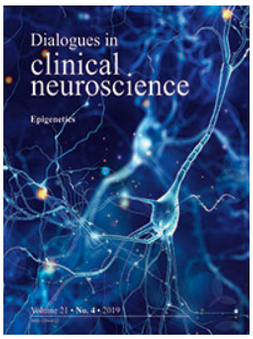

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Basic concepts of depression

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This paper reviews concepts of depression, including history and classification. The original broad concept of melancholia included all forms of quiet insanity. The term depression began to appear in the nineteenth century, as did the modern concept of affective disorders, with the core disturbance now viewed as one of mood. The 1980s saw the introduction of defined criteria into official diagnostic schemes. The modern separation into unipolar and bipolar disorder was introduced following empirical research by Angst and Perris in the 1960s. The partially overlapping distinctions between psychotic and neurotic depression, and between endogenous and reactive depression, started to generate debate in the 1920s, with considerable multivariate research in the 1960s. The symptom element in endogenous depression currently survives in melancholia or somatic syndrome. Life stress is common in various depressive pictures. Dysthymia, a valuable diagnosis, represents a form of what was regarded earlier as neurotic depression. Other subtypes are also discussed.

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Historical background

Prior to the late 19th century, although detailed systems of classification abounded, the main problem for psychiatric nosology was the establishment of the broad major disorders. Melancholia was recognized as early as the time of Hippocrates, and continued through Galenic medicine and medieval times. The earlier connotation of the term was very wide, and included all forms of quiet insanity. It was linked with the humoral theory of causation, specifically, as the term indicates, with black bile.

Most psychiatric terms have changed meaning over their history, and they are always partly dependent on language. Melancholia later became more clearly associated with the more modern idea of melancholy or despair, for instance, in the classic work of the English Renaissance author, Richard Burton, *The Anatomy of Melancholy*,[1] first published in 1621. The alternation of melancholia and mania in what is now termed bipolar disorder or manic-depressive disorder, although in some respects suggested in the writings of Arateus of Cappadocia, and those of later authors, was not clearly described until 1854, independently by the French psychiatrists, Falret and Baillarger. The term depression also began to appear in the 19th century, to indicate a state of sadness. Detailed accounts of these aspects and later history can be found in Jackson[2] and Berrios.[3]

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When Kraepelin, in the late nineteenth century, built on the work of his predecessors and simplified it to delineate the foundations of the modern classification of psychiatric disorders, one of his major categories was that of manic-depressive insanity. Kraepelin's classic textbook went through successive editions, which included some changes in his views. Initially he distinguished a further category, involutional melancholia, but in later editions[4] he returned it to the manic-depressive category. The latter not only included cases of alternating mania and melancholia, but all cases of mania, and seemed to include all depressions.
Kraepelin regarded psychiatric disorders as disease entities based on a medical, neurological model, with specific, organic etiology and pathology. He believed that manic-depressive insanity was largely independent of psychological stress. While such stress might precede the onset of some attacks, it could not be the true cause, but merely something akin to a trigger mechanism. He did, however, regard some pathological depressions as psychogenic in origin. While he did not completely clarify his views on their position in his classification, or how they were to be distinguished from manic-depressive illness with incidental stress, he appeared to regard them as a separate, but relatively small and unimportant, group.
At the same time as Kraepelin and others were establishing a generally accepted classification of the major psychiatric disorders in terms of disease entities based on a medical model and organic etiology, another growing school of European psychiatrists were developing a very different approach. These were the psychoanalysts. Freud and Abraham, in a perceptive group of studies, developed a theory of the origin of depression in relation to actual or symbolic losses of a love object. Here was a theory regarding the origin of most, if not all, depressions as psychogenic.
The case material of Kraepelin, and others like him, consisted of severely ill patients in institutions. The first depressed patients studied psychoanalytically were also severely ill. Subsequently, increasing attention began to be paid to milder forms of disorders, at first particularly by the psychoanalysts. Psychological theories of causation became more wide1y accepted for these disorders. A challenge now arose as to how to reconcile these theories with older ones of organic causation. Adolf Meyer, a Swiss psychiatrist who became the highly influential head of the Henry Phipps Psychiatric Clinic at Johns Hopkins University, moved away from the idea of clearcut disease entities, and viewed all psychiatric disorders as reaction types, or psychobiological reactions of the organism to stress.[5] Both psychological and organic factors had to be taken into account. Others preferred to retain a view which kept separate the two types of psychiatric disorders. On one hand were the psychoses, severe illnesses requiring admission to an asylum, and presumed to have organic causes. On the other hand were the neuroses, milder and not requiring admission to an institution, regarded as more related to psychological stress, and amenable to psychological treatment. The stage was now set for two competing theories as to the classification of depression, which were to figure strongly in debates about subtypes in later years, and will be reviewed in due course.

The modern concept of depression

The modern concept of depression, as viewed by most psychiatrists and enshrined in the two official classifications, *The ICD-10 Classification of Mental and Behavioral Disorders. Clinical descriptions and diagnostic guidelines (ICD 10)*[6] and *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed. *(DSM-IV)*,[7] is essentially one of a clinical syndrome, defined by presence of a number of clinical features, but not requiring a specific etiology, and acknowledging the possibility of both psychological and biological causative factors in a somewhat Meyerian way. *DSM-IV* does exclude states where the symptoms are "better accounted for by bereavement," an imprecise criterion, which is expanded by specifications of not persisting for longer than 2 months, or characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation. The value of this exclusion has been debated.[8] Evidence from symptom studies indicates considerable similarities to nonbereavement depression. Further studies are still needed, particularly some which focus on the 2-month period which is crucial in the *DSM-IV* definition, and include investigations which ask if the picture of bereavement depressions in this period is different from other depressions, and whether they subside or continue outside this time.
This definition of depression is essentially syndromal and medical, resembling that of a syndrome in other fields of medicine. This implies a cluster of symptoms and signs which tend to occur together, which are assumed to reflect a common pathophysiology, that may not yet be understood, but may have diverse etiologies in different cases. Examples from internal medicine include the malabsorption syndrome, and congestive cardiac failure.

This is an aspect of the medical theory of diseases. In the medical concept each disease is regarded as having a specific, well defined etiology, pathology, clinical picture, and often a specific treatment. The advantages of being able to assign individuals to the correct disease have been great. Essentially, as pointed out many years ago by a philosopher, C. G. Hempel,[9] they involve generalization of information. Once a patient is correctly diagnosed, much additional information is available regarding such aspects as underlying mechanisms, causation, prediction of outcome, and best treatment.

A syndrome at the level indicated above does not correspond fully to a disease, since multiple causes, and therefore separate diseases, may underlie it. In psychiatry, matters are more complex and often not clearcut. Different syndromes may overlap and co-occur. Defining pure diseases by etiology has generally not succeeded, since causes often appear to be multiple, even in the single case, and not all etiological factors are known. Nevertheless, many of the above advantages do apply to syndromal diagnoses, including assignment of appropriate treatment and prediction of outcome. It is possible that, as genes involved in psychiatric disorders become elucidated, endophenotypes reflected in underlying disturbances, and genetically defined disorders, may come to correspond more closely to true diseases.

The classical method of identifying a disorder, for most of the history of psychiatry, was for the influential psychiatrist to discern and describe disorders based on his or her clinical experience, with little attempt at precise definition or method-based research. The main method of forming diagnoses in modern psychiatric nosology has been by committee agreement, based sometimes on quite limited empirical research. Diagnostic criteria are then defined by listing certain symptoms, to define the number necessary for the diagnosis, with duration of time, other requirements, and exclusions. In *DSM-IV*, eight symptoms are listed as qualifying for major depression, with a requirement that at least five be present, including at least one of two core symptoms, together with duration of 2 weeks or more, presence of clinically significant distress or impairment of function, with absence respectively of mixed episode, direct effects of a drug of abuse, a medication or other substance, or of a general medical condition, or of bereavement, and for depressive disorder, of bipolar disorder or certain other psychotic diagnoses. For dysthymia, fewer symptoms are required, but for a longer period of 2 years, and from a shorter list of eligible symptoms.

For *ICD-10* depressive episode, the definitions in the clinical criteria are not tightly specified, but they are well specified in the separate Research Criteria, where they tend to be more restrictive than in the clinical criteria. The Research Criteria are less used, and the existence of two different sets of criteria in the classification causes some obvious problems. Eligible symptoms for depressive episode are the same as in *DSM-IV*, with the addition of one further symptom, loss of confidence or self-esteem, with the number of symptoms required to be present depending on the severity of the episode, and a third symptom, fatigue, placed as eligible with the two other core symptoms, rather than in the additional list. There is an identical minimum length, of 2 weeks, and somewhat similar excluding criteria, but without specifying bereavement. The list of eligible symptoms for dysthymia is longer, with three required.

The core symptoms

The core symptoms of depression, of which at least one is required in *DSM-IV*, are depressed mood, and loss of interest or pleasure. The further eligible symptom in *ICD-10* is decreased energy or fatigability, but, since two core symptoms must be present, in effect depressed mood or loss of interest/pleasure are required in this schema also. The reason for the addition of decreased energy to the core is not clear.

These core symptoms reflect the view that depressive disorder is essentially a disorder of mood or affect. Although textbooks have suggested variously that the word "affect" should refer to short-term states or states which are observable, and "mood" for sustained ones or internal ones, there has been much confusion regarding these two terms.[10] In practice they have long been used as more or less synonymous. The term "depression" came into use in the 19th century, originally as "mental depression," to describe lowering of spirits, and came to replace melancholia as a diagnosis. The English-language word really uses an analogy, and its earlier, and also still valid, meaning relates to being pressed down, or an area of something which is pressed down. It can now also refer to other quite different phenomena of lowering, one economic and one barometric, as used in meteorology.

The modern word for loss of interest and pleasure, anhedonia, came into official English-speaking psychiatry with *DSM-III*.[3] The absence of pleasure, and occurrence of feelings of emptiness and flatness rather than overt sad-

ness, have often been described as occurring in some depressions, particularly the more severe ones.
The assignment of mood lowering to centrality in the disorder is to some extent a Western concept, and a more recent one. Other aspects of the disorder such as behavioral change were seen as more important earlier. In other cultures, physical and other disturbances may be seen as more important, and may be reflected in the terms used to describe what appears to be the same disorder.
A more neurobiological view would regard all these phenomena as peripheral and subjective, and would regard the key disturbance as a neurobiological one, not yet adequately elucidated, which drives the other phenomena.

Additional symptoms

The additional symptoms which contribute towards a diagnosis of depression in the two official schemes are: appetite or weight loss or gain; insomnia or hypersomnia; agitation or retardation; loss of energy or fatigue (*DSM-IV*); loss of confidence or self-esteem (*ICD-10*); worthlessness or guilt; reduced concentration or indecisiveness; thoughts of suicide or suicide attempt. The wording and definitions are not always identical in the two schemes. Some of these symptoms are not specific, as they can be caused by other physical or mental disorders. For some, such as pathological guilt, this is not the case, and the placing in the subsidiary list does seem to reflect more the view that the mood disturbance is central.
The diagnostic concept as reflected in the use of criteria which in essence count the number of symptoms present has not usually been formulated explicitly. What there appears to be is an assumption that as the disease becomes more severe it also becomes more pervasive, sucking in more of the accompanying symptoms and disturbances. There are not many empirical studies which have looked directly at the validity of this assumption. Studies using latent trait analyses have tended to produce a dimension corresponding to a list of core symptoms.[11,12] Clinically, as depression becomes more severe, it does also tend to involve the presence of more symptoms.
There have been some studies which have examined the frequency of symptoms present in diagnosed depressions. A classic study was that of Aaron Beck.[13] In an early phase of the work that led ultimately to the genesis of cognitive therapy, and more immediately to his well-known Beck Depression Inventory, he and his colleagues tabulated the frequency of symptoms in a large sample of psychiatric patients. Dividing depressive symptoms into emotional, cognitive, motivational, physical and vegetative, and delusions, they showed that all increased with severity of depression present, and all except delusions were common with severe depression.

Classification

Depressive disorders have long been recognized as heterogeneous. Their subclassification has generated as much research, and as much heat, as any controversy in psychiatry.
The two official schemes are parallel, but not identical, and neither is entirely satisfactory.[14] *DSM-IV* is simpler. Its major categories are depressive disorders and bipolar disorders. Both have subcategories. Within depressive disorders (unipolar depression), the main concern of this paper, the major subcategories are major depressive disorder (itself divided into single episode and recurrent disorder), dysthymic disorder, and the catch-all required to make any official scheme comprehensive for all users, depressive disorder not otherwise specified. The most recent episode can be additionally specified by a set of severity/psychotic/remission specifiers; as chronic; with catatonic features; with melancholic features; with atypical features; with postpartum onset. There is also a further major category for other mood disorders, which include mood disorders due to general medical conditions and substance-induced mood disorder.
In *ICD-10* the major categories are manic episode; bipolar affective disorder; depressive episode; recurrent depressive disorder; persistent mood (affective) disorders (dysthymia, cyclothymia); other mood (affective) disorders; unspecified mood (affective) disorder. The two major axes are really bipolar-unipolar, and course (single episode, recurrent, persistent). Within any depressive episode, single or recurrent, there are subcategories by severity (mild, moderate, severe without psychotic symptoms, with psychotic symptoms, in remission for recurrent disorders) and an additional specifier is available for somatic syndrome (melancholia).
DSM-III and *ICD-10* represented quite major advances on their predecessors, *DSM-II* (rooted much more in psychoanalytic and Meyerian concepts of reaction types) and *ICD-9*, by their use of structured criteria and their use of modern concepts. Structured criteria were used particularly in *DSM-III* and successors. *ICD-10* is

ambiguous in this respect, with its two sets of criteria, the Research Criteria which are well defined, the clinical criteria which are not. Both classifications do also have disadvantages.[14] They are complex, in their fine categories. They are not identical, and, national susceptibilities aside, would be much better fused to a single classification, employing the advantages of each, without the disadvantages, sometimes different, that each has. The strong separation into single episode and recurrent is not justified by empirical research, and it is not useful as a major division: all disorders which become recurrent are single episode on the first occasion. The DSM definitions are better. The specification in *DSM-III* of depressions related to medical disorder and to substance use is not helpful, since there is little to show they differ from the rest of depressions in any major ways.

Bipolar and unipolar disorder

Much of the discussion about the nosology of affective disorder concerns various subtypes. Depression was for many years a fertile ground for classifiers.[15,16] Although much of the heat and pressure have subsided, the issues still complicate diagnostic schemes.

The best-accepted and best-substantiated distinction is the bipolar-unipolar one. This was not always so. As described above, Kraepelin viewed all affective disorders as manic-depressive. As late as *ICD-9*, published in 1978, the ICD did not clearly make the separation, although hidden within the subcategories of manic-depressive disorder (296) for readers of very small print, was a distinction between 296.1, manic-depressive, depressed, which was meant to be unipolar, and 296.3, manic-depressive, circular, depressed, which was meant to be bipolar. Most users of the classification did not realize this, so the distinction was in practice very erratically recorded. The unipolar-bipolar distinction was incorporated into *DSM-III* when it was issued in 1980, and later into the ICD when *ICD-10* was issued.

It was pathfinding work in the 1960s by Angst[17] and Perris[18] that established the value of the distinction. They had been influenced by descriptions by Karl Leonhard, a 20th-century German psychiatrist with a very 19th-century approach to nosology based on his mental hospital clinical experience, of monopolar and bipolar cycloid psychoses.[19]

The bipolar-unipolar distinction is clearcut by definition, depending on the occurrence of a manic episode. Usually it is also so in practice, although late first manic episodes lead to embarrassing changes of diagnosis, and it is hard to be sure of the nature of minor mood elevations, in some cases which are regarded as bipolar II disorder or cyclothymic disorder, or in some subjects with milder mood changes in community epidemiology studies. The status of single-episode mania is debated, but is accepted by most as indicating true bipolar disorder. Some would regard recurrent depression as related to bipolar disorder, but there is not good evidence that this is the case. There are good validating features for the distinction.[20,21] Bipolar disorder is more familial, and there is much more evidence of bipolar disorder in first-degree relatives of bipolars than in relatives of unipolars, although about half the cases of affective disorder in the relatives of bipolars are nevertheless unipolar. There is also better evidence from twin studies that the familial elevation is genetic. Molecular genetic evidence of different genes could confirm the distinction, but this evidence is not yet clearcut. There is a different sex ratio in bipolar disorder, equal or nearly so, possibly a more equal social class distribution, and some association with milder cyclothymic disorder, although the full status of more recent work on cyclothymia still requires confirmation by validating studies. Treatment response differs, with a better response to maintenance lithium and possibly to anticonvulsants, although in unipolars the evidence is not yet adequate. More manic episodes occur on antidepressants. Bipolar disorder has an earlier onset than severe unipolar disorder, and tends to be more recurrent. Onsets in women are not uncommonly postpartum, particularly in the case of mania.

The present review mainly concerns unipolar depression. There have been a number of recent reports comparing bipolar and unipolar depressions.[22-24] In addition to the history features indicated above, bipolar depressions have variously been reported to show more of the following symptom features compared with unipolar: more retardation, hypersomnia, anxiety, mood lability, psychotic features (especially when the age is under 35); less evidence of sad mood, and various somatic complaints. However, often the pictures are indistinguishable.

Psychotic depression and melancholia/somatic syndrome

The greatest controversy of a previous era concerned a dualistic theory of depression, with a dichotomy between

what was variously termed psychotic or endogenous depression on the one hand, and neurotic or reactive depression on the other. Starting in the later 1920s, and throughout the 1930s, fierce debates took place, particularly in British psychiatry, between those advancing a dualistic view and those taking a unitary stance, viewing all depressions as part of a single disorder, without any clear separation into subtypes.[15] The debate subsided with the greater preoccupations of World War II, and reappeared in the form of empirical studies using multivariate statistics in the 1960s.[16]

Terminology was confused. The term "psychotic" refers to a severe disorder with delusions and hallucinations, "neurotic" to a milder disorder without these, and often with the connotation of a vulnerable personality. "Endogenous" and "reactive" refer in this context to absence or presence of life stress. The reason for the partial fusion is that, in the fully evolved concept, there were viewed as three aspects: (i) absence of life stress; (ii) presence of a clinical picture characterized by greater severity, sometimes delusions or hallucinations, diurnal variation with morning worsening, delayed insomnia with early-morning wakening, greater somatic disturbances such as loss of appetite and weight, psychomotor retardation or agitation; this was the so-called endogenous clinical picture, or what in the 1970s was termed endogenomorphic depression[25]; (iii) a personality, associated with reactive or neurotic depression, which was stress-vulnerable or maladaptive.

Over time, the concept of psychotic depression has become separated from that of endogenous depression. Psychotic depression has retained a secure place in the official schemes, as a variant of severe depression. It is clearly definable, by presence of delusions (particularly if mood-congruent) or hallucinations and there is validating evidence, for instance in the better response of such depressions to electroconvulsive therapy (ECT) or antipsychotic drugs, than to antidepressants alone.

Endogenous depression and its opposite are more problematic, both regarding classificatory status and terminology. There is evidence in support. The factor-analytic and cluster-analytic studies of the 1960s and 1970s in most cases found a dimension or group.[26] On detailed examination, this sometimes looks more like the psychotic element and sometimes the melancholic. However, neurotic depression did not emerge as clearly as a single group in these studies, and is heterogeneous.[26] Dexamethasone nonsuppression occurs predominantly in the endogenous group, and to some extent, so do other neuroendocrine abnormalities, such as blunting of growth hormone response to clonidine and prolactin response to tryptophan. Regarding treatment, the best ECT response is associated with the presence of psychomotor retardation and depressive delusions, characteristic of psychotic depression.[27] The endogenous picture may be useful as a characteristic of depressions that respond better to antidepressants than placebo, but this is not clear. However, boundaries are weak, with mixed cases common, and distributions on factors do not show consistent and convincing bimodality which would indicate separation of disorders. The relationship to severity, the loose and confusing definitions, and the overlap between psychotic depression and melancholia bedevil the area.

Terminology has remained unsatisfactory. The term neurotic has dropped out of use, particularly in American psychiatry, where it was abandoned because of its diversity of meaning,[28] partly because of previous associations with psychoanalysis, and partly to avoid the emphasis placed earlier in the US on personality and characterological aspects. Dysthymia, a chronic disorder that would earlier have been regarded as one form of neurotic depression, is now viewed as a mood disorder. In a somewhat parallel way, the term cyclothymic personality has been replaced by cyclothymic disorder, a form of bipolar mood disorder. The term endogenous was abandoned in official schemes because it is really a symptom syndrome that we refer to these days. The term melancholia, used in *DSM-III* and its successors, is only nonloaded once its original meaning of black bile is forgotten. It seems preferable to the term somatic depression used in *ICD-10*, because it is easier to use in English, where it easily forms the adjective melancholic. The concept of somatic depression can also refer to something quite different, associated with somatic disease, or with somatization. Whether this classification will survive forthcoming revisions of the official schemes in the next few years remains to be seen, but it still figures extensively in research and the literature. A spirited case has recently been made for its retention.[29]

The place of life stress in this distinction has changed considerably. In older views of endogenous and nonendogenous depression, life stress had a central role. However, the distinction is now made on the basis of symptom pattern rather than causal factors. Studies[30] have shown that there is little relationship between measures of preceding life events and the presence of melan-

cholic symptoms. Most depressions are preceded by some life stress, often not sufficient to fully account for the episode so that other factors are also involved. In three of our own studies, in two of which the symptom data and the life event data were collected by different interviewers, we found little relationship between symptom type and previous life events.[30] Other studies of patients with and without the symptom pattern have found little difference between the groups regarding the occurrence of stressful life events prior to onset. However, there may be some differences once depressions have become severe and recurrent. In a sample of depressed females, when a melancholic/psychotic score based on the presence and severity of biological and psychotic symptoms was used, then severe life events were significantly less frequent in the melancholic/psychotic group.[31] This significant difference emerged only when episodes other than the first were included. In another study,[32] in which the sample comprised highly recurrent depressives, fewer life events were found in endogenous than nonendogenous Research Diagnostic Criteria subtypes, which depend on symptom features.

Severity and minor depression

The severity issue deserves further consideration. It is elevated to an important consideration in *ICD-10*. As an episode qualifier it is useful, since severity does carry implications for treatment, and severe depressions also tend to have worse outcome than do mild. It is not well recognized that, in practice, *ICD-10* mild depressive episode is by no means minor, at least in the Research Criteria. The definitions for individual symptoms and the absence of some symptoms from the list means that subjects who fit these criteria usually have sufficient depression also to qualify as *DSM-IV* major depressives.

This raises another issue, the lower boundary to distinguish pathological depression from normal mood change. Although defined by the number of symptoms present, it is not in fact well-defined, since the thresholds for individual symptoms are not clear or easy to be sure about: when does lowering of mood, even if present every day, cross the threshold in severity to count as being present? The issue is not crucial in the clinic, but it has become important as psychiatric research has extended to the community, and to community epidemiology. Comparatively high rates of depression are found in community prevalence studies.[33] It is not clear whether all these depressions share fully the qualities of depression presenting for medical or psychiatric treatment. Similar issues arise in the use of "symptomatic volunteers" for research.

There have been a number of studies examining boundaries of DSM major depression.[11,12,34-36] These do support the validity of summing the number of symptoms, although it is doubtful whether there is any true threshold rather than a somewhat arbitrary cutoff on a continuum, and one which as defined may be a little too high. In terms of treatment response, there is good evidence of a threshold, a little below major depression, at which superiority of tricyclic antidepressants to placebo first appears.[37] The threshold for response to serotonin reuptake inhibitors may be a little lower.[38]

Dysthymia and subsyndromal depression

Dysthymia was introduced into official classifications in *DSM-III*, using a term which had been originated by the 19th-century French psychiatrist, Janet. The concept had previously been introduced for research in the predecessor of *DSM-III*, The Research Diagnostic Criteria, as Intermittent Depression.[39] Partly the use of the term was to avoid use of the term neurotic depression. It reflected the wider modern trend to view such chronic phenomena as primarily disorders of mood rather than of personality.

In the last 20 years dysthymia has proved a useful concept, delineating a form of mood disorder which can produce many problems and have an adverse impact on the life of the sufferer, and it has generated much research.[40] There is a high rate of comorbidity, particularly of anxiety disorders and substance abuse. The majority of dysthymics ultimately also develop an episode of major depression, and such episodes, so-called double depression, have a worse prognosis than pure major depression, both in respect of remission and of recurrence. There appears therefore to be continuity between dysthymia and major depression. The *DSM-IV* definition rules out an episode of major depression in the first 2 years, but the *ICD-10* definition does not. In practice the differentiation of dysthymia from milder chronic major depression or from the residual symptoms with partial remission which frequently occur after major depression,[41] is difficult and may be artificial. There is evidence that dysthymia responds to antidepressants,[42] but controlled trials do not always distinguish uncomplicated dysthymia

from superimposed major depression.

A further, milder chronic disorder which has been delineated, recurrent brief depression,[43] has not received general acceptance. Described as comprising frequent episodes of depressive symptoms sufficiently severe for major depression, but only lasting a few days, it does not appear to be very common in patients presenting for treatment, and has not been found to respond to antidepressants in the few studies which have been undertaken.

There is also another *DSM-IV* diagnosis, minor depression, which is included in an appendix of the manual as a provisional category for research. Minor depression was included in the Research Diagnostic Criteria (RDC), but not in *DSM-III*. Both RDC and the possible criteria in *DSM-IV* refer to episodes of depression milder than major depression, rather than persistent dysthymia. Minor depressive episodes, excluding dysthymia, have been found to be more prevalent than major depression in an epidemiological study.[44]

In recent years, there has been a growing literature regarding so-called subthreshold or subsyndromal depressions, which are common in the community and can cause considerable disability.[45] It is not always clear whether this is episodic or chronic, or residual after major depression and what its overlap is with dysthymia or other milder syndromes.

There may be a case for inclusion of one or more diagnoses equivalent to minor or subsyndromal depression in the official schemes in the future. This would be useful in primary care, and in postpartum depression, where much of the literature refers to mild depressions which are important because of their potential impact on the baby. On the other hand, minor depression as defined in the RDC occurred less commonly than might have been expected, perhaps because by the time the criteria were reached, most depressions also fitted another RDC subcategory which was not included in *DSM-IV*, probable (but not definite) major depression. More research in this area would be timely.

Single depressive episode versus recurrent depression

The strong *ICD-10* distinction between single depressive episode and recurrent depression is not useful. Its appearance in *ICD-10* was rather unexpected, as it has not been used much in the past in affective disorder. Unfortunately, in the light of what we have learned in the last 15 years about the risk of recurrence of depression, the distinction is not helpful. If high proportions of people with their first depressive episodes have further episodes and are redefined later as recurrent, the distinction becomes of little value. There is not much to distinguish first depressive episodes from recurrences in other respects, except where the depression has become quite recurrent, when the role of life stress becomes less, response to treatment poorer, and risk of recurrence higher.

A step change has indeed occurred in conceptualization of depression in the last 30 years. In the 1960s and earlier 1970s, the disorder was seen as an episodic one, with complete remission and often without recurrence. Since then follow-up studies of hospitalized depressives have shown that at least 60% will be readmitted over 16 years, and rates for recurrence of episodes any severity, not necessarily needing hospital admission, may be up to 90%.[46] We do not yet know if this is true for milder depressions outside hospital, and probably there are many single episodes at community level, linked to stress, which do not recur, but severe depression is undoubtedly a recurrent disorder. Moreover, since remission may be incomplete and partial, and mild and subsyndromal disorder are common in the community and may ultimately be followed by major episodes, it is now common to view depression as often a chronic disorder encompassing, and varying through, a spectrum or continuum.

Other subtypes

There are also some other subtypes. *ICD-10* does not have them, nor do previous DSM versions. This is understandable in view of the need not to clutter official classifications with the evanescent. The problem is that clinicians do commonly recognize and use some of them, but have nowhere to record them. *DSM-IV* does include some which are not coded and seem to be viewed somewhat tentatively.

There are four subtypes among the specifiers in *DSM-IV*, in addition to those already considered and others related to course. The first is postpartum depression. This is potentially important; although the issue goes wider than mood disorder.[14] At present, researchers and others interested in postpartum disorders have a major problem: there is no official way of recording the disorder. Frequencies of treated disorder are unobtainable. Retrospective identification of subjects for follow-up and

other studies is not possible from coded diagnostic records. *ICD-10* has a category of mental disorders associated with the puerperium (F53), but it can only be used if the criteria for disorders coded elsewhere are not met. There is also a qualifier, in the research criteria only, to indicate disorder associated with the puerperium, but as it is not in the clinical guidelines, few people know about it. *DSM-IV* does have a noncoded specifier for postpartum onset which can be applied to major depression, mania, mixed episode, or brief psychotic disorder, but it is limited to these disorders and the onset requirement, which is within 4 weeks of delivery, is too short. Case register and other studies indicate a peak of onsets which goes on longer, up to 3 months.[47] What is needed is a specifier which can apply to any disorder, is coded, and applies to the onset in the first 3 postpartum months. Inclusion of this should be a high priority for the future.
The second specifier is for seasonal depression. There is now a vast literature on seasonal affective disorder and its treatment.[48] It is time that it was included in official classifications.
A third specifier in *DSM-1V* is for atypical depression, defined in terms of increased sleep, increased appetite, and other symptoms. Here, the case for inclusion is less clearcut, and there are arguments in either direction. The concept originally came from William Sargent and colleagues at St Thomas' Hospital, London. The meaning of the term has fluctuated. The originators probably had in mind nonendogenous depression and later, depression with anxiety or anxiety disorder alone, rather than the more recent meaning of the term, which focuses on vegetative symptoms reversed from their usual directions in endogenous depression.[49] The concept has always been associated with response to monoamine oxidase inhibitors (MAOIs), but the evidence that atypical depression in its current meaning is associated with good MAOI response is mainly limited to one very influential US research group. Other evidence would point to anxious or phobic patients, but in general, selectivity appears to be weak, and there is evidence that MAOIs, in a high enough dose, are effective in quite a range of depressives.[50] In practice, it appears that clinicians, at least in Europe, do not use the term as much today as they did in the 1960s and 1970s, and its importance may be diminishing. So, too, is research in relation to it.
The fourth *DSM-IV* specifier is for the presence of catatonic features. This is idiosyncratic, and does not correspond to much in the earlier literature. Its meaning is not very clear, but much of the description seems to be that of psychomotor retardation. Retarded depression does have a considerable lineage, but has not proved a very useful classification and is not very stable between episodes. This subtype could be dropped without loss.
DSM-IV has some other provisional classifications in its appendix. Recurrent brief depression appeared and generated much excitement, but has not proven very useful and now receives less attention, so the case has not been made for its continuing inclusion. Mixed anxiety-depression is common, but can easily be handled by modern ideas of comorbidity and two diagnoses. Premenstrual dysphoric disorder is not purely depressive, and is beyond the scope of this review.

Transcultural aspects

The modern concept of depression, with emphasis on psychological feelings, is particularly Western, and to some extent a 20th-century development. Earlier Western concepts were less psychological. Some other cultures and languages place emphasis on other aspects.[51]
It was thought at one time that mood disorders were less common in other cultures, for instance African, than in Western. In general this does not now appear to be the case, but to have been an artefact of previous Western psychiatrists failing to recognize the disorder in other cultural and linguistic groups. Mood disorders do appear to be universal, once they are sought by local psychiatrists who understand the culture, language, and metaphors used to express mood. Rates may differ to a lesser extent, but this is not clear, since it faces formidable problems in establishing equivalence of translated interview instruments and questionnaires.
Presentations may differ. In Zimbabwe, the language lacks a term directly equivalent to depression, and presentations are typically with somatic symptoms.[52] In Chinese subjects, presentations may also be more somatic, but there is evidence that with Western acculturation, this changes.[53] In Hong Kong,[54] lower rates of depression and higher rates of anxiety have been reported than in similar epidemiological studies from the US and other Western countries, suggesting some redirection of symptoms.
It is also possible that other syndromes limited to one or more cultures may be equivalents of depression. Equivalence to depression is difficult to prove in nondepressed subjects. It would, however, be inappropriate to

imply that these are any less valid than disorders seen in Western cultures. There may also be additional non-Western subtypes which justify inclusion in international diagnostic schemes.

Conclusion

The concepts involved in depression are complex. They have evolved over the years, and often, as is common in psychiatry, the meanings have changed subtly in the process. The core elements, and workable definitions for the disorder and its boundaries, are now well established. Some aspects of classification remain problematic, but the separation of bipolar and unipolar disorder was a major advance. Depressions are the most common disorders in psychiatry, both for psychiatrists and for general practitioners, so that understanding of their elements is important. ❑

REFERENCES

1. Burton R. *The Anatomy of Melancholy*. London, UK: JM Dent and Sons; 1972.
2. Jackson SW. *Melancholia and Depression. From Hippocratic to Modern Times*. New Haven, Conn: Yale University Press; 1986.
3. Berrios GE. *The History of Mental Symptoms. Descriptive Psychopathology Since the Nineteenth Century*. Cambridge, UK: Cambridge University Press. 1996.
4. Kraepelin E. *Manic-Depressive Insanity and Paranoia*. 8th ed. by Barclay RM, trans. Edinburgh, UK: E and S Livingstone; 1921.
5. Meyer A. Interrelations of the domain of neuropsychiatry *Arch Neurol Psychiatry*. 1922;8:111-121.
6. World Health Organization. *The ICD-10 Classification of Mental and Behavioral Disorders. Clinical descriptions and diagnostic guidelines*. Geneva, Switzerland: World Health Organization; 1992.
7. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. Washington, DC: American Psychiatric Association; 1994.
8. Zisook S. Kendler K. Is bereavement-related depression different than non-bereavement-related depression? *Psychol Med*. 2007;37:779-794.
9. Hempel CG. Introduction to problems of taxonomy. In: Zubin J, ed. *Field Studies in Mental Disorders*. New York, NY: Grune and Stratton; 1961.
10. Serby M. Psychiatric resident conceptualizations of mood and affect within the mental status examination. *Am J Psychiatry*. 2003;160:1527-1529.
11. Slade T Andrews G. Latent structure of depression in a community sample: a taxometric analysis. *Psychol Med*. 2005;35:489-497.
12. Aggen SH, Neale MC, Kendler KS. DSM criteria for major depression: evaluating symptom patterns using latent-trait item response models. *Psychol Med*. 2005;35:475-87.
13. Beck AT. *Depression. Clinical, Experimental and Theoretical Aspects*. New York, NY: Hoeber Medical Division, Harper and Row; 1967.
14. Paykel ES. Mood disorders: review of current diagnostic systems. *Psychopathology*. 2002;35:94-99.
15. Kendell RE. *The Classification of Depressive Illness*. Maudsley Monograph No 18. London, UK: Oxford University Press; 1968.
16. Paykel ES Have multivariate statistics contributed to classification? *Br J Psychiatry*. 1981;139:357-362.
17. Angst J. *Zur Ätiologie und Nosologie Endogener Depressiver Psychosen. Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie*. Berlin, Germany: Springer 1966.
18. Perris C. A study of bipolar and unipolar recurrent depressive psychoses. *Acta Psychiatr Scand*. 1966;42(suppl 194).
19. Leonhard K Cycloid psychoses--endogenous psychoses which are neither schizophrenic nor manic-depressive. *J Ment Sci*. 1961;107:633-648.
20. Perris C. Bipolar-unipolar distinction. In: Paykel ES, ed. *Handbook of Affective Disorders*. 2nd ed. Edinburgh, UK: Churchill Livingstone; 1990;57-75.
21. Goodwin FK and Jamison KR. *Manic-Depressive Illness. Bipolar Disorders and Recurrent Depression*. 2nd ed. New York, NY: Oxford University Press; 2007.
22. Muzina DJ, Kemp DE, McIntyre RS. Differentiating bipolar disorders from major depressive disorders: treatment implications. *Ann Clin Psychiatry*. 2007;19:305-312.
23. Perlis RH, Brown E, Baker RW, Nierenberg AA. Clinical features of bipolar depression versus major depressive disorder in large multicenter trials. *Am J Psychiatry*. 2006;163:225-231.
24. Bowden CL. A different depression: clinical distinctions between bipolar and unipolar depression. *J Affect Disord*. 2005;84:117-125.
25. Klein DF. Endogenomorphic depression. A conceptual and terminological revision. *Arch Gen Psychiatry*. 1974;31:447-454.
26. Paykel ES. Classification of depressed patients: A cluster analysis derived grouping. *Brit J Psychiat*. 1971;118:275-288.
27. Buchan H, Johnstone E, McPherson K, Palmer RL, Crow TJ, Brandon S. Who benefits from electroconvulsive therapy? Combined results of the Leicester and Northwick Park trials. *Br J Psychiatry*. 1992;160:355-359.
28. Klerman GL. The scientific status of neurotic depression. *Psychopathology*. 1985;18:167-173.
29. Taylor MA, Fink M. Restoring melancholia in the classification of mood disorders. *J Affect Disord*. 2008;105:1-14.
30. Paykel ES. Life events and affective disorders. *Acta Psychiat Scand*. 2003;108:(suppl 418)61-66.
31. Brown GW, Harris TO, Hepworth C. Life events and endogenous depression: a puzzle reexamined. *Arch Gen Psychiatry*. 1994;51:525-534.
32. Frank E, Anderson B, Reynolds CF, Rietnour A, Kupfer DJ. Life events and the research diagnostic criteria endogenous type. *Arch Gen Psychiatry*. 1994;51:519-524.
33. Paykel ES, Brugha, Fryer T. Size and burden of depressive disorders in Europe. *Eur Neuropsychopharmacol*. 2005;15:411-423.
34. Kendler KS, Gardner CO Jr. Boundaries of major depression: an evaluation of DSM-IV criteria. *Am J Psychiatry*. 1998;155:172-177.
35. Solomon A, Ruscio J, Seeley JR, Lewinsohn PM. A taxometric investigation of unipolar depression in a large community sample. *Psychol Med*. 2006;36:973-985.
36. Sakashita C, Slade T, Andrews G. Empirical investigation of two assumptions in the diagnosis of DSM-IV major depressive episode. *Aust N Z J Psychiatry*. 2007;41:17-23.
37. Paykel ES, Hollyman JA, Freeling P, Sedgwick P. Predictors of therapeutic benefit from amitriptyline in mild depression: a general practice placebo-controlled trial. *J Affect Disord*. 1988;14:83-95.
38. Judd LL, Rapaport MH, Yonkers KA, et al. Randomized, placebo-controlled trial of fluoxetine for acute treatment of minor depressive disorder. *Am J Psychiatry*. 2004;161:1864-1871.
39. Spitzer RL, Endicott J, Robins E. Research diagnostic criteria: rationale and reliability. *Arch Gen Psychiatry* 1978;35:773-782.

Conceptos básicos sobre la depresión

Este artículo revisa conceptos sobre la depresión, los que incluyen historia y clasificación. El amplio concepto original de melancolía incorporaba todas las formas leves de locura. El término depresión comenzó a aparecer en el siglo XIX, y al igual que el concepto moderno de trastornos afectivos, consideró como esencial la alteración del ánimo. En la década de 1980 se produjo la introducción de criterios definidos en esquemas diagnósticos oficiales. La moderna separación entre el trastorno unipolar y bipolar surgió a partir de la investigación empírica de Angst y Perris en la década de 1960. Las distinciones que se traslapan parcialmente entre depresión psicótica y neurótica, y entre depresión endógena y reactiva comenzaron a generar debate en la década de 1920 con una importante investigación de múltiples variables en la década de 1960. El síntoma central de la depresión endógena perdura actualmente en la melancolía o el síndrome somático. El estrés de la vida es común a varios cuadros depresivos. La distimia, un diagnóstico útil, representa una forma de lo que antes se consideró la depresión neurótica. También se discuten otros subtipos de depresión.

Concepts de base de la dépression

Cet article fait le point sur les concepts de la dépression, incluant son histoire et sa classification. À l'origine, la notion large de mélancolie englobait toutes les formes de folie calme. Le terme de dépression est apparu au XIX^e^ siècle, comme le concept moderne de troubles affectifs, les troubles de l'humeur étant aujourd'hui au cœur de la dépression. Des critères précis ont fait leur apparition dans les années 80 avec des arbres diagnostiques officiels. La séparation moderne entre maladie uni- et bipolaire a suivi la recherche empirique d'Angst et Perris dans les années 60. C'est dans les années 20 que les distinctions, bien que comprenant des similarités, entre dépression psychotique et névrotique, et entre dépression endogène et réactive ont commencé à faire débat, générant une recherche considérable dans les années 60. La mélancolie ou le syndrome somatique persistent aujourd'hui comme symptômes dans la dépression endogène. Les événements de vie stressants sont fréquemment retrouvés dans des tableaux dépressifs variés. La dysthymie, entité diagnostique valable, est une forme de ce que l'on appelait autrefois dépression névrotique. L'article analyse également d'autres types de dépression.

40. The WPA Dysthymia Working Group: (Akiskal HS, Costa e Silva JA, Frances A, et al) Dysthymia in clinical practice. *Br J Psychiatry*. 1995;166:174-183.
41. Paykel ES, Ramana R, Cooper Z, Hayhurst, H, Kerr J, Barocka A. Residual symptoms after partial remission: an important outcome in depression. *Psychol Med.* 1995; 25:1171-1180.
42. de Lima MS, Hotoph M, Wessely S. The efficacy of drug treatments for dysthymia: a systematic review and meta-analysis. *Psychol Med*. 1999;29:1273-1289.
43. Pezawas L, Angst J, Kasper S. Recurrent brief depression revisited. *Int Rev Psychiatry*. 2005;17:63-70.
44. Cuijpers P, de Graaf R, van Dorsselaer S. Minor depression: risk profiles, functional disability, health care use and risk of developing major depression. *J Affect Disord*. 2004;79:71-79.
45. Judd LL, Paulus MP, Wells KB, Rapaport MH Socioeconomic burden of subsyndromal depressive symptoms and major depression in a sample of the general population. *Am J Psychiatry*. 1996;153:1411-1417.
46. Kennedy N, Abbott R, Paykel ES. Remission and recurrence of depression in the maintenance era: long-term outcome in a Cambridge cohort. *Psychol Med*. 2003;33:827-838.
47. Kendell RE, Wainwright S, Hailey A, Shannon B. The influence of childbirth on psychiatric morbidity. *Psychol Med*. 1976;6:297-302.
48. Westrin A, Lam RW. Seasonal affective disorder: a clinical update. *Ann Clin Psychiatry*. 2007;19:239-246.
49. Paykel ES, Parker RR, Rowan PR, Rao BM, Taylor CN. Nosology of atypical depression. *Psychol Med*. 1983;13:131-139.
50. Paykel, ES. Clinical efficacy of reversible and selective inhibitors of monoamine oxidase A in major depression. *Acta Psychiat Scand*. 1995;91(suppl 386):22-27.
51. Bhugra D, Bhui K. *Textbook of Cultural Psychiatry*. Cambridge, UK: Cambridge University Press; 2007.
52. Patel V, Abas M, Broadhead J, Todd C, Reeler A. Depression in developing countries: lessons from Zimbabwe. *BMJ*. 2001;322:482-484.
53. Parker G, Chan B, Tully L, Eisenbruch M. Depression in the Chinese: the impact of acculturation. *Psychol Med*. 2005;35:1475-1483.
54. Chen CN, Wong J, Lee N, Chan-Ho MW, Lau JT, Fung M. The Shatin community mental health survey in Hong Kong. II. Major findings. *Arch Gen Psychiatry*. 1993;50:125-133.