

# The depression paradigm and beyond

## The practical ontology of mood disorders

*Ilpo Helén*

This paper is an analysis of three elements of which depression as the primary target of current Western psychiatry and mental health care is made: the quest of psychiatrists to identify a depressive disease proper; the category of 'major depression' as defined by the diagnostic manuals; and the epidemiological view emphasising risk factors of depression. These elements are pivotal to the present understanding and experience of what depression is since they delineate the space of reasoning in which claims about depression are presented, problematised, and disputed. The paper presents how these elements have historically evolved and coalesced, and how depression has been formed and transformed as an object of knowledge and treatment in psychiatry and how the claims about depressive disorders acquire objectivity in the current mental health discussions. The paper also demonstrates how the quest of depression as a neurophysiological disease, consolidation of Major Depression as the diagnostic core of mood disorders, and the central role of the epidemiological notion of risk are both interlocked and discordant with each other in the current depression paradigm. In addition, tendencies of subversion of the depression paradigm are discussed.

*Keywords:* psychiatry, depression, ontology of mental illness

Depressive illness has acquired a paradigmatic position in Western mental health thinking and practice during the past quarter of a century. Depression is considered the most common mental disorder globally, and it is estimated to become the second most severe public health problem worldwide by 2020 (WHO, 2001). It also epitomises how mental disorder or disturbance of psychological well-being is understood today by both professionals and the lay public. In this context, 'depression' provides a name and form to a common mental health problem that can be faced by anyone. Widespread

depression is also considered a major burden for economy since it reduces the capacity to work and productivity of the individuals all over the world (e.g. WHO, 2001). Moreover, depression epidemic is often seen as symptomatic of contemporary culture or 'the crisis of the self', as a kind of epochal disease (e.g. Solomon, 2002; Ehrenberg, 2010).

Depression as the paradigm of mental disorder—or talk and concern about it—is a composite of parallel yet divergent facts and definitions. This polyvalence is due to the dispersion of professional and public settings where mental health is discussed and taken

care of; it is an amalgam characterised by equivocality and contestation. In fact, 'depression' refers to a *problematization* and is thus capable of bringing together different aspects of contemporary mental health care, including all its tensions and fragmentation. (Helén, 2007a.) In this paper, I analyse the *epistemic* elements of which depression, the primary target of current Western mental health care, is made. My aim is to show how these elements have historically evolved, coalesced, and created the current notion of depressive illness, with its ambiguities and problems. My analysis is focused on the ways depression is understood as an object of knowledge and treatment in mental health care today. In other words, I study how we today conceive of depression as *something*.

The discussion on depression is certainly so voluminous that the point of reference of the term is vague. However, the following three notions are present in almost any account of depression: the quest of psychiatrists to identify a depressive disease proper; the diagnostic category of 'major depression'; and the epidemiological view emphasising the risk factors of depression. These three notions are pivotal to our understanding and experience of what depression *is* since they delineate the space of reasoning in which claims about depression are presented, problematised, and disputed.

I think of depression as a mental disease, diagnostic criteria of Major Depression and depression risks as the forms to understand and define maladies involving dejection or despondency by the mental health experts. Following Ian Hacking (2002: 178-199), the subject of my analysis is how depression is formed and transformed as *an object* of knowledge and treatment and how the claims about depressive disorders acquire objectivity in the current mental health discussions. The data for

my analysis is collected mainly from the realm of psychiatry (see below). However, the primary focus of my study is not the discussions at the forefront of psychiatric science and research. Instead, I concentrate on a particular kind of psychiatric discourse in which science is applied. As is well known, treatment of depressive disorders mainly falls within the domain of primary health care and is thus carried out by GPs. In this context, depression management procedures are highly standardized and rather homogenous all over the Western world. (Callahan & Berrios, 2005; Helén, 2007a.) Therefore, I focus mostly on a psychiatric discourse that facilitates the homogenization of depression treatment by giving guidance and setting standards to diagnostic and therapeutic practices in primary health care and for professionals with limited psychiatric expertise. I analyze an 'applied' discourse that creates a framework of thought in which depression becomes conceivable and its treatment gains a reasonable basis in current mental health care, disseminated in numerous contexts and institutions of health care, social services and education in which plethora of mental problems are encountered.

My study is based on analyses of three sets of research data. The first corpus of data consists of discussion on definition and classification of depression in Anglo-Saxon psychiatry, mainly in Britain, from the 1920s to 1980s. In addition, I have analysed the Anglo-Saxon psychiatric epidemiology and discussion on the management of depression in primary care from the late 1960s to 2000s, authorised guidelines included. Finally, the main corpus of my research material consists of papers and research reports on clinical aspects, epidemiology, and treatment of depressive illness in Finnish medical and psychiatric journals (*Psychiatria Fennica*, *Duodecim*, and *Suomen lääkärilehti*), textbooks, special

issues of journals, and guidelines and instructions for diagnosis and treatment of depression and for the use of antidepressants from the 1970s to the present day. Articles published in international psychiatric or medical journals by Finnish doctors are also included in the data. I have also used information from 8 interviews with Finnish primary care physicians, carried out in 2007 and 2008, to reflect the impact of psychiatric discourse to practices. The list of my data is attached at the end of the paper.

### Ontology in practice

Why do I focus my analysis in this manner? Definitions of what depression is and the objectivity of them are essentially formed and tested in practices and institutions for treatment of people suffering from depressive illness. For this reason, my analysis is focused on reasoning relevant for the public health and clinical practices, instead of manifold psychiatric theories of depression and mood disorders. Public health and clinical contexts are primary for the management of depression, therefore they are also primary epistemologically and ontologically.

Since I emphasize that discussion and knowledge over depression are embedded in practices of mental health care, I implicitly also claim that our understanding of depression is connected to health and social policy and practices of social control and regulation by public authorities and experts in our society. I have studied elsewhere (Helén, 2007a; 2007b; 2010; Helén, Hämäläinen & Metteri, 2011; Hautamäki, Helén & Kanula, 2011) the reciprocity of the emergence of the current notion of depressive disorder and the changes in the psychiatric institution and mental health policy or, in Jasanoff's (2004) terms, 'co-production' of an assemblage of mental health management and depression

as an object of knowledge. In this paper, the dimensions of social control and mental health politics are secondary and not systematically analyzed, while I concentrate on the epistemic aspects of formation of the depression assemblage. The subject of this paper is a history of the mode by which depression is known today, i.e. a history of depression as an epistemic thing, evolved with depression as a thing to be treated.

I approach depression as a mental disease, diagnostic criteria of Major Depression and depression risks by tracking their emergence and metamorphosis in a specific context of mental health thinking and practice. In the first two sections of my paper, I focus on the psychiatric discussion of *endogenous depression* that is a historical attempt and problem to define depressive illness as a clear-cut disease with definite physiological cause and course, in a manner similar to modern medicine. My discussion in these sections is based on the analysis of the discussion on definition and classification of depression in Anglo-Saxon psychiatry, mainly in Britain, from the 1920s to 1980s, and I situate my analysis in the historical context by reviewing research literature on the topic.

The next section highlights the most important element of the current depression paradigm, namely the diagnostic category of *Major Depression* in the DSM-III, DSM-IV and ICD-10 manuals. My discussion is primarily based on review and re-interpretation of research literature, but I also ground my argument on the analysis of the Anglo-Saxon and Finnish mental health discussion on depression from the 1970s to the present day. In this section, I particularly emphasise the role of the new nosographic rationale in psychiatry, aimed at stabilising diagnostic entities for clinical and research purposes, in creating the current diagnostic confusion in current mental health practices and facilitating the expansion of depression

into an epidemic proportion (see Helén, 2007b; Horwitz & Wakefield, 2007; Rose, 2006).

In the two sections to follow, the focus of analysis is on depression as a public health problem. My discussion is based primarily on the analysis of the Anglo-Saxon and Finnish psychiatric epidemiology of depression and the Finnish discussion on depression by mental health experts and medical authorities from the 1970s to the present day, and this analysis is put in a historical context by reviewing research literature on the topic. In particular, I discuss the implementation of the idea of risk into mental health thinking and practice by psychiatric epidemiology and the role of risk rationale in the treatment of depressive illness. A core result of my analysis is that epidemiology and the notion of risk have had pivotal impact on current mental health care and depression treatment rationale.

The topic of my discussion in the closing section on the paper is how the quest for depression as a neurophysiological disease, consolidation of Major Depression as the diagnostic core of mood disorders, and the central role of the epidemiological notion of risk are both interlocked and discordant with each other in the current depression paradigm. In addition, I also point out tendencies subverting the depression paradigm.

Since I study what depression is for us and how we conceive of it as an object, as something, I discuss *ontology* of depression in this paper. Following Ian Hacking (2002), I consider this ontology *historical*, which means that the ways depression exists for us or, rather, we think and feel depression exists, can change and is even bound to change. Further, the analysis of historical ontology of depression relates to historical and philosophical reflection or a critique of ourselves, our time and our world, our *condition humane*. Finally, historical

ontology of depression is about being a person, or moral subject, today:

Historical ontology [analyses] to what is possible to be or to do. (...) [It] is about the ways in which the possibilities for choice, and for being, arise in history (...) in terms of explicit formations in which we constitute ourselves. Historical ontology is not so much about the formation of character as about the space of possibilities for character formation that surround a person, and create potentials for 'individual experience'. (Hacking, 2002: 22-23.)

However, I do not study here the relationship between the concepts of the self and personhood within the depression paradigm or practices of the self under depression treatment. Instead, I focus on depression as a thing or an object. However, I do not assume that talking about endogenous depression, major depression or the risks for depression is a way to objectify the experience of depression. On the contrary, I claim that experiences become conceivable for us *as depression* only by becoming objects of mental health care, i.e. by becoming something to which objectivity can be attributed in the context of mental health thinking and practice. In other words, psychiatric objectivity is a prerequisite of our experience of depression.

This claim or assumption is based on an idea, inspired by Annemarie Mol (2002), that ontology of depression is *practical*. What is is not detachable of what is *done*, and that is the reason why I concentrate on discussions and reasoning relevant for public health and clinical management of depressive illness. This focus is also historically justified, because our time is characterised by the conviction that something can and should be done about persistent sadness,

low spirits or feelings of nothingness. Consequently, numerous efforts to detect, control and treat this 'depression' have arisen out of this therapeutic ethos. The requirements of treatment from diagnosis to cure assessment determine how depression is conceived of and defined, i.e. what we consider depression to be is based on the idea of its *treatability*.

All in all, my aim in this paper is to show how the conceptualisations of depression as a neurophysiological disease, as a diagnostic category of Major Depression, and as risk factors provide treatable objects for current practices and technologies of depression management and direct the actual treatment. I will also highlight the problematisations in which these three concepts of depression are involved, both as solutions to shortcomings of mental health thinking and practice and as sources of problems. My argument is that at the core of the both merits and perils of the current depression paradigm is the tendency to think of depression as well as other mood disorders as objects instead of as an experience with a specific context; and, as Emily Martin (2007: 220) says, 'movement toward thing-like status makes mania and depression seem possible to identify, manipulate, and optimize through the technology of psychotropic drugs and through taxonomic apparatuses.'

### Endogenous depression

In the current professional and lay discussions of depression, the term 'endogenous depression' is rarely used. Why then do I discuss it here? The reason is simple. Dispute over 'endogenous depression' provides a historical example of how two tendencies of psychiatric understanding of depression have interfered with one another. On the one hand, mental health professionals have tended to conceive of

depression as a broad, multidimensional phenomenon. On the other hand, they have shown a persistent attempt, even urge, to isolate a depressive disease proper with strictly defined symptoms and aetiology, often neurophysiological aetiology, from the amorphous multiplicity of depressiveness. In a sense, the concept of endogenous depression was an early definition, although rather obscure, of depression as a biological condition, a 'brain disease'. Today, neuropsychiatric concepts of depression suggest far more specific neurophysiological 'causes' of depressive disorders; nevertheless, the problem of defining the borders of depressive illness has not vanished from the mental health discussion, and the topic is even more intensively debated today than in the early 20<sup>th</sup> century.

The term 'endogenous' leads back to the beginnings of biopsychiatry in the late 19<sup>th</sup> century. The term was first used as a scientific concept in early 19<sup>th</sup>-century botany, and it was introduced into psychiatry in 1892 by Paul Möbius, a psychiatrist from Leipzig. He used 'endogenous' to mean a special kind of biological aetiology of mental illness, in contrast to mental illnesses that he called 'exogenous'. He used 'exogenous' to refer to brain lesions caused by accidents or somatic illness, but it also referred to *Irresein* due to external causes like poisoning. By contrast, 'endogenous' referred to biological causes of mental illness that were internal to the individual, and of these causes Möbius considered hereditary predisposition to be the most relevant. In other words, the term 'endogenous' originally emerged from the 19<sup>th</sup> -century theory of degeneration. (Jackson, 1986, 211-212; Lewis, 1971: 191; Schmidt-Degenhard, 1983: 100.)

The concept was also significant to Emil Kraepelin. Throughout the various editions of *Psychiatrie*, in which he presented his influential definition of the

two major forms of mental illness, he also contrasted endogenous mental illnesses with exogenous illnesses as well as with functional ones. However, 'endogenous' was not one of Kraepelin's key concepts, and its meaning remained equivocal. He did not associate it with the ideas of degeneration as many German psychiatrists at the turn of the century did (see Lewis, 1971: 192). He mostly used the term to refer to the progression of mental dementia, and it had a primarily clinical meaning. In the famous sixth edition of his psychiatric textbook, he considered both dementia praecox and manic-depressive disease endogenous. In later editions, Kraepelin related 'endogenous' to the main criteria by which he differentiated dementia praecox from manic-depressive disease. While the course of the former illness was a chronic condition and its course was characterised by progressive dementia, the latter was periodic, did not involve cognitive decline, and recovery from it was possible; thus manic-depressive illness was a functional psychopathology, not an endogenous one. (Healy, 2008: 71-74; Jackson, 1986: 189-190; Schmidt-Degenhard, 1983: 100-101). There is some irony in this because depressive illness was at the centre of psychiatric debate over endogenous diseases in the early 20<sup>th</sup> century (see below).

As Kraepelin's influence increased in Western psychiatry during the first three decades of the 20<sup>th</sup> century, the opposition between endogenous and exogenous also became widely applied in the psychiatric discourse, first in Germany and later in Britain and the USA. The meaning of the dichotomy varied and became fuzzy. It disappeared from the sphere of biological aetiology and became a general psychiatric concept, with both aetiological and clinical meanings. Gradually, psychogenic causes slid into the exogenous category, and many psychiatrists began to call all, even

presumed, organic causes as endogenous. (Jackson, 1986: 212-213; Lewis, 1971: 191-193.) Aubrey Lewis commented on the situation retrospectively that eventually the term 'endogenous' referred to 'hypothetical, intangible, elusive predispositions, constitutional or hereditary forces which could be conjectured but not demonstrated.' In the end, most psychiatric authorities avowed that 'the endogenous concept, though logically requisite, was really a cover for a purely negative approach representing as internal causes what was left when all external causes had been eliminated.' (Lewis, 1971: 193.)

The development by which the division between endogenous and exogenous mental diseases was changed, multiplied and blurred was particularly important for psychiatric thinking on depressive illness; in fact, the terms were mainly applied in discussions of depression. Kraepelin's subsumption of all depressive pathologies into the category of manic-depressive illness (see Healy, 2008: 71-74) faced much criticism in early 20<sup>th</sup>-century psychiatry. Critical opinions gave the impetus for the introduction of numerous definitions of depression proper, i.e. endogenous depression, as well as the introduction of the idea of 'reactive' or 'neurotic' depression. This happened in Germany already in the 1910s, whereas in Britain and the U.S. depressive psychopathologies were discussed from the late 1920s onwards. Unsurprisingly, the view on depressive states was far from uniform. Mental health experts disagreed about the definition of endogenous depression as distinct from 'reactive,' 'neurotic' or 'exogenous' depressions—and quite a few authorities were eager to present their own classifications. Moreover, psychiatrists debated whether or not endogenous depression as a rigid disease entity even exists. The discussion was most intensive in the Anglo-Saxon psychiatry in

the 1930s and early 1940s. Particularly in British psychiatry, depressive illness was a major topic at that time. Differentiation between 'autonomous' (i.e. endogenous) and 'reactive' depression by Robert Gillespie (1929) provided a starting point for numerous attempts to define clinically valid categories and descriptions of the different types of depression (e.g. Curran, 1937; Hamilton & White, 1959; Lewis, 1934b; Pollitt, 1965). However, an unambiguous definition of clinical depression as a disease was not consolidated, and until the 1970s psychiatric discussion on depression was a farrago of idiosyncratic definitions and theories.<sup>1</sup> (Hill, 1968: 447-450; Lewis, 1934b: 361-363; 1971: 192-196; see also Jackson, 1986: 211-216.)

*Text Book of Psychiatry* by David Henderson and Robert Gillespie provides an illustration of this conceptual instability. The book promoted clinical emphasis in British psychiatry and was very influential since it remained a major reference from the 1920s to the 1960s. In the first edition from 1927, manic-depressive psychosis was seen as a major form of the affective reaction type, and Henderson and Gillespie separated endogenous manic-depressive psychosis from 'reactive depression.' In the former, there was no readily perceptible cause for the onset of illness, whereas a precipitating factor was involved in the latter. However, when discussing the neurotic reaction type in the 9<sup>th</sup> edition from 1962, Henderson and Gillespie concluded that the difference between endogenous and reactive depression was arbitrary. Aubrey Lewis, the biggest authority in British psychiatry, shared this view and emphasised that depressive illness should be seen as a continuum (Lewis, 1971).

An obvious reason for the above conceptual confusion was Adolf Meyer's view on depression. Meyer was very influential in U.S. mental health care before

the Second World War, and his ideas also weighed heavily in British psychiatry, e.g. through the works of Aubrey Lewis and David Henderson. One way to characterise Meyer's concept is to say that he rejected a categorical view of mental illness in favour of a dimensional one, so that depression referred to a dimension of mental health and ill health. The core of his 'psychobiology' was the idea that psychopathologies should be considered types of abnormal reactions instead of disease categories. Abnormality referred to deficiencies in adjustment of an individual, and reaction type was an extremely flexible concept covering a range of meanings from biological adaptation to personality. Meyer listed six reaction types, or 'disorders', and one of them was 'affective reaction type', with 'manic-depressive type', 'anxiety type' and 'simple depression' as its subtypes. (Meyer, 1952.) This view formed a bedrock for much of psychiatric discussion of depression before the late 1960s and even later, as the above discussion of depression by Henderson and Gillespie illustrates. Furthermore, the Meyerian approach focused on the case, the person under treatment, his or her personality type and difficulties in personal 'adjustment.' For such a view, clearly demarcated disease entities were secondary in understanding and treating mental illness. (Lewis, 1934a: 33-34; Hill, 1968: 450-451; Jackson, 1986: 195-200.) Such an emphasis certainly kept endogenous depression or other ideas of depressive illness being as a brain disease on the sidelines.

Before moving on, it should be noted that the above discussion took place in the world of mental hospitals and hard-core psychiatry working mostly with hospitalised patients, often with psychotic and chronic conditions, or with patients in intensive outpatient care. Private psychotherapeutic practice and general mental hygiene were marginal in this discussion, and the phenomenon we know

today as primary care depression would have seem quite strange to the practice and the professional experience that the above discussion of depression was embedded in. Moreover, depressive pathologies were marginal subjects in both psychiatry and psychodynamic therapy. Especially in the U.S., schizophrenia dominated thinking and practice in institutional psychiatry, while mental health experts with psychoanalytic orientation were interested in phenomena included under the broad heading 'anxiety' (e.g. Cooper et al., 1972; Hale, 1995: 47-52). Consequently, the context for problematisation of depression was different from ours. Today, the modification of psychiatry and its expansion into mental health care and the rise of biological explanations of mental disorders characterise the situation in which depression is the paradigm of mental disorders.

### **Pharmacological takeover**

Confusion over the distinction between endogenous and neurotic depressions remained more or less unchanged until the late 1950s and early 1960s. In fact, the psychiatric discussion of depression was rather subdued on both sides of the Atlantic after the Second World War. The rising field of psychopharmacology was an exception, and 'endogenous depression' was re-conceptualised in this context. As is well known, an antidepressant compound was simultaneously discovered by Roland Kuhn in Switzerland and Nathan Kline in the U.S. in 1957. Due to this invention, the idea of depression as a distinct brain disease was revived, and a new rationale emerged to provide empirical evidence for this idea. Roland Kuhn provided a sort of paradigm for the new pharmacological reasoning. In the mid-1950s he tried out a compound later known as imipramine on hospitalised

patients diagnosed as depressives. He noticed that the medication reduced the patients' symptoms, and he claimed that the effect revealed a specific biological disorder. Using Kurt Schneider's term from the 1920s, Kuhn called it 'vital depression'. Kuhn's study did not cause a sensation in psychiatry, and the pharmaceutical company Geigy, which Kuhn worked for, was not very impressed, either. The remarkable thing about Kuhn's study was its logic. Kuhn's claim that this mental disease existed was based on the existence of a chemical compound that had an effect on patients with a certain diagnosis. In the 1960s and 1970s, the emphasis on neurochemistry increased in psychopharmacology and neuropsychiatry, and receptor and neurotransmitter activities became the main points of interest. This development did not sweep away the rationale illustrated by Kuhn's study and on the contrary consolidated it. Theories of neurochemical malfunctions as causes of major mental illness were presented on the basis of drug effect evidence, among them the monoamine hypothesis of depression. The idea of depression as a 'pure' brain disease also played a role. In discussions of the monoamine hypothesis, the term 'endogenous depression' referred to, besides a certain cluster of symptoms, abnormal metabolism of a neurotransmitter in the brain, and it was contrasted with heterogeneous forms of 'reactive' or 'neurotic' depression. (Healy, 1997: 48-56, 76-77, 155-165; 2002, 198-219.)

Geigy's 1959 promotion leaflet for Tofranil, the product name of imipramine, illustrates well how this re-interpretation, or takeover, of endogenous depression by psychopharmacology happened. Already in the late 1950s, scientific reservations and ambiguities were not included in commercial information about psychotropic drugs—a practice that continues even today in the marketing of psychotropic drugs (see e.g. Lacasse & Leo, 2005). In Geigy's leaflet,



lack of clarity and controversies about depression were brushed aside by means of old recipes. First, elements of 'classic' melancholia were deployed to delineate depressive disease; second, endogenous depression was claimed to have a clear-cut aetiology in the same manner that 19<sup>th</sup>-century psychiatrists thought of psychic constitution and hereditary insanity. The new element in the argument was the close connection between the drug and depressive illness.

Endogenous depression is the major indication for the thymoleptic Tofranil. Of all the depressive states, true endogenous depression provides the 'classic' picture of Depression. The cardinal triad of symptoms, namely, depressed mood, lowering of vital functions and psychomotor retardation, together make up the actual depressive syndrome. (...) Heredity is an important factor in endogenous depression and can be demonstrated in the majority of patients. Before the outbreak of the psychosis patients are often of cyclothymic disposition with a tendency of 'mood-swings'. Most of them belong to the pycnic constitutional body type. About 70% are women. (Geigy, 1959: 1-3.)

The way the connection between Tofranil and the definition of depressive illness is presented in the leaflet seems quite current. However, this reasoning was still situated in the context of Kraepelin's concept of manic-depressive disease. The description of depression is congruent with the contemporary clinical picture of the depressive phase of the mania-depression cycle, and the list of symptoms also include delusions, which refers back to early 19<sup>th</sup>-century medical views of melancholy or even earlier discussions. There is also something new. Earlier in the 20<sup>th</sup> century, 'endogenous' referred to a plausible but unknown 'brain

defect' causing depressive illness. In Geigy's leaflet this logic of the unknown is applied to the drug: imipramine/Tofranil definitely affects depression, but the biochemical or neurophysiological mechanism of the effect is not (yet) known:

The marked success with Tofranil in endogenous depression can be explained by the fact that it directly influences the depressive syndrome and acts causally on the underlying pathological mental state, the 'core' of the depression. It must remain an open question where this 'core' is to be found and whether it is related to a particular part of the brain. (Geigy, 1959: 1)

The close connection between endogenous depression and the drug in Geigy's leaflet reflected the attempts to define and classify mental diseases on the basis of *drug cartography* (see Radden, 2003: 44-45). In the 1950s and 1960s, psychopharmacology researchers were occupied with drug cartography, when they tried to define depressive disease proper and other mental disorders on the basis of drug effects.

At that time, quite a few studies were carried out that defined subtypes of affective disorders on the basis of drug response, and many scholars firmly believed that the nosography of mental disorders could soon be anchored in neurophysiology and neurochemistry. (E.g. Overall et al., 1966; Schildkraut et al., 1978; see Healy, 1997: 52-77.) As psychopharmacology and neuropsychiatry have expanded in Western mental health care, one may assume that endogenous depression or a similar concept has been equally triumphant in the current mental health discourse (cf. Healy, 1997: 71-72, 76-77). But it has not. In the past three decades, the distinction between endogenous and neurotic depression and even the terms themselves have been widely dismissed.

The fading away of the concept of endogenous depression was embedded in two developments in Western psychiatry. First, there has been an internal tendency of psychopharmacology to define the object of anti-depressant medication ambiguously. For Roland Kuhn, vital depression was a specific disease for which imipramine was a cure, whereas Nathan Kline, the other pioneer of antidepressant medication, thought that all kinds of depressive states had a biological origin and that medication was therefore suitable for depression in general. In fact, he called the antidepressants 'psychic energisers'. (Healy, 1997: 66, 70-72.) This controversy between the specific and nonspecific biopsychiatric models of depression did not emerge in the discussions of psychopharmacology. Instead, it stemmed from the debate in the 1930s and 1940s whether electric shocks were useful only in treating depression 'proper' or also neurotic depressions. A similar debate was carried out in psychopharmacology in the late 1950s and 1960s, and the latter opinion has gradually proved to be victorious. (Ehrenberg, 2004: 55-57, 74-82; Hill, 1968: 449-450.) However, there was still ambiguity about the scope of use of the tricyclic antidepressants from the late 1960s to the 1980s. Imipramine and amitriptyline were marketed for a wide range of depressions, but quite a few psychopharmacologists and clinicians working with patients in psychiatric hospitals had an understanding that antidepressant medication was a treatment for a specific depressive disease. Donald Klein suggested naming the condition 'endogenomorphic depression' (Jackson, 1986: 218), but the psychiatrists usually discussed vital or endogenous depression. Yet, this view was controversial among Anglo-Saxon psychiatrists and it was gradually abandoned by the late 1980s; at that time Prozac and other SSRI were introduced in the mental health care

practices. (Healy, 1997: 70-77; Hill, 1968: 449-450.)

The second and most important development that has eclipsed the concept of endogenous depression is the implementation of the new system of classification of mental disorders in Western mental health care since the 1980s. I discuss the making of the DSM-III manual and the breakthrough of new classificatory rationale in detail in the next two sections; here I make only a brief remark about its impact.

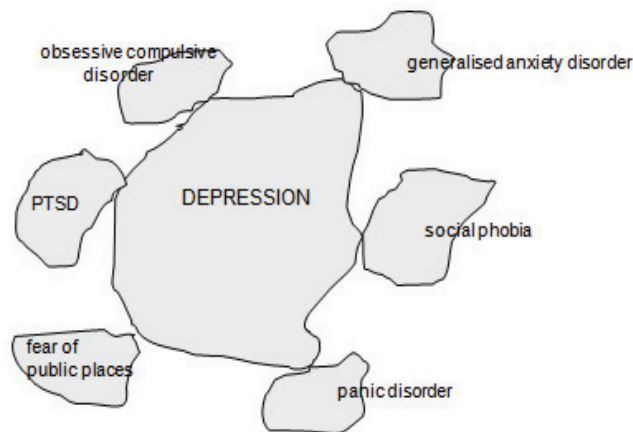
At first sight, it seems a bit strange that the idea of depression as a specific psychopathology was blurred by the consolidation of DSM-rationale in mental health care. The purpose of the new classification was to provide well-defined categories of mental disorders that can be applied in all clinical work and psychiatric research. The experimental reasoning of biopsychiatry and the classificatory and statistical reasoning of neo-raepelinian psychiatry also converge in the idea that depression can be defined as a distinct pathological entity, so it was conceived of as a natural kind of object. Historically, psychiatrists who oriented themselves towards biological research were the first to develop a unified diagnostic classification for research purposes. In general, the revival of classificatory psychiatry and the rise of mental health epidemiology have paved the way for the triumphant march of biopsychiatry during the past two decades. (Cooper, 2004: 18; Healy, 1997: 98-101, 233-234; 2002: 299-302; Mayes & Horwitz, 2005: 263-265.) Against this background, it seems justified to say that the category of 'major depression' in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) is firmly rooted in biopsychiatry.

However, 'major depression' as the central category of mood disorders is constructed on the basis of clinical and epidemiological data in DSM-III and

other similar psychiatric classifications (DSM-IV and ICD-10). In these manuals, major depression is conceived of as a purely descriptive entity, consisting of the listed symptoms without reference to any biological or other cause of illness. Empirical reasoning, aimed at being clinically relevant, is the foundation of neo-Kraepelinian psychiatry, and it cuts depression's umbilical cord to *neuroaetiology*. Robert Spitzer, the mastermind of the DSM revolution, summarised this rationale in the following way:

The general approach towards classification to be taken in DSM-III is to use aetiology as a classification axis if there is convincing evidence to support it. In the absence of such evidence, categories are grouped together if they share important clinical-descriptive features. (...) For this reason, we have decided to group together nearly all of the disorders which are characterized by a disturbance of mood. This includes all of the depressions and manias ... (Spitzer et al. 1977; Sit. Jackson, 1986: 218.)

By the end of the 20<sup>th</sup> century, the diagnostic criteria of major depression in DSM-III, DSM-IV and ICD-10 became the gold standard definition of depressive illness in clinical, administrative, policy and research activities in Western mental health care (see below), and 'major depression' defined the target of an antidepressant drug also in psychopharmacological research, especially in clinical trials (Healy, 2002: 303-308). As a consequence, the 'specific' object of the antidepressant medication was primarily the symptoms listed in the classification manual. However, this model of depressive disorder is also shattering because of a growing tendency in both professional and lay understanding of depression to follow a new kind of drug cartography. A reverse mode of mapping mood disorders according to drug effects as compared to the drug cartography of the 1950s and 1960s has become dominant in recent decades. When SSRI antidepressants (Selective Serotonin Reuptake Inhibitors) were introduced into clinical use and the market in the late 1980s, they were mostly used in the treatment of 'mild' depressions. Quite soon they were also applied in 'off label' treatment of, for example, obsessive



**Figure 1.** Elements of depression (Tamminen, 2001: 57).

compulsive disorder, panic disorder, eating disorders, states of anxiety or phobia, and even sexual paraphilias, and they were later licenced for these uses. (Healy, 1997: 176-177; 211-213; Rose, 2004: 112-118.)

The Finnish depression expert Tapani Tamminen (2001) summarises the current trend in the figure 1. It shows the main rock of depression and smaller rocks of disorders hidden in 'the sea of life,' which have the potential to wreck personal mental health. With the expansion of SSRI treatment, the interfaces of depressive illness multiply, and the dilemmas of differential diagnosis become more difficult. As a consequence, the scope of depression keeps on widening and becoming more heterogeneous.

### Major depression

The third and fourth editions of the *Diagnostic and Statistical Manual of Mental Disorders*, authorised by the American Psychiatric Association, and the new nosographic rationale serving as its foundation have had a major role in the profound transformation of mental health care seen in the Western world and even globally during the past three decades. This is an incontestable historical fact. (E.g. Mayes & Horwitz, 2005.) The classification of mental illnesses was re-enthroned and reformulated as a response to the problems of professional integrity that weighed down especially Anglo-Saxon psychiatry in the 1950s and the 1960s. The fundamental problems were related to both clinical practice and scientific research.

The situation of depression diagnostics with numerous mutually incompatible classifications of the disorder illustrates well the general situation in Western psychiatry after the Second World War. In the early 1960s, psychiatrists on both sides of the Atlantic were alarmed by the growing evidence of inconsistent diagnostic practices. The diagnosis of schizophrenia

was particularly problematic; patients diagnosed as schizophrenic by U.S. clinicians were diagnosed as depressed or neurotic by the British. Conferences of key figures of psychiatric professions were summoned to tackle this problem. At the same time, the World Health Organization started efforts to clarify disease classification, including that of mental diseases. (Healy, 2002: 297-299.)

Idiosyncratic diagnostic classifications were obstacles also for psychiatric research. During the 1960s, the interest in biological studies of mental illness and in epidemiology grew and clinical drug trials increased, especially in the U.S. These developments created a demand for eradicating fuzzy definitions of mental disorders. Diagnostic criteria for psychiatric research, out of which DSM-III evolved, were started to develop in the late 1960s. The research group lead by Eli Robins and Samuel Guze at Washington University in St. Louis constructed the classifications of 15 mental disorders, depression included, on the basis of statistical analysis of symptoms. This classification, known as the *Feighner Criteria*, was published in 1972. Three years later the group of scholars from many U.S. universities, advocated by the National Institute of Mental Health, published the operational criteria for 25 central mental conditions to be applied in psychiatric research.<sup>2</sup> This *Research Diagnostic Criteria* (RDC) was important for two reasons. For the first time, 'major depression' was used to name the main category of depressive illness. Furthermore, Robert Spitzer, later the chair of the DSM-III committee, was a core member of the research group that defined the RDC. At this point, the list of diagnostic criteria was considered to be a standardised tool for psychiatric research, and the idea of its use in clinical or therapeutic purposes had not yet occurred to mental health experts. (Healy, 2002: 299-301; Horwitz & Wakefield, 2007: 91-100.)

In 1974, the APA set up a committee to make a new classification of mental diseases. The project was to a great extent manoeuvred by scientifically minded epidemiologists and biopsychiatrists. Their motive was not only to turn psychiatry into a proper medical science but also to challenge psychoanalytic hegemony in U.S. mental health care. From the perspective they called neo-Kraepelinian, mental illness was conceived of as clear-cut 'disorders' that can be unambiguously defined as groups of symptoms, free of metapsychological or aetiological concepts and elements. The symptom lists and the grouping of disorders in the new classification were based on evidence from epidemiological studies, on statistical meta-analyses of former studies and clinical field trials, and on consensus views of subcommittees specialised in particular groups of psychiatric illness. (Healy, 1997: 233-237; Mayes & Horwitz, 2005: 258-263; Wilson, 1993; for a detailed analysis, see Kirk & Kutichins, 1992.) Clinical experience had a secondary role in this work since bed- or couchside knowledge was considered too idiosyncratic and not objective enough. (Healy, 2002: 320-333; Helén, 2007a: 160)

The result was published in 1980 as DSM-III, and the process to revise it started immediately. The revised version came out in the late 1980s, the new edition (DSM-IV) in 1994, and the fifth edition is expected to be released in 2012, at the earliest. There are a number of reasons why efforts to define a unified basis for psychiatry have turned into an ongoing process. Administrative manoeuvres, professional competition, and pressures from outside the psychiatric field have been equally, if not more, influential than scientific criteria in drawing up the DSM, and that has made the mental disorder categories perplexing and suspect (for an overview, see Kutichins & Kirk, 1997; Mayes & Horwitz, 2005). Moreover, epistemological

and ontological problems in defining the concept mental disorder and in delineating clinical entities are conspicuous, and many clinicians and psychotherapists find the categories and diagnostic criteria of both the DSM and the WHO's ICD-10 in many ways inconvenient or trivial when applied to individual cases in clinical practice (e.g. Cooper, 2004; Riikonen & Mattila, 1994; Wakefield, 1992; Wakefield et al., 2002; see also Kirk & Kutichins, 1992; Rose, 2006: 477-478; Horwitz & Wakefield, 2007).

Despite the shortcomings of the DSM and ICD-10 and the criticism they have faced, the new nosological approach has proved to be revolutionary in Western psychiatry, and the classifications are now an important cog in the mental health machinery. The key to their success lay in the expanded use of the diagnostic criteria. After DSM-III, they were no longer restricted to research purposes; instead, '[the] highest priority has been to provide a helpful guide to clinical practice', as the editors of DSM-IV defined their mission. They also acknowledge that the official classification of mental disorders 'must be applicable in a wide diversity of contexts [and] be usable across settings' (DSM-IV-TR: xxiii). The DSM and ICD-10 manuals have fulfilled this task so well that they are today indispensable for almost any activity of mental health care because they enable the *translation* of mental disorders from one context to another. The checklists of symptoms provide a common point of reference and a delineation of psychiatric phenomena especially suited for administration, policy making, and health insurance institutions. They can also be easily adopted and deployed in epidemiological, pharmaceutical and clinical research, in clinical and therapeutic practices and institutions, as well as in drug marketing and self-help guidebooks or on Internet sites. The lists of symptoms are both specific enough and flexible enough to

allow the notions of, for example, depression or anxiety to move between different mental health settings. In fact, the expansion and metamorphosis of psychiatry into mental health care work in every corner of society would have been impossible without such a medium of communication. Thus, the DSM provides the common language. (Lakoff, 2005: 12-14; Horwitz, 2002: 70-80; Mayes & Horwitz, 2005: 250-251, 263-266; Wilson, 1993: 408-409.)

Depressive illness and affective disorders were not among the most passionately debated diagnostic categories in the making of DSM-III. At the end of the 1960s and the beginning of the 1970s, many commentators representing both clinically oriented psychiatrists and researchers focusing on the biology or chemistry of depression were ready to give up the problematic idea of endogenous depression and suggested 'primary depression' as a simple descriptive category to replace it (e.g. Heron, 1965; Hill, 1968; Lewis, 1971; Robins & Guze, 1972; see also Whybrow, Akiskal & McKinney, 1984: 46-50). This was essentially carried out through the establishment of the category of 'major depression.' However, the DSM revolution not only clouded the category of endogenous depression but also did away with neurotic depression. This was due to the efforts of Spitzer's steering committee to eradicate psychoanalytic influence from the new classification. In particular, anything referring to neurosis was a red flag to the neo-Kraepelinian proclamation. (Healy, 1997: 235-236; 2002, 303-304; Mayes & Horwitz, 2005: 261-263; Wilson, 1993: 407). As 'neurotic depression' was erased, the dichotomy between endogenous and neurotic or reactive depression also vanished, which further obscured the idea of a depressive disease proper.

Mood disorders were thus combined in one class of mental disorders in DSM-III, DSM-IV and, ICD-10, and major depression

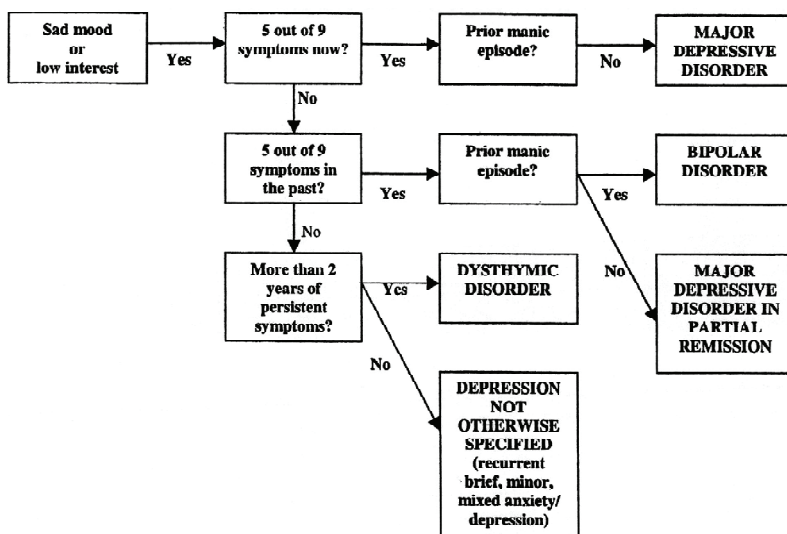
became the paradigm. Major depression is a nosological concept, comprising a well-known cluster of depressive symptoms. When applied in clinical, therapeutic, or other practical contexts, the concept can be used—and is routinely used—to turn depressive illness into a continuum ranging from simple symptoms to severe chronic illness. Such a shift has created the problem of how to distinguish 'clinically significant' depression from normal sorrow. (Horwitz & Wakefield, 2007.)

In Western countries today, depressions are mostly treated in primary health care, so it is a general practitioner who in most cases is responsible for diagnosis and treatment. This is the gold standard defined unanimously by national guidelines for depression treatment and is the state of affairs in real life. (Callahan & Berrios, 2005: 136-153; Helén, 2007a: 151-152, 158-159; Horwitz & Wakefield, 2007: 184-185.) Originally, the category of major depression was defined on the basis of clinical and epidemiological research in psychiatric hospitals and outpatient clinics. However, it has been shown by numerous studies (e.g. Blacker & Clare, 1987; Freeling, 1995; Freeling et al., 1985; Goldberg, 1979; Paykel & Priest, 1992; Poutanen, 1996) that the appearance of depression in primary care is considerably different from its appearance in specialised psychiatric care. GPs predominantly encounter patients with rather non-specific symptoms of dejectedness or disorderly mood, and various forms of depression 'masked' in somatic or behavioural symptoms, instead of patients showing symptoms that readily indicate major depression and the degree of the disorder (mild, moderate, or severe). As a result, mild and chronic forms of depression and a great variety of depressive symptoms form the core of depressive illness in primary health care.

Current mental health reasoning has responded to this problem by disseminating depressive illness. Depression can for example be considered a psychosomatic illness due to the fact that depressive disorders are in many cases eclipsed by somatic symptoms and patients' complaints about physical illness. In addition, two different ways exist of trying to solve the difficulties of GPs in making a distinction between depression and normal reactions caused by stressful situations and in distinguishing between a depressive disorder and an anxiety disorder when the patient's symptoms are a mixture of both. Depression has been conceived of as either being an adjustment disorder—which brings it into the realm of personality disorders—or being comparable to a stress disorder. The DSM language provides a foundation for these views because it offers means to define different variations through categories such as 'dysthymic disorder,' 'adjustment disorder with depressed mood,' 'adjustment disorder with mixed anxiety and depressed mood,' 'atypical depression,' 'depression not otherwise specified,' etc (for a detailed

analysis, see Horwitz & Wakefield, 2007: 104-122). However, this diagnostic diversity can subvert the paradigmatic position of major depression by shifting the focus of depression diagnosis and treatment to the detection of individual symptoms, especially in primary health care.

This dissolution has been to a great extent facilitated by diagnostic devices that mental health authorities, professional organisations and pharmaceutical companies design and distribute to GPs to help them detect and diagnose depressive illness more efficiently. 'Rating scales' used as diagnostic instruments in epidemiological, pharmaceutical, and clinical research have been modified into a variety of depression scales in the form of checklists that are readily available for physicians on the Internet and as notepads with fill-in forms (see below). In addition, the GPs are provided with the kinds of 'tools' for differential diagnosis shown in Figure 2. This particular scheme is from an authorised depression management guideline in the U.S., and a similar type of diagnostic path is also presented in



**Figure 2.** Differential diagnosis of primary mood disorders (*Depression in primary care*, vol. 1, 1993: 20).

Appendix A of DSM-IV. A noteworthy route in the diagram leads from the symptoms of 'sad mood or low interest' to the diagnosis of 'depression not otherwise specified'. Although the latter is considered a residual category, it nevertheless is a diagnostic option that a physician can use when deciding whether or not a patient showing few depressive symptoms is suffering from a treatable mental disorder. This implicates a kind of reduction of the depressive disorder to single symptoms and ultimately leads to a view solely focused on a person's low *mood* or apathetic existence. However, with such a focus, it is difficult to see where depression begins and where it ends. Yet, these kinds of 'algorithms' encourage and justify the use of antidepressants to relieve patients' sadness, hopelessness, or 'feeling blue'. It is a clinical pathway to a medication of mood.

### **Risk factors**

Mental health experts and public authorities involved in the mainstream mental health discussion often consider feelings and behaviour traits listed as the symptoms of depressive disorders in the dominant DSM-IV definition also as health risks to the person. However, thinking of depression in terms of risk is actually related to the problematisation of depressive illness in the population and as a public health issue. Thus, the risk concept of depression is closely related to the public health management of depressive illness aiming at prevention, early detection and early intervention by the means of population screening and making diagnostic practices and treatment of depression in primary health care more efficient (see e.g. Helén, 2007b; Horwitz & Wakefield, 2007: 145-163). Moreover, the idea of risk greatly facilitated the transformation of depression into a major public health problem, as I will show below.

The number of people with a depressive disorder is today enormously larger than 50 or 60 years ago; one can even speak of a thousand-fold increase in the prevalence of depression (Healy, 2004: 2). Consequently, depression has become a major public health problem, worldwide. This amazing development would not have happened without psychiatric epidemiology, which has become a cornerstone of psychiatry and mental health care since the 1960s. Without studies focusing on 'mental morbidity' in the population mental illness would not have become a phenomenon of great numbers—epistemically, commercially and as a matter of public policy. Moreover, in the attempts to make psychiatry more scientific the triumph of the styles of reasoning familiar from epidemiology and social medicine—statistical induction, probability calculus and risk estimation—has been much more crucial than the rise of psychopharmacology and neuropsychiatry. It is nowadays almost impossible to present claims about mental health facts without supporting them by statistical analysis of data from epidemiological questionnaires or randomised clinical trials. (Healy, 2002: 320-333; Horwitz & Wakefield, 2007:123-143.)

Censuses of the mentally ill for the purposes of general population census and poor relief administration were carried out in the U.S., Britain, Germany and the Nordic countries since the late 19<sup>th</sup> century. However, modern psychiatric epidemiology is not about counting the number of inmates classified as insane in institutions of incarceration but about analysis of all types of factors related to mental disturbances in individuals and populations. Such an approach emerged in Anglo-Saxon psychiatry during the Second World War. In Britain and the U.S., this new 'social psychiatry' originated from two sources. First, studies on the psychological effects of



wartime conditions and on the prevalence of psychiatric symptoms in the general population were launched and extensive surveys of the psychological conditions of the combat forces were carried out, especially in the U.S. Second, some psychiatrists started surveying large numbers of psychiatric patients to search for social factors that apply to all patients.<sup>3</sup> (Healy, 2002: 136-139; Horwitz & Wakefield, 2007:124-126; Rose, 1996: 133-135.) Toward the late 1950s, the rise of community psychiatry supported the growth of population studies of mental disorders, as did the increasing interest in studying the prevalence and treatment of mental illness in primary health care. In the 1960's, more and more studies on mental illness in general population were started on the both sides of the Atlantic. First results of the Midtown Manhattan project were published in the U.S. in 1962, and Michael Shepherd's group published the pioneering work *Psychiatric illness in general practice* in Britain in 1966. An epidemiology of depressive illness focusing on the general population and primary care practice grew out of this soil. This started in the early 1970s and expanded in the 1980s and 1990s. (Callahan & Berrios, 2005: 133-136; Horwitz & Wakefield, 2007: 127-138; Healy, 1997: 229.) This research has been seminal in helping the national and international health authorities consolidate the present depression management standard, and it has greatly facilitated the increase in antidepressant medication (Helén, 2007a: 157-167).

The epidemiological approach and reasoning have induced changes in the object of mental health thought and practice. Mental diseases are no longer considered more or less rigid clinical entities, and mental health care is instead directed towards 'psychiatric morbidity', which refers to a wide range of impaired psychological or behavioural functionality.

Such a widening of the target area of mental health care has required three epistemic transitions in psychiatry. The first one is the emergence of the concept of mental illness as *multifactorial*, as a system or synergism of biological, psychological, and social factors. This idea achieved its breakthrough alongside the community and social psychiatry movements from the late 1950s to 1970s. It was mainly related to the reform and policy that made outpatient care the main form of psychiatric care in the West. (Helén, 2007b.) More important epistemic shifts in the context of this paper are the increasing emphasis on *symptoms* and the development of *health risk* as a core concept. These two are more directly related to psychiatric epidemiology.

Structured questionnaires and assessment scales are the main instruments focusing more intensively on the symptoms and risks of mental disorders. They are essential in psychiatric epidemiology when data is analysed and decisions are made whether a 'case' belongs to the group of people suffering from the disorder or not. The first structured 'diagnostic instruments' providing data suitable for statistical analysis were developed for the purposes of clinical research in the late 1950s and 1960s. They provided a model for questionnaires and scales designed for epidemiological research of mental disorders, such as the DSM based Diagnostic Interview Schedule (DIS) and Composite International Diagnostic Interview (CIDI) that are commonly used in the current epidemiology of depressive illness.<sup>4</sup> However, they are by no means the only ones. In the early 1990s, one could find over 30 'rating scales', both general and more specified, in research literature (Snaith, 1993; for a review, see Rabkin & Klein, 1987; Sartorius & Ban, 1986), and their number has increased since then. Quite a few are also readily available on the Internet. However, none of them

has succeeded in becoming the ultimate criterion for psychiatric epidemiology. On the contrary, each of the scales is subject to on-going dispute over the validity, reliability, and applicability of the results it provides. In practice, studies seldom rely on only one rating scale and instead deploy two to four diagnostic instruments side by side (e.g. Poutanen, 1996).

Despite their problems, rating scales play a key role in the epidemiology of mental disorders for two reasons. First, they ensure that the symptoms enter into the field of epidemiological perception. Second, they connect risks with concrete phenomena and thus establish risk factors. In fact, diagnostic instruments form the core of the equipment used to understand and observe mental health that epidemiology brought into the heart of psychiatry. This apparatus has been made part of the routines of every field of mental health care. It is a tool for clinical trials as well as population surveys; it is an essential element of clinical assessment in fields ranging from general practice to psychotherapy; it is deployed in the marketing of drugs and other therapies; and such scales are available for personal mental health assessment and mood monitoring on the Internet and in popular literature. (Hautamäki, 2007: 126-127; Healy, 1997, 97-100; Horwitz & Wakefield, 2007: 130-136, 140-164; Martin, 2007: 177-196.)

Most importantly, this apparatus has made it possible to see psychopathologies—or, in more current terms, ‘mental disorders’—as a widespread public health problem. The rating scale equipment has brought about a shift in psychiatric perception of mental phenomena. This shift has implied that symptoms and risks are the primary targets of mental health thinking and practice. In addition, the focus on symptoms and risks allows the kinds of arguments and estimations presented by recent studies in the U.S. and the EU according to which

27–28 % of the adult population suffers from a diagnosable mental disorder<sup>5</sup>, and every other person will become ill with a mental disorder sometime in their lives (Kessler et al., 2005; Wittchen et al., 2005; see Rose, 2006: 467-470.) Such a view of the prevalence of mental disorders is not a recent development, but it is an intrinsic trait of the epidemiological approach, notable already in the early community mental health surveys in the 1960s and early 1970s (Helén, 2007b; Horwitz & Wakefield, 2007: 123-138). For example, the first Finnish population surveys of mental illness allowed epidemiologists to state that up to 30% of Finns will suffer from a mental disorder during their adult life (Lehtinen & Väisänen, 1979: 121) and would benefit from psychiatric consultation or treatment (Lehtinen, 1975: 121). In a similar vein, depression literature, both professional and lay, underlines time and again that ‘almost everybody will fall ill with a minor depressive episode during his or her life and one out of four will suffer from major depression’ (Tamminen, 2001: 52).

Epidemiological understanding of the object and scope of mental health care is certainly congruent with the change in clinical understanding, epitomised by the DSM definition of mental disorder:

Each of mental disorders is conceptualized as a clinically significant behavioural or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g., a painful symptom) or disability (e.g., impairment in one or more important areas of functioning) or with significantly increased risk of suffering death, pain, disability, or an important loss of freedom. (...) Whatever its original course, it must currently be considered a manifestation of a behavioural, psychological, or biological

dysfunction in the individual. (DSM-IV-TR: xxxi.)

The definition clearly states that the object of mental health care is neither madness nor disease. But what is it? First, mental disorder is always related to the individual, to his or her behaviour or psyche; it is personal but not the person. Furthermore, the disorder should be clinically verified. But what should a physician, psychiatrist, or therapist look for? The definition lists a range of options including distress or malady personally experienced by the patient or impairment, dysfunctionality, or risks observed by health professionals. Thus, the field of work of mental health care is defined as very broad, while the object of mental health care remains equivocal.

Widening of the scope of psychiatry and the inclusion of multiple and heterogeneous objects into its domain is not a particularly recent development. At the end of the Second World War and during the decades after war, deployment of psychiatry and psychotherapy multiplied and expanded in social work, general medicine, education, management etc. throughout the Western world, the American psychodynamic psychiatry and also early psychopharmacology leading the way. This development can be traced back to the mental hygiene movement in many Western countries between the two World Wars and even to the late 19<sup>th</sup> century campaigns against 'nervousness'. (E.g. Hale, 1995: 257-299; Horwitz, 2002: 38-55; Thomson, 1995). This preceded expansion explains a great deal of the success of the catch-all diagnostic categories of the new DSM and ICD-10 manuals. They are well suited to dispersing mental health care and a situation in which mental health experts try to manage 'disorders' whose variety seems to be boundless. Important aspects of this transition are certainly that

the disease model has been extended into many areas of social life and morality, and that providers of mental health care have become a major agent of social control. (Horwitz, 2002; Mayes & Horwitz, 2005; Rose, 2006.) However, what seems crucial regarding the present development is the shift of mental health care towards the field of risk management (Helén, 2007b; Rose, 2006.)

A close affinity between the epidemiological, classificatory and pharmacological ways of approaching their objects (see Helén, 2007a: 160, 163-164) has greatly facilitated the transition of mental health care into risk management. The diagnostic instruments used in population surveys and clinical trials are compatible with the DSM and ICD manuals, since they define mental disorders as a thing-like entity and describe it as a collage of symptoms. This compatibility is important for clinical practice, and the checklist format provides the basis for making 'diagnostic toolkits' for general practitioners to help them recognise depression-or bipolar, anxiety, or attention disorder or any other of a number of 'brain diseases' for which such detection devices are developed. These tools are simplified questionnaires and rating scales aimed at indicating if the patient shows symptoms or not. (see Horwitz & Wakefield, 2007: 147-163.) Today, a great variety of mental rating scales are also available on the websites of public health authorities, professional and patients' organisations, and pharmaceutical companies to be used as a kind of everyman's self-assessment device. (Hautamäki, 2007: 126-127; Martin, 2007:177-196.)

My example is the DEPS scale (see Figure 3), developed in the Tampere Depression Study in the early 1990s in Finland. The scale has a double function. It was originally developed as a diagnostic tool for GPs, but it also served as an epidemiological assessment device in the study (Salokangas,

During the last month, I have	Not at all	A little	Quite a lot	Ex-tremely
Suffered from insomnia	0	1	2	3
Felt blue	0	1	2	3
Felt everything was an effort	0	1	2	3
Felt low in energy or slowed down	0	1	2	3
Felt lonely	0	1	2	3
Felt hopeless about the future	0	1	2	3
Not got any fun out of life	0	1	2	3
Had feelings of worthlessness	0	1	2	3
Felt all pleasure and joy has gone from life	0	1	2	3
Felt that I can not shake off the blues even with help from family and friends	0	1	2	3

**Figure 3.** The DEPS scale (Salokangas, Poutanen & Stengård, 1995: 11).

Poutanen & Stengård, 1995; on the TADEP study, see Helén, 2007a: 157-158). DEPS also exemplifies a close connection between the above-mentioned concepts of depression and medication. Notepads with empty DEPS forms were widely disseminated to Finnish physicians in order to familiarise them with the scale; the notepads and their distribution were sponsored by Lundbeck, the manufacturer of Cipramil, which is the most sold SSRI antidepressant in the Nordic countries. Today, the DEPS scale is also easily accessible on a website for medical advice ([www.tohtori.fi](http://www.tohtori.fi)) sponsored by Lundbeck.

An essential issue here is a tendency that emerges when depressive symptoms are approached through structured questionnaires and scales, for then the clinical and epidemiological interpretations of the answers start to merge. In the clinical-therapeutic context, the answers and the figures provided by the rating scales are considered symptoms of depressive disorder. In the epidemiological context, they are however indications of an increased likelihood or susceptibility, i.e. risk, of the onset of depression. In the current mental health discussion in the West, these aspects

are merged so that talk of risks tends to become personalised, and the symptoms are increasingly seen as indications of possible or probable illness.

The DEPS scale epitomises both the tendency to mix symptoms and risks and to focus increasingly on risks. This is an important development. The epidemiological approach has induced an anticipatory rationale in psychiatry and mental health care so that these are no longer targeted at actual mental diseases or disorders but at risks and susceptibilities, at *potential* mental disorders.

Depression has been related to mental health risks in two ways. Originally, epidemiological studies emphasised the relationships between depressive illness and suicide so that depression represented the most ‘predictive factor’ for suicide. This was an important impetus for anti-suicide and anti-depression campaigns in different countries from the 1980s onwards. (e.g. Lönnqvist, 1988.) Today, epidemiology predominantly emphasises the risks of depressive illness itself, with the assumption that any of the symptoms listed in the rating scales or DSM/ICD checklists indicate an increased risk of depression. Or as a

Finnish manual puts it: ‘The emergence of depressive symptoms in a patient indicates a fourfold increased threat of falling ill with depression, as compared to the ones without symptoms’ (Tamminen, 2001: 52). This perspective has also been widely adopted in clinical practice. All over the Western world, a central practical problem with the depression epidemic was the shortcomings of primary care physicians in recognising depression; this, in turn, led to an underdiagnosis of the illness. What GPs were actually supposed to ‘recognise’ were the patient’s symptoms, and this should have been followed by anticipatory measures targeted at preventing the onset of an illness that could possibly threaten the patient. According to this logic, depression treatment extended into the area between normal ‘feeling low’ or sorrow and severe major depression, including also mild depression and other depressive conditions mentioned above. (Callahan & Berrios, 2005: 139-153; Helén, 2007a, 151-153; Horwitz & Wakefield, 2007.) As a result, a plethora of maladies and irregular behaviour became treated as depression.

### Care and control

If symptoms and risks are emphasised in the current concept of depression, what actually is treated when ‘depressive disorder’ is treated? What are the aims of the treatment, and how does the dominant concept direct the therapeutic rationale?

The depression treatment standard was gradually developed in the West from the mid-1980s to the late-1990s (see Callahan & Berrios, 2005: 121-153; Helén, 2007a: 151-152, 158-159). The rationale was adopted from maintenance treatment, a mainstream form of psychiatric treatment that was established with the expansion of the outpatient care and psychopharmacology. Mental health professionals use

‘maintenance treatment’ to refer to a procedure applied in cases of severe mental disorder when medication is continued after the patient’s symptoms have disappeared and he or she has recovered. This is done to prevent relapses, and the medication in fact becomes permanent not only for some but, in fact, most patients. In the case of depressive disorders, the practice is justified by the epidemiological fact of high recurrence of major depression. In other words, the main factor predicting an onset of depressive illness is a previous episode of depression, and therefore depression is treated *presymptomatically* in the form of maintenance treatment. The object of medication is a potential disorder that is articulated as the patient’s *risk* of recurring depression.

The above treatment rationale was formulated at the same time as the epidemiology of depression began to cumulate internationally and the tricyclic antidepressants acquired the leading therapeutic role in the 1970s and 1980s. Thus the idea of maintenance treatment of depression preceded the breakthrough of the SSRIs. What Prozac, Cipramil and other serotonin selective substances did was to facilitate the expansion of the application of risk logic in overall management of depressive disorders, including general practice. The current standard of depression treatment contains a preventive rationale implicitly promoting medication even in less severe depressions. Depression is regarded as a not only highly recurrent but also progressive. This view implies the idea that minor depressive symptoms left without treatment will become more severe, multiply, and finally lead to severe major depression. Mental health experts therefore consider the immediate treatment of depressive symptoms (low mood, sleeping difficulties or irritability, for example) vitally important in order to

prevent the onset of depressive disorder. In the early 1990s, when Prozac and other SSRIs had just entered the market and the activities for implementing the depression management standard were at their peak in the U.S. and many European countries, the antidepressants, especially the SSRIs, were recommended for the treatment of all types of depression and depressive symptoms, because they were claimed to be the easiest means of acute intervention. (Healy, 2004: 9-11, 30-39; Helén, 2007a: 164-166; Horwitz & Wakefield, 2007: 179-193.) Later, mental health experts have become uncertain about the effectiveness of drug treatment for mild depression as international research began providing contradictory evidence on the issue. Today, many authorised guidelines recommend brief cognitive therapy as the first-choice treatment of mild depressions. (E.g. Spigset & Mårtensson, 1999; NICE, 2004; *Käypä hoito: Depressio*, 2009.) However, institutions providing mental health care lack psychotherapy resources, and medication remains the first option available for physicians, especially in primary care when treating depressive symptoms and mild depression and trying to prevent the condition from becoming more severe.

All in all, depressive symptoms are not primarily perceived to be indications of mental illness in the context of today's depression management. Instead, they are seen as an increased risk of the person to develop depression, originating either from his or her life situation or inborn disposition. In any case, the acute treatments of depressive symptoms or 'mild depression' by drugs or by therapy based on talking are largely targeted at the risk of depressive illness and are thus *preventive* procedures.

However, the preventive nature of depression treatment is equivocal. The mainstream notion in both professional and lay discussions is that depression is a severe and, in most cases, chronic disease.

Due to this, mental health rationale puts a sort of double exposure on the treatment of depressive symptoms. On the one hand, medication, supportive talk, or psychotherapy is seen to prevent the onset of illness, but on the other, they are considered acute therapies of a disease that shares many characteristics of chronic diseases like diabetes. In fact, diabetes and insulin treatment are the most popular parallel to depressive illness and antidepressant medication in the literature. This ambiguity has engendered a tendency of depression to become a chronic condition through the treatment procedure targeted at depressive symptoms; medication is particularly inclined to do this (see Healy, 2004: *passim*.; Karp, 2006: 62-95).

The core idea of 'curing' symptoms by an antidepressant or other means implies yet another mode of treatment. This is also anticipatory care, and it may go beyond both the reduction of depression risks and the treatment of illness. Perhaps it can be called *proactive* treatment.

It has been claimed that SSRI antidepressants permit patients to reform themselves instead of being on guard against and struggling with the illness. Such alleged effects of medication have been presented especially in discussions of 'cosmetic psychopharmacology' and 'medical enhancement' incited by Peter Kramer's *Listening to Prozac* (1994). On the basis of his cases, Kramer claimed that SSRIs are able to provide people a means to alter the appearance of the self, to empower them and give them a choice over their emotions, reactions, and behaviour in different situations, in a word, to change their personality. Although he exaggerated or misconstrued the effects of the drugs—the SSRIs are not ecstatic drugs, and they are too weak to be able to cause personality alteration (see Healy, 2004: 266-269)—his bestseller gave an impetus to the notion

that antidepressant medication is not just a remedy but also a means to modify what a living person is and what he or she is capable of doing. Prozac, Cipramil, Seroxat, etc. are among these enhancing or enabling medical devices that are claimed to be able to contribute to three types of modifications. First, they may enable the person to overcome, replace or circumvent his or her existing deficiency or even a characteristic that fits within the limits of normal variation. Second, they may help a person to manage personal risks or susceptibilities, or resolve minor symptoms. Kramer's discussion was related to these two uses of 'prozac'. Finally, the drug may restore the person within the normal limits of experience, behaviour and functionality; this aspect is usually emphasised in the personal narratives of antidepressant users (Helén, 2006; see also Karp, 2006: 62-95).

Prozac-type drugs as agents of normalisation, risk management or enablement are targeted at mood-related neurochemistry. Their effect is not immediate, since it takes a couple weeks for a therapeutic response to appear and they have to be used regularly. So, what Cipramil and similar drugs may contribute are alterations in the person's general functionality and the tuning of their personal existence—'mood' often refers to the latter in psychological and psychiatric discourse (e.g. Larsen, 2001: 129-130; Whybrow, 1997: 19). These changes may facilitate persons to carry out a significant transformation in their lives or even 'personality changes', but predominantly the users see the drugs helping them in normalising feelings and behaviour, or 'getting their life back' (Helén, 2006; see also Karp, 2006: 62-95). Thus, medication affecting biochemical functions in the brain is conceived of as a device for regulating moods and bringing personal life under control.

Pharmaceutical technology facilitates a transformation of depression treatment into a practice of adjusting personal mood to the demands of the situation. This could be called *mood control*. Some aspects of Kramer's thinking and the widespread Prozac hype in the early 1990s shed light on the relationship between medication and mood control. Kramer did not reduce personality to the brain but instead considered it multiple. What someone is as a person is determined by what happens at the cell and molecular level in the brain, by interaction and relationships with other people, and by the care, upbringing, and life events encountered, especially the ones experienced in early childhood. The reason Kramer emphasised neurochemistry was his experience as a psychotherapist. In the light of his therapeutic experience, it seemed to be *easier* to reduce personal susceptibility to depression or panic attacks by regulating the biochemical functions of the brain than by analysing and influencing—or 'working through' in psychoanalytic language – the role, behaviour, and emotional reaction patterns that the person had internalised in the process of personality development. (Fraser, 2001.) From this perspective, all that we are as persons – biologically, socially, and psychologically—and every aspect of the condition we live in can be seen as divisible into elements, factors, and causal relations, which can in turn be measured, calculated, monitored, and manipulated.

### **Beyond depression?**

The depression paradigm seems very firm in mental health care worldwide. The epistemic foundation of the paradigm is a close affinity between the epidemiological, classificatory and pharmacological approach of the objects of mental health care. The DSM and ICD manuals define depression and other mental disorders in

such a manner that they provide targets specific enough for clinical treatment, especially for psychotropic medication. They also define mental illness as entities that can be measured and calculated both in epidemiological research and clinical trials in psychopharmacology, as well as in mental health policy, administration and economics. Standardisation of mental illness in a manner of depression is particularly important in the context of health insurance reimbursements. This epistemic interlocking of epidemiology, classification, clinical evaluation and therapeutic knowledge may well have created a harmony of illusions about both the object of mental health care and its effectiveness. In any case, the depression paradigm is able to facilitate the functions of globally expanding mental health machinery. (See Helén, 2007a: 160, 163-164; Horwitz & Wakefield, 2007: 104-191; Mayes & Horwitz, 2005: 250-251, 263-266; Wilson, 1993: 408-409.)

The depression paradigm has also strong institutional back up. It has been instrumental in making evermore heterogeneous objects of knowledge and practice manageable and even improving their manageability when Western psychiatry has gone through a fundamental restructuring during the past 60 years. As well known, the change is characterised by dehospitalisation and a growing emphasis on out-patient care, community psychiatry and prevention, and an increasing blending of mental health care into many branches of medicine and social work. With this development depressive illness became the focus of mental health care. Moreover, the depression paradigm has been beneficial to the profession of the psychiatrists by helping them to maintain their professional status and integrity as psychiatry has been disseminated into society. (E.g. Grob, 1991; Horwitz, 2002.) Finally, it has provided

the basis for many SSRI antidepressants to become blockbuster drugs and thus served well the economic interest of the multinational pharmaceutical enterprises (see Healy, 2004; Barber, 2008).

Despite its seemingly solid position, the depression paradigm may scatter in the near future. The growing criticism of the diagnostic system (e.g. Cooper, 2004; Horwitz & Wakefield, 2007; Wakefield et al., 2002), the SSRI-antidepressants and medication regimes (e.g. Healy, 2004; Horwitz & Wakefield, 2007: 179-193; Moncrieff & Cohen, 2005), and the role of the psychiatric profession in creating and maintaining the paradigm (e.g. Healy 2004; Shorter, 2009) both inside and outside the psychiatric profession is undermining the current depression management standard. In the light of my analysis, I would however emphasise subverting features inherent in the depression paradigm.

I claim that the dissolution of the very object of depression as both epistemic and practical core of the current mental health care seems quite plausible. The ways depression is defined in the present depression paradigm include a tendency of dissolution. The medical concept of a depressive *disease* with clear somatic causes and aetiology seems to be scattered in the nosological rationale of current mental health thinking and the drug cartography of mental disorders. The definition of depressive *disorder* embedded in neo-Kraepelinian nosology seems to disintegrate into the detection of individual depressive symptoms. Finally, symptoms seem to turn into risk factors in the context of epidemiology and preventive mental health care, and the role of risks in cutting-edge neuropsychiatry is diminishing as the focus is switched to personal or, rather, molecular susceptibilities on the levels of genome and neurochemistry (on the latter development, see Rose, 2007: 204-205).



Furthermore, what research and theoretical discussion have dissolved, practices seem to be unable to bring into coherence. Within the current standard of depression management the object of care and treatment is fundamentally ambivalent and presumably not what it is supposed to be as common reactions to loss or distress are treated as illness (see Horwitz & Wakefield, 2007). It seems that depression treatment covers a continuum extending from severe pathological conditions to symptoms and prodromal signs and further to risks and susceptibilities.

Depressive disorder is often defined in the framework of the disease model as independent psychopathological entity in the current mental health thinking and practice. This view is embedded in the psychiatric knowledge and experience derived from the treatment of people who suffer from severe or chronic depression at mental hospitals and outpatient clinics. The wide autobiographical literature tends to support this concept, since the authors usually tell about their personal struggle with depressive *disease*. However, my analysis in the previous section suggests that the focus of the professional rationale is not primarily the treatment of mood disorders but *risk management*. Since observation and practices in mental health care are primarily targeted at symptoms and risks, the most of depression treatment takes place in the zone between normal sadness and severe depression, in the grey area of 'moderate', 'mild' and 'under threshold' depressive conditions. This is the case especially with the so called primary care depression.

When physicians conceive of symptoms as indications of the increased risk of depressive illness, they do not see deep dejection, overwhelming sadness, low spirits or similar conditions as expressions of an actual illness. Rather, the latter are signs of possible and forthcoming

disturbance of mind and behaviour, danger or susceptibility of the person's inner life to become disordered. With the latter focus, diagnosing a patient, intervention into his or her 'depression' and restoring of normalcy with the help of drugs, talk or both are anticipatory and preventive procedures aimed at an onset illness the patient might possibly face. Psychiatric intervention, psychotherapy or psychotropic medication is thus not a device of medical care but of control of living.

This result of my analysis—that the aspect of monitoring and anticipatory control of personal living is increasingly emphasised in depression management—is congruent with the observations of Nikolas Rose and Emily Martin. According to them, Western mental health care is moving towards the practices of assessing, anticipating, and controlling impairments and differences of human behaviour and experience and towards the management of personal feelings, desires and conduct *beyond the treatment of illness* (Rose, 2006; Martin, 2007: 197-219). This trend may well do away with medical concepts like 'depression disorder' that alludes to pathology and abnormality. A sort of promise of tailored mental management, similar to the visions of 'personalised medicine', will possibly be raised, if psychiatric applications of molecular genetics and neuroscience progress further and also the techniques of psychological and social risk assessment become more accurate. Then, treatment, care or control could be targeted directly to the factors, risks, and prospects of a problematic situation or to the characteristics, personality traits, or susceptibilities of a particular individual or group of people—especially to those related the neurophysiology. However, the actualisation of these hopes is not likely to happen very soon. (E.g. Rose, 2007: 198-209; Singh & Rose, 2009.)<sup>6</sup>

The shift in depression treatment towards the control of moods and personal conduct as a form of risk management has created a space for reasoning and discussion within mental health care in which numerous candidates to replace the depression paradigm have been presented. For example, the idea of a mood disorder spectrum (e.g. Akiskal & Akiskal, 2005; Angst & Cassano, 2005; see also Healy, 2008: 198-219) and the resurrected concept of temperament—today conceived of as a biopsychological typology of personality traits and closely connected with the ideas of individual variation based on brain chemistry and with genetic susceptibilities to mental disorders (see Rose, 2007: 188-209)—have been promoted as frameworks to diagnose and treat affective irregularities. Both of these concepts are open to many interpretations and may thus serve well numerous efforts to control and management of feelings and behaviour.

However, even if ‘mood spectrum’, ‘temperament’ or similar concepts managed to overshadow the disorder concept defined by the DSM rationale as the cornerstone of mental health thinking and practice, the basic dilemma would remain unsolved: when mental functions of the person become disordered, should his or her condition be conceived of as a natural kind of object or a personal experience and a state of existence, happening in unique circumstances?

## Notes

<sup>1</sup> When consulting the World Health Organization in making the section on psychiatric and neurological diseases for ICD at the end of the 1950s and the early 1960s, Erwin Stengel was presented with about 60 different classifications of depressive illnesses, with considerable variation between them (Stengel, 1964).

<sup>2</sup> NIMH became interested in the problems of diagnostic categories when it launched a big research programme to test Joseph Schildkraut’s (1965) hypothesis that depression is caused by a reduction in brain catecholamines. When trying to recruit a homogeneous group of depressives for tests, it became obvious to the research group that the diagnostic criteria were equivocal. Attempts to solve this problem led to the development of the RDC. (Healy 2002: 299-301.)

<sup>3</sup> The pioneers in using the methods of social medicine in psychiatry were George Rosen in the U.S. and Aubrey Lewis in Britain (Healy, 2002: 136-137).

<sup>4</sup> Hamilton’s scale was developed by the British psychiatrist Max Hamilton for clinical trials of tricyclic antidepressants at the end of the 1950s, while Beck’s inventory is an offspring of Aaron Beck, an American psychiatrist and the developer of the cognitive therapy for depression (Healy, 1997: 98-99). Schedule for Assessment of Affective Disorders and Schizophrenia (SADS), one of the first structured interview for clinical use, was developed by Robert Spitzer in the late 1970s, and it provided an important model for DIS, deployed in the first national epidemiology of mental illness in the U.S. in the early 1980s, and CIDI. (Horwitz & Wakefield, 2007: 95, 130-131).

<sup>5</sup> This represents over 57 million Americans and well over 80 million people in the EU.

<sup>6</sup> It should be emphasized that the current SSRI drugs do not have specific effects in this sense, although Prozac, Cipramil and other similar drugs were marketed as a kind of magic bullets for depression. Their therapeutic effects are imprecise, and for a great number of patients they either are ineffective or cause considerable adverse effects. Moreover, the therapeutic scope of SSRIs has become so wide that they

seem to be a sort of patent remedy to all sort of distress and mental malady. (Healy, 2004: 263-265; Moncrieff & Cohen, 2005; Rose, 2007: 201-203.)

### Primary Sources

- Achté, K. & T. Tamminen (eds) (1987) *Lääkäri ja depressiopotilas* (Helsinki: Depressionvastustamistyöryhmä & Ciba-Geigy).
- Achté, K. & T. Tamminen (eds) (1993) *Depressio ja sen hoito* (Klaukkala: Recallmed).
- Blacker, C.V.R. & Clare, A.W. (1987) 'Depressive Disorder in Primary Care.' *British Journal of Psychiatry*, 150: 737-751.
- Beresford Davies, E. (ed) (1964) *Depression. Proceedins of the Symposium Held at Cambridge 22 to 26 September 1959* (Cambridge: Cambridge University Press).
- Curran, D. (1937) 'The differentiation of neuroses and manic-depressive psychoses,' *Journal of Mental Science*, 83: 156-174.
- Depressio - Käypä hoito -suositus. (2004) *Duodecim* 120(2004): 744-764.
- Depressio - tunnistaminen ja hoito. (1995) *Suomen Akatemia ja Suomalainen Lääkäriseura Duodecim*. [SA 1995]
- Depression: Management of depression in primary and secondary care (2004) NICE clinical guideline 23. [NICE 2004]
- Depression: Treatment and management of depression in adults (2009) NICE clinical guideline 90. [NICE 2009]
- Depression in Primary Care. Vol. 1: Detection and Diagnosis. (1993) *Clinical Practice Guideline Nr 5*. U.S. Department of Health and Human Services.
- Depression in Primary Care. Vol. 2: Treatment of Major Depression. (1993) *Clinical Practice Guideline Nr 5*. U.S. Department of Health and Human Services.
- Diagnostic and Statistical Manual of Mental Disorders. Fourth edition, Text revision (2000) Washington, DC: American Psychiatric Association. [DSM-IV-TR]
- Duodecim* 110(1994):3 [Special issue on depression]
- Duodecim* 125(2009):16 [Special issue on depression]
- Endogenous depression. (1959) Manchester: Geigy.
- Freeling, P. (1993) 'Diagnosis and treatment of depression in general Practice,' *British Journal of Psychiatry*, 163, suppl. 20: 14-19.
- Freeling, P. et al. (1985) 'Unrecognised depression in general practice,' *BMJ*, 290: 1880-1883.
- Garmany, G. (1958) 'Depressive states: their aetiology and treatment,' *BMJ*, 2: 341-344.
- Gillespie, R. (1929) 'The clinical differentiation of the types of depression,' *Guy's Hospital Reports* 79: 306-344.
- Goldberg, D. (1979) 'Detection and assessment of emotional disorders in a primary care setting,' *International Journal of Mental Health*, 8: 30-48.
- Hamilton, M. & J.M. White (1959) 'Clinical syndromes in depressive states,' *Journal of Mental Science*, 105: 985-998.
- Henderson, D. & R. Gillespie (1927) *A Text-Book of Psychiatry for Students and Practitioners* (London: Humphrey Milford).
- Henderson, D. & R. Gillespie (1962) *A Text-Book of Psychiatry for Students and Practitioners*, the 9<sup>th</sup>, revised edition (Oxford: Oxford University Press).
- Heron, M.J. (1965) 'A note on the concept endogenous-exogenous,' *British Journal of Medical Psychology*, 38: 241-245.
- Hill, D. (1968) 'Depression: disease, reaction or posture?,' *American Journal of Psychiatry*, 125: 445-457.

- Isometsä, E., M. Henriksson, H. Aro, M. Heikkinen, K. Kuoppasalmi & J. Lönnqvist (1994) 'Suicide in Major Depression.' *American Journal of Psychiatry*, 151: 530-536.
- Katon, W. (1987) 'The Epidemiology of Depression in Primary Care,' *International Journal of Psychiatry and Medicine*, 17: 93-111.
- Katon, W., M. von Korff, E. Lin, E. Walker, G.E. Simon, GE, T. Bush, P. Robinson & J. Russo (1995) 'Collaborative Management to Achieve Treatment Guidelines: Impact on Depression in Primary Care,' *JAMA*, 273: 1026-1031.
- Katon, W. & H. Schulberg (1992) 'Epidemiology of Depression in Primary Care,' *General Hospital Psychiatry*, 14: 237-247.
- Käypä hoito: depression. (2009) [www.kaypahoito.fi](http://www.kaypahoito.fi) consulted 19 Oct 2009.
- Lehtinen, V. (1975) Psykiatrisen hoidon tarve sekä mielisairauteen kohdistuvat asenteet. Kansaneläkelaitoksen julkaisuja: AL 3.
- Lehtinen, V. (ed) (1994) Depression hoito terveyskeskuksessa. Tunnistamis- ja hoitomalli (Helsinki: Stakes).
- Lehtinen, V. & M. Joukamaa (1994) 'Epidemiology of depression: Prevalence, risk factors and treatment situation,' *Acta Psychiatrica Scandinavica*, 89, Supplementum 377: 7-10.
- Lehtinen V. & E. Väisänen (1979) Psykiatrisen sairastavuus ja hoidon tarve Suomessa. Kansaneläkelaitoksen julkaisuja: AL 12.
- Lewis, A. (1934b) 'Melancholia: A clinical survey of depressive states,' *Journal of Mental Science*, 80: 277-378.
- Lewis, A. (1971) "Endogenous' and 'exogenous': a useful dichotomy?" *Psychological Medicine*, 1: 191-196.
- Lönnqvist. J. (1988) 'National suicide prevention project in Finland: A research phase of the project,' *Psychiatria Fennica*, 19: 125-132.
- Mapother, E. (1926) 'Manic-depressive psychosis,' *BMJ*, 2: 872-876.
- Overall, J., L. Hollister, M. Johnson & V. Pennington (1966) 'Nosology of depression and differential response to drugs,' *JAMA*, 195: 946-949.
- Paykel, E.S. & R.G. Priest (1992) 'Recognition and management of depression in general practice: Consensus statement,' *BMJ*, 305: 1198-1202.
- Pollitt, J. (1965) *Depression and its treatment* (London: Heinemann).
- Poutanen, O. (1996a) *Depressio terveyskeskuspotilailla* (Tampere: Tampereen yliopisto).
- Poutanen, O. (1996b) 'Depression in the primary health care patient: Prevalence, recognition, Definition and the consulting situation.' *Psychiatria Fennica*, 27: 115-126.
- Priest, R.G. (1994) 'Improving the Management and Knowledge in General Practice in the UK Educational Programme,' *British Journal of Psychiatry*, 164: 285-287.
- Raitasalo, R. (1977) *Depressio ja sen yhteys psykoterapian tarpeeseen*. Kansaneläkelaitoksen julkaisuja: A 13.
- Regier, D.A., I.D. Goldberg & C.A. Taube (1978) 'The de facto US Mental Health Services System: A Public Health Perspective,' *Archives of General Psychiatry*, 35: 685-693.
- Regier, D.A., R. Hirschfeld, F. Goodwin, J. Burke, J. Lazar & L. Kudd (1988) 'The NIMH Depression Awareness, Recognition, and Treatment Program: Structure, aims, and scientific basis,' *American Journal of Psychiatry*, 145: 1351-1357.
- Rimón, M. and R. Rimón (1992) 'Depression hoito terveyskeskuksessa,' *Suomen Lääkärilehti*, 47: 2381-2385.
- Rutz, W., J. Wålander, G. Eberhard, G. Holmberg, A-L von Knorring, L. von Knorring, B. Wistedt & A. Åberg-Wistedt (1989) 'An Educational Programme

- on Depressive Disorder for General Practitioners on Gotland: Background and Evaluation,' *Acta Psychiatrica Scandinavica*, 79: 19-26.
- Salokangas, R. (1997) *Kliininen depression (Porvoo: WSOY)*.
- Salokangas, R. O. Poutanen & E. Stengård (1995) 'Screening for depression in primary care: Development and validation of the depression scale (DEPS), a screening instrument for depression,' *Acta Psychiatrica Scandinavica*, 92: 10-16.
- Salokangas, R., O. Poutanen, E. Stengård, R. Jähi & T. Palo-oja (1996) 'Prevalence of Depression among Patients Seen in Community Health Centres and Community Mental Health Centres,' *Acta Psychiatrica Scandinavica*, 93: 427-433.
- Schildkraut, J.J. (1965) 'The catecholamine hypothesis of affective disorders: A review of supporting evidence,' *American Journal of Psychiatry*, 122: 509-522.
- Schildkraut, J.J. et al. (1978) 'Toward a biochemical classification of depressive disorders,' *Archives of General Psychiatry*, 35: 1427-1433, 1436-1439.
- Shneidman, E. M. & Ortega (eds) (1969) *Aspects of depression (Boston: Little, Brown & co)*.
- Spigset, O. & B. Mårtensson (1999) 'Drug treatment of depression,' *BMJ*, 318: 1188-1191.
- Spitzer, R., J. Endicott, R.A. Woodruff jr. & N. Andreasen (1977) 'Classification of mood disorders,' in G. Usdin (ed) *Depression: Clinical, biological and psychological perspectives*. New York: Brunner/Mazel.
- Spoov, J. & E. Syvälahti (1996) *Depressiolääkkeet: Käyttöaiheet ja hoidon periaatteet (Helsinki: Lääkelaitos & KELA)*.
- Stengel, E. (1964) 'Appendix I: Classifications of depressions,' in E. Beresford Davies (ed) *Depression. Proceedings of the Symposium Held at Cambridge 22 to 26 September 1959 (Cambridge: Cambridge University Press)*.
- Tamminen, T. (1995) *Depression tunnistaminen: Kliininen diagnostiikka ja hoidon arviointia (Espoo: Novo Nordisk)*.
- Tamminen, T. (2001) *Mielet maasta (Klaukkala: Recallmed)*.
- Tohtori.fi-terveyden tietolähde. <http://www.tohtori.fi/masennusklinikka>; consulted 31 March 2008.
- Väisänen, E. (1975) *Mielenterveyden häiriöt Suomessa. Kansaneläkelaitoksen julkaisuja AL: 2*.
- Widmer, R.B. & R.J. Cadoret (1978) 'Depression in Primary Care: Changes in Pattern of Patient Visits and Complaints during a Development of Depression,' *Journal of Family Practice*, 7: 293-302.

## References

- Akiskal, H. & K. Akiskal (2005) 'The theoretical underpinnings of affective temperaments: implications for evolutionary foundations of bipolar disorders and human nature,' *Journal of Affective Disorders*, 85: 231-239.
- Angst, J. & G. Cassano (2005) 'The mood spectrum: Improving the diagnosis of bipolar disorder,' *Bipolar Disorders*, 7, suppl. 4: 4-12.
- Barber, Ch. (2008) *Comfortably numb (New York: Pantheon)*.
- Callahan, Ch. & G. Berrios (2005) *Reinventing depression (Oxford et al: Oxford University Press)*.
- Cooper, R. (2004) 'What is wrong with the DSM?' *History of Psychiatry*, 15, 5-25.
- Cooper, J.E., R.E. Kendell, B.J. Gurland, L. Sharpe, J.R.M. Copeland & R. Simon (1972) *Psychiatric Diagnosis in New York and London (London: Oxford University Press)*.

- Deleuze, G. (1995) 'Postscript on control societies,' In G. Deleuze: Negotiations 1972-1990 (New York: Columbia University Press).
- Ehrenberg, A. (2010) *The weariness of the self* (Montreal et al: McGill-Queen's University Press).
- Ewald, F. (1991) 'Insurance and risk,' in G. Burchell, C. Gordon & P. Miller (eds) *The Foucault effect* (Hemel Hempstead: Harvester Wheatsheaf).
- Fraser, M. (2001) 'The nature of Prozac,' *History of the Human Sciences*, 14(3): 56-84.
- Grob, G. (1991) *From asylum to community* (Princeton, NJ: Princeton University Press).
- Hacking, I. (2002) *Historical ontology* (Cambridge, MA & London: Harvard University Press).
- Hale, N. (1995) *The rise and crisis of psychoanalysis in the United States* (New York & Oxford: Oxford University Press).
- Hautamäki, L. (2007) 'Kenen tahansa masennus,' *tiede & edistys*, 32: 199-134.
- Hautamäki, L., I. Helén & S. Kanula (2011) 'Mielenterveyden hoidon lääkkeellistyminen Suomessa,' in I. Helén (ed) *Reformin pirstaleet. Mielenterveyspolitiikka hyvinvointivaltion jälkeen* (Tampere: Vastapaino (forthcoming)).
- Healy, D. (1997) *The antidepressant era* (Cambridge, MA & London: Harvard University Press).
- Healy, D. (2002) *The creation of psychopharmacology* (Cambridge, MA & London: Harvard University Press).
- Healy, D. (2004) *Let them eat Prozac* (New York & London: New York University Press).
- Healy, D. (2008) *Mania: A short history of bipolar disorder* (Baltimore: The Johns Hopkins University Press).
- Helén, I. (2006) 'Managing risky moods: Antidepressants and the relation to the self,' Paper presented at Qualitative Research on Mental Health conference, 29 June-1 July 2006, University of Tampere.
- Helén, I. (2007a) 'Multiple depression: Making mood manageable,' *Journal of Medical Humanities*, 28: 149-172.
- Helén, I. (2007b) 'Masennus massamittassa: Epidemiologinen välineistö ja psykiatrian muodonmuutos,' *tiede & edistys*, 32: 103-118.
- Helén, I., P. Hämäläinen & A. Metteri (2011) 'Komplekseja ja katkoksia - Psykiatrian hajaantuminen suomalaisen sosiaalivaltioon,' in I. Helén (ed) *Reformin pirstaleet Mielenterveyspolitiikka hyvinvointivaltion jälkeen*. Tampere: Vastapaino forthcoming.
- Horwitz, A. (2002) *Creating mental illness* (Chicago & London: Chicago University Press).
- Horwitz, A. & J. Wakefield (2007) *The loss of sadness* (Oxford & New York: Oxford University Press).
- Jackson, S. (1986) *Melancholia and depression* (New Haven & London: Yale University Press).
- Jasanoff, S. (2004) 'The idiom of co-production,' in S. Jasanoff (ed) *States of knowledge. The co-production of science and social order* (Milton Park & New York: Routledge).
- Karp, D. (2006) *Is it me or my meds? Living with antidepressants* (Cambridge, MA: Harvard University Press).
- Kessler, R.C., W.T. Chiu, O. Demler & E.E. Walters (2005) 'Prevalence, severity and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication,' *Archives of General Psychiatry*, 62: 617-627.
- Kirk, S. & H. Kutchins (1992) *The selling of DSM* (New York: de Gruyter).

- 
- Kramer, P. (1994) *Listening to Prozac* (London: Fourth Estate).
- Kutchins, H. & Kirk, S. (1997) *Making us crazy: DSM—the psychiatric bible and the creation of mental disorders* (New York: Free Press).
- Lacasse, J.R. & Leo, J. (2005) 'Serotonin and depression: A disconnect between the advertisements and the scientific medicine,' *PLoS Medicine*, 2: 1211-1216.
- Lakoff, A. (2005) *Pharmaceutical reason* (Cambridge: Cambridge University Press).
- Larsen, R.J. (2001) 'Toward a science of mood regulation,' *Psychological Inquiry*, 11: 129-141.
- Lemke, Th. (2000) 'Die Regierung der Risiken: Von der Eugenik zur genetischen Gouvernamentalität,' in U. Bröckling, S. Krasmann & Th. Lemke (eds) *Gouvernamentalität der Gegenwart* (Frankfurt am Main: Suhrkamp).
- Lewis, A. (1934a) 'Melancholia: A historical review,' *Journal of Mental Science*, 80: 1-42.
- Martin, E. (2007) *Bipolar expeditions: Mania and depression in American culture* (Princeton & Oxford: Princeton University Press).
- Mayes, R. & A. Horwitz (2005) 'DSM-III and the revolution in the classification of mental illness,' *Journal of the History of Behavioral Sciences*, 41: 249-267.
- Meyer, A. (1952) *The collected papers of Adolf Meyer* (Baltimore: Johns Hopkins University Press).
- Mol, A. (2002) *The body multiple* (Durham and London: Duke University Press).
- Moncrieff, J. & D. Cohen (2005) 'Rethinking models of psychotropic drug action,' *Psychotherapy and Psychosomatics*, 74: 145-153.
- Rabkin, J. & D. Klein (1987) 'The clinical measurement of depressive disorders,' in A.J. Marsella, R.M.A. Hirschfeld & M. M. Katz (eds) *The measurement of depression* (Chichester: John Wiley).
- Radden, J. (2003) 'Is this dame melancholy? Equating today's depression and past melancholia,' *Philosophy, Psychiatry & Psychology*, 10: 37-52.
- Rose, N. (1996) *Inventing our selves*. Cambridge & New York: Cambridge University Press.
- Rose, N. (2004) 'Becoming of neurochemical selves,' in N. Stehr (ed) *Biotechnology: Between commerce and civil society*. New Brunswick & London: Transaction.
- Rose, N. (2006) 'Disorders without borders? The expanding scope of psychiatric practice,' *BioSocieties*, 1: 465-484.
- Rose, N. (2007) *Politics of life itself* (Princeton & Oxford: Princeton University Press).
- Sartorius, N. & T.A. Ban (1986) *Assessment of depression* (Berlin & New York: Springer).
- Schmidt-Degenhard, M. (1983) *Melancholie und Depression* (Stuttgart: Kohlhammer)
- Singh, I. & Rose, N. (2009) 'Biomarkers in psychiatry,' *Nature*, 460: 202-207.
- Snaith, Ph. (1993) 'What do depression rating scales measure?,' *British Journal of Psychiatry*, 163: 293-298.
- Solomon, A. (2002) *The noonday demon* (New York et al: Touchstone).
- Thomson, M. (1995) 'Mental hygiene as an international movement,' in P. Weindling (ed) *International health organisations and movements 1918-1939* (Cambridge: Cambridge University Press).
- WHO (2001) *Mental health: New understanding, new hope* (Geneva: WHO).
- Whybrow, P. (1997) *A mood apart* (New York: Basic Books).
- Whybrow, P., H. Akiskal & W. McKinney (1984) *Mood disorders: Toward a new psychobiology* (New York & London: Plenum).

Wilson, M. (1993) 'DSM-III and the transformation of American psychiatry: A history', *American Journal of Psychiatry*, 150: 399-410.

Wittchen, H.U., B. Jönsson & J. Oleson (2005) 'Towards a better understanding of the size and burden and cost of brain disorders in Europe', *European Neuropsychopharmacology*, 15: 355-356.

Ilpo Helén  
P.O.Box 18 (Unioninkatu 35)  
00014 University of Helsinki  
Finland  
ilpo.helen@helsinki.fi