accepted "theory of the brain". Therefore, the new mechanism is devoted to the question of how concrete explanations in the neurosciences work. The result is a diverse mosaic of individual explanations rather than a general theory. The concept of mechanism has been identified as central to this (Craver 2007; Glennan 2017; Machamer et al. 2000). Phenomena are explained by identifying in detail the mechanisms that produce and sustain the phenomena, involving multiple levels and integrating different fields of science.

This approach is an approach of philosophy of science because it defines the concept of mechanism in an abstract and general way and then applies it in detail to specific phenomena in the field of neuroscience. However, mechanisms can also be found for mental (Bechtel 2008) and social phenomena (Hedström and Ylikoski 2000). A mechanism is defined as "a set of entities and activities organized to produce the phenomenon that is to be explained" (Craver 2007, p. 5). Entities are parts or components

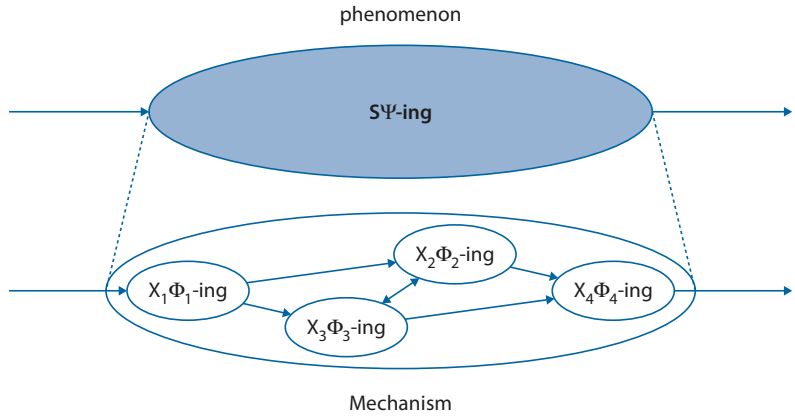

Fig. 9.3 The figure shows a phenomenon *Ψ* (pronounced: Psi) explained by a mechanism (*S*) (upper part of the figure). The lower part shows (abstract) details of the mechanism, namely entities (circles) and activities (arrows) that have a specific spatio-temporal organization. (After Craver 2007, p. 7); *t*, *S* = mechanism as a whole, *Ψ* (Psi) = phenomenon, *X* = entity or component, *Φ* (Phi) = activity

9

of the mechanism that exhibit activities that produce causal effects. Crucially, the organisation of these active components in space and time and in a hierarchy is what ensures that the mechanism produces the phenomenon. Craver (2007) goes through this extensively using the example of explaining neurotransmitter release and long-term potentiation. His general scheme for a mechanism has now become a canonical account (Fig. 9.3).

Already the abbreviation "Psi" in this scheme, which in philosophy often stands for psychological, indicates that the mechanism scheme is also meant to be applied to mental processes. However, there is much more about the brain than about the mind in Craver's writings, and nowhere is it stated exactly what the mental actually is (cf. ▶ Sect. 9.4.6 on the mentalism problem). For a detailed account of the new mechanism as an explanatory approach in cognitive neuroscience, see Kästner (2017). There, various problems with the approach are discussed, such as the question of the difference between the constitution of a mental process and its causal effects, how causal effects must be conceptualized across different levels, and what notion of causality is experimentally and conceptually relevant to neuroscience. Crucially for us at this point is, that the new mechanism is designed from the outset to move away from the physics centered approach in philosophy of science, and rather is concerned with real-world explanations of relevant phenomena in neuroscience, and has been conceived from the outset as a multilevel and integrative approach. Moreover, it refrains from the widespread correlationism that all too easily leads back to dualism, but claims to describe causal processes in the production of phenomena.

In a 2011 paper, psychiatrist and geneticist Kenneth Kendler, along with philosopher of science Carl Craver and philosopher of psychiatry Peter Zachar, made an attempt to use the new mechanism for a contemporary theory of mental disorders (Kendler et al. 2011; for another recent attempt, see Kästner 2019). In doing so, the authors take their cue from evolutionary biology approaches to explaining what a species is and apply them to types of disease. They somewhat awkwardly call their approach *mechanistic property cluster theory*, or MPC. According to MPC, disorders are like biological species, i.e. fuzzy "populations"

with paradigmatic central and more marginal specimens. One could also say that disorders are accumulations of (causally relevant) properties. In this context, different specimens of a species (disorders) do not share all properties with each other, but rather exhibit a family resemblance. In the high-dimensional property space of all (causally relevant) properties that contribute to their emergence, they are found close to each other, they "cluster". However, these properties are not surface properties (such as only symptoms), but the "co-occurrence of these properties between individuals is explained by causal mechanisms that regularly ensure that these properties are realized together." (Kendler et al. 2011, p. 1147). In this context, different levels can interact with each other to causally produce specimens of a species in the first place. Examples include epigenetic effects from stress, effects of substance abuse on the brain, or the effect of insomnia on depressed mood. Here, then, the authors combine elements of the new mechanism (mechanisms, that is, spatially and temporally organized active components causally producing a phenomenon, a disorder) and elements of network theories (symptoms may causally influence each other and this interplay may be part of the mechanism). Moreover, MPC is a multilevel theory and is able to integrate different theoretical approaches (biological, sociological, phenomenological) without giving up the claim to causal explanations.

In the introduction to their article, Kendler et al. (2011) categorize the types of diseases that have been theoretically explained as essentialist (a disease has an underlying essence as gold does, i.e., a single biological cause), socially constructed (invented for extra-medical reasons), and pragmatic (useful for the practice of medicine, such as the DMS-5). The MPC approach allows all kinds of mechanisms (biological, psychological, social factors, societal) as parts of a mechanistic explanation. Some disorders might be more essentialist (schizophrenia), others more socially constructed (hysteria) and some more constituted by social circumstances (anorexia nervosa). Only time will tell whether the MPC theory, which has not yet been worked out in great detail, will become accepted; it certainly will not under its complicated name. However, the new mechanistic approach will probably play an important role in any scientifically based psychiatry in the future.

Summary and Outlook

In the present chapter we have seen how difficult it is to define, diagnose and distinguish mental disorders from neurological diseases, life problems and from each other. Any theory of disorder in psychiatry will inevitably have to draw boundaries and live with the fact that those are fuzzy. Given the historical background of psychiatry, it is understandable why there are so many controversies about mental disorders. Any definition must contain both positive and negative criteria and must include the personal level refering to the person's ability to function and the subjective suffering component of the postulated dysfunction. At present, a system trimmed to reliability (DSM-5 and ICD-10 or -11) dominates the practice of psychiatry with all its advantages (no commitment to possibly incorrect etiological theories) as well as disadvantages (inflation of diagnoses, demarcation problems aggravated, a non-adaptive system with a "lock-in syndrome"). On the scientific and philosophical level, there are new approaches, all of which are more or less pluralistic and include different levels of explanation, but—in contrast to the DSM and ICD-10—place great emphasis on including causal mechanisms in the definition and explanation of mental disorders. None of these has yet become generally accepted, and it is possible that for different disorders different theories may be appropriate. In practice, however, the established diagnostic systems will persist for a long time. In the future, the inclusion of biological findings

will become increasingly standard, although it is hard to imagine that the central role of subjective experience in the diagnosis of most mental disorders will change.

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