

Part I

Unipolar Depression

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The Classification and Epidemiology of Unipolar Depression

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Introduction

In the revised version of this chapter, the original structure has largely been maintained. After all, the principles of classification and the problems it poses for the epidemiology of depression remain the same. However, I have summarized the slow progress toward revision of the ICD and DSM classificatory systems. In the original chapter, I provided illustrative examples of the epidemiology of depression in terms of age, gender, life stress, and childhood antecedents of depression. In the current version, I have, somewhat selectively, updated the account of research in these topics, but have changed the emphasis away from well-established issues such as the effect of gender, and toward the epidemiology of depression in the workplace. This seems particularly relevant in a world where inexorable economic advancement is no longer guaranteed and retrenchment has major effects on work and workers.

Classification and Unipolar Depression

Psychiatric classification is quintessentially a medical procedure. The study of medicine is based on the establishment of separate categories of disorder (illnesses, diseases).¹ These are distinguished in terms of particular types of attributes of the people held to be suffering from them. These attributes comprise *symptoms* (based on self-report) and *signs* (based on observation). Individual disorders are conceived in terms of concatenations of symptoms and signs, which are termed syndromes. Such syndromes are provisional constructs, whose validity (or otherwise) is then established by using them as the basis of different sorts of theory: etiological theories, and theories of course and

¹The terms illness and disease in ordinary usage strongly suggest a biological basis, while disorder is more neutral, and in psychiatry is generally preferred.

outcome, of treatment, and of pathology (Bebbington, 2011; Wing, Mann, Leff, & Nixon, 1978). There is no doubt that the medical approach to malfunction has been a very effective one, generating new knowledge quickly and efficiently by testing out theories of this type (Bebbington, 1997).

Psychiatric disorders are classified in the hope that the classification can provide mutually exclusive categories to which cases can be allocated unambiguously (the process of case identification). The medical discipline of *epidemiology* is the study of the distribution of diseases (i.e., medical classes) in the population, and is based on categories of this type. It has been a very powerful method for identifying candidate causal factors, and is thus of great interest to psychiatrists and psychologists, as well as to clinicians from other specialties.

The idea of unipolar depression involves the application of this syndromal approach to psychological disturbance, and is therefore primarily a medical concept. However, the distinctiveness of psychiatric syndromes is both variable and limited. Unipolar depression, in particular, resembles, overlaps, and needs distinguishing from other disorders characterized by mood disturbance: anxiety disorders, other depressive conditions, and bipolar mood disorder. The particular problem with bipolar disorder is that its identification depends on the presence of two sorts of episode in which the associated mood is either depressed or predominantly elated. It is distinct from unipolar disorder in a variety of ways (e.g., inheritance, course, and outcome), and the distinction is therefore almost certainly a useful one. However, depressive episodes in bipolar disorder cannot be distinguished symptomatically from those of unipolar depression. As perhaps half of all cases of bipolar disorder start with a depressive episode, this means that unipolar depression is a provisional category—the disorder will be reclassified as bipolar in 5% of cases (Ramana & Bebbington, 1995).

Symptoms and Syndromes

The first stage in the establishment of syndromes is the conceptualization of individual symptoms. Symptoms in psychiatry are formulations of aspects of human experience that are held to indicate abnormality. Examples include abnormally depressed mood, impaired concentration, loss of sexual interest, and persistent wakefulness early in the morning. They sometimes conflate what is abnormal for the individual with what is abnormal for the population, but they can generally be defined in terms that are reliable. Signs (which are unreliable and rarely discriminating in psychiatry, and thus tend to be discounted in diagnosis) are the observable concomitants of such experiences, such as observed depressed mood, or behavior that could be interpreted as a response to hallucinations. Particular symptoms (and signs) often coexist in people who are psychologically disturbed, and this encourages the idea that they go together to form recognizable syndromes. The formulation of syndromes is the first stage in the disease approach to medical phenomena, as syndromes can be subjected to investigations that test out the various types of theories described above.

It is often said, in both medical and lay discourse, that psychiatric disorders are like (or just the same as) disorders in physical medicine. This is not strictly true. Self-reports in general medicine relate to bodily sensation and malfunction in a way that

can be linked to pathological processes. Thus the classical progression of symptoms in appendicitis is related straightforwardly to the progression of inflammation from the appendix to the peritoneal lining. Symptoms in psychiatry, in contrast, are essentially based on idiosyncratic mental experiences, with meanings that relate to the social world. Reformulating psychiatric disorders in terms of a supposed biological substrate would therefore result in the conceptualization of a different condition, which would map imperfectly onto the original disorder.

While syndromes are essentially lists of qualifying symptoms and signs, individuals may be classed as having a syndrome while exhibiting only some of the constituent symptoms. Moreover, within a syndrome there may be theoretical and empirical reasons for regarding particular symptoms as having special significance. Other symptoms, however, may be relatively nonspecific, occurring in several syndromes. Even so, clusters of such symptoms may achieve a joint significance. This inequality between symptoms is seen in the syndrome of unipolar depression: depressed mood and anhedonia are usually taken as central, while other symptoms (e.g., fatigue or insomnia) have little significance on their own. This reflects a serious problem with the raw material of human mental experience: it does not lend itself to the establishment of the desired mutually exclusive and jointly exhaustive categories that underpin medical classification.

In an ideal world, all the symptoms making up a syndrome would be discriminating, but this is far from true, and decisions about whether a given subject's symptom pattern can be classed as lying within a syndrome usually show an element of arbitrariness. The result is that two individuals may both be taken to suffer from unipolar depression despite exhibiting considerable symptomatic differences.

This is tied in with the idea of symptom severity: disorders may be regarded as symptomatically severe either from the sheer number of symptoms or because several symptoms are present in severe degree. In practice, disorders with large numbers of symptoms also tend to have a greater severity of individual symptoms. In classifications that rely on relatively few symptoms to establish diagnoses of depressive conditions, the issue of severity may need to be dealt with by including other markers, particularly impairment of social engagement and activity, and disabilities in self-care.

The Limits of Classification

As classification aspires to “carve nature at the joints,” the empirical relationships between psychiatric symptoms create special difficulties of their own. In particular, symptoms are related nonreflexively: thus, some symptoms are common and others are rare, and in general they are hierarchically related, rather than being associated in a random manner. Rare symptoms often predict the presence of common symptoms, but common symptoms do not predict rare symptoms. Deeply (i.e., “pathologically”) depressed mood is commonly associated with more prevalent symptoms such as tension or worry, while in most instances tension and worry are *not* associated with depressed mood (Sturt, 1981). Likewise, depressive delusions are almost invariably associated with depressed mood, whereas most people with depressed mood do not have delusions of any kind. The consequence is that the presence of the rarer,

more “powerful” symptoms indicates a case with many other symptoms as well, and therefore a case that is more symptomatically severe. It is because of this set of empirical relationships between symptoms that psychiatric syndromes are themselves quite largely hierarchically arranged. Thus, schizophrenia is very often accompanied by affective symptoms, although these are not officially part of the syndrome. Likewise, psychotic depression is not distinguished from nonpsychotic depression by having a completely different set of symptoms, but by having extra, discriminating symptoms such as depressive delusions and hallucinations.

Leaky Classes and Comorbidity

The operational criteria set up to identify and distinguish so-called common mental disorders cut across the natural hierarchies existing between symptoms. The consequence is that many people who have one of these disorders also meet the criteria for one or more of the others. This *comorbidity* has generated much interest, and was even incorporated into the titles of recent major US epidemiological surveys (the National Comorbidity Survey and its replication Kessler, McGonagle, Zhao, et al., 1994, Kessler et al., 2003). Researchers then divide into two camps: those who think the comorbidity represents important relationships between well-validated disorders, and those who think it arises as an artifact of a classificatory system that is conceptually flawed and fails adequately to capture the nature of affective disturbance.

Depression and the Threshold Problem

The final difficulty with the classification of depression is that it involves imposing a categorical distinction on a set of phenomena that look more like the expression of a continuum. The empirical distribution of affective symptoms in the general population is characteristic: many people have a few symptoms, while few people have many.

For some authorities, this pattern of distribution calls into question the utility of a medical classification. It certainly makes case definition and case finding contentious, as decisions have to be made about the threshold below which no disorder should be identified. People who have few symptoms may still be above this threshold if some of their symptoms are particularly discriminating, but in general the threshold is defined by the number of symptoms. There has always been a tendency in medicine to move thresholds down, particularly as many people who may be regarded by primary care physicians as meriting treatment fall below the thresholds of DSM-IV or ICD-10.

The threshold problem has encouraged a considerable literature relating to sub-threshold, subclinical, minor, and brief recurrent affective disorders (Schotte & Cooper, 1999). The tendency to extend the threshold downward is apparent in the establishment of the category of *dysthymia*, a depressive condition characterized only by its mildness (i.e., a *lack* of symptoms) and its chronicity. It has, nevertheless, become a study in its own right: it has clear links with major depression presumably because it is relatively easy for someone who already has some depressive symptoms to acquire some more and thereby meet criteria for the more severe disorder.

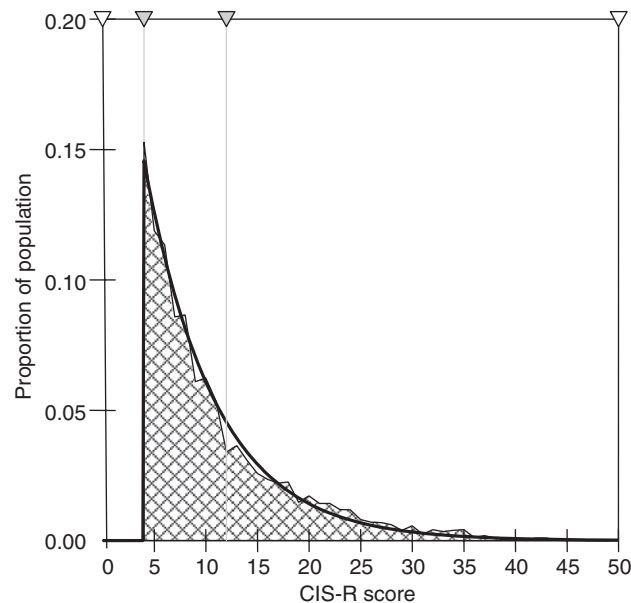


Figure 1.1. Proportion of Population by Truncated Range of CIS-R Scores, and Fitted Exponential Curve. Reproduced with permission from Melzer et al. (2002)

The imposition of a threshold on an apparent continuum would be less arbitrary if it were possible to demonstrate a naturally occurring “step-change” in the distribution. Thus, while the distribution of IQ is largely continuous, Penrose (1963) noted a clear excess of subjects at the bottom of the continuum who are characterized by a distinct and identifiable pathology. Many have argued that no such distinction exists in affective symptoms (Goldberg, 2000; Tyrer, 1985). While it might be possible to create a threshold that represented a step-change in social disability (Hurry, Sturt, Bebbington, & Tennant, 1983), the evidence does, overall, suggest that affective symptoms are distributed more like blood pressure than IQ. Melzer, Tom, Brugha, Fryers, and Meltzer (2002) used symptom data from the British National Survey of Psychiatric Morbidity to test out the smoothness of the distribution. A single exponential curve provided the best fit for the whole population, but there were floor effects that produced deviations at symptom counts from zero to three. Truncation of the data to take account of this provided an excellent fit (Figure 1.1). This was not affected by selecting subgroups characterized by especially high or low prevalence for analysis.

It can be concluded from this discussion that the epidemiological literature on depressive disorder will need to be interpreted cautiously. We have disorders that are identified as classes imposed on what is empirically a continuum, and which in any event overlap each other. This is made worse because the classificatory schemes are changed at regular intervals. Moreover, two major schemes exist side-by-side. Added to this is the issue of how the symptoms of common mental disorders can be elicited, identified, and used in order to decide if together they can be said to constitute a case.

Competing Classifications

The indistinctness of psychiatric syndromes and of the rules for deciding if individual disorders meet symptomatic criteria has major implications for attempts to operationalize psychiatric classifications. Two systems have wide acceptance: the Diagnostic and Statistical Manual (DSM) of the American Psychiatric Association and the World Health Organization's International Classification of Disease (ICD). In the early days, revision of classificatory schemata relied almost wholly on clinical reflection. However, since the classifications are set up primarily for scientific purposes, they should properly be modified in the light of empirical research that permits definitive statements about their utility. The standardized and operationalized classifications now available offer an opportunity for using research in this way, and current attempts to modify them are being based on extensive reviews of the evidence.

In the past, much of the pressure for change originated in clinical and political demands. In particular, the establishment of a diagnosis is central to accessing health care in the United States, in distinction to the more problem-based approach in Europe. Moreover, revisions sometimes had the appearance of tinkering in order to capture some imagined essence of the disorders included (Birley, 1990). What looks like fine-tuning can nevertheless make considerable differences to whether individual cases meet criteria or not, and thus disproportionately affects the putative frequency of disorders. We should only jettison classifications on grounds of inadequate scientific utility and as seldom as possible, since too rapid revision defeats the objective of comparison.

Like all such classifications, DSM and ICD are created by committees. It can be argued that the natural tendency for horse trading between experts selected precisely because they are powerful and opinionated has led to an overelaborate structure, an excess of allowable classes and subclasses, and complicated defining criteria. Thus, in DSM-IV (American Psychiatric Association [APA], 1994) there are potentially 14 categories to consider before allocating someone with depressed mood, and in ICD-10 (World Health Organization [WHO], 1992b) there are 22. Greater utility would probably accrue from limiting the primary categories to three (bipolar disorder, unipolar depressive psychosis, and unipolar nonpsychotic depression), and epidemiological research often uses these categories in any case.

Box 1.1 provides a comparison of the definitions of depressive disorder under DSM-IV (APA, 1994) and ICD-10 (WHO, 1992b), slightly simplified. Over the years there has been considerable convergence between the systems. Nevertheless, the differences remain important. The categories are too close together for empirical studies to establish their relative validity, as this would demand enormous samples. However, they are far enough apart to cause discrepancies in identification. Relatively severe cases are likely to be classified as a depressive disorder under both systems. However, milder disorders may be cases under one system and not the other. This becomes important in epidemiological studies of depressive disorder in the general population because such studies usually report their results under one system or the other, and the degree of comparability is hard to quantify. Thus, the use of different classificatory systems is one barrier to comparison between studies; there are others.

Box 1.1 Criteria for depressive episode*DSM-III-R/DSM-IV**ICD-10*

Symptoms present nearly every day in the same 2-week period

Change from normal functioning

Key symptoms (n = 2)

Depressed mood

Anhedonia

Ancillary symptoms (n = 7)

Fatigue/loss of energy

Weight/appetite loss/gain

Insomnia/hypersomnia

Observed agitation/retardation

Low self-esteem/guilt

Impaired thinking/concentration

Suicidal thoughts

Criteria: 1 key, 5 symptoms in total

Plus

Significant distress

Or

Social impairment

Exclusions

Not mixed episode

Not substance related

Not organic

Not bereavement

Not psychotic

Episode must have lasted at least 2

weeks with symptoms nearly every day

Change from normal functioning

Key symptoms (n = 3)

Depressed mood

Anhedonia

Fatigue/loss of energy

Ancillary symptoms (n = 7)

Weight and appetite change

Sleep disturbance

Subjective or objective

Agitation/retardation

Low self-esteem/confidence

Self-reproach/guilt

Impaired thinking/concentration

Suicidal thoughts

Criteria

Mild episode: 2 key, 4 symptoms in total

Moderate episode: 2 key, 6 symptoms in total

Severe episode: 3 key, 8 symptoms in total

Exclusions

No history (ever) of manic symptoms

Not substance related

Not organic

It is interesting to see the effect of applying algorithms for the diagnostic categories defined by different systems to a common set of symptom data. The Schedules for Clinical Assessment in Neuropsychiatry (SCAN; WHO, 1992a) allow diagnosis under both DSM and ICD. Table 1.1 shows the effect of applying ICD-10 and DSM-IV criteria to the dataset from a community survey (McConnell, McClelland, Gillespie, Bebbington, & Houghton, 2002) on the identification of cases of depressive episode (ICD) and depressive disorder (DSM). Of the 18 participants diagnosed with a depressive condition by either classification, two-thirds were diagnosed by both. Five cases of depressive episode were not diagnosed as DSM depressive disorder, whereas only one case of depressive disorder was not diagnosed as an ICD depressive episode. In contrast, DSM recognized many more cases of anxiety disorder. Fifteen of the cases defined by DSM were not classed as anxiety disorders by ICD, while only two classified

Table 1.1.

DSM-III-R and ICD-10 Classification Based on the Same Symptom Data: The Derry Survey

	<i>No depressive diagnosis</i>	<i>Depressive disorder DSM</i>
No depressive diagnosis	289 (94%)	1 (0.3%)
Depressive episode ICD-10	5 (1.6%)	12 (3.9%)
Kappa		0.79
	<i>No anxiety diagnosis</i>	<i>Anxiety disorder DSM</i>
No anxiety diagnosis	269 (87%)	15 (4.9%)
Anxiety disorder ICD	2 (0.7%)	21 (6.8%)
Kappa		0.68

by ICD were not so classed by DSM. Thus, the ICD criteria appear to be less stringent for depressive episode, while the reverse is true of anxiety. The results suggest that the difference between the two systems arises because of differing thresholds rather than because of wide differences in the symptom contents of the classes.

Revising the Classifications

It has been planned to publish new versions of both dominant classificatory systems: DSM-V in 2013, and ICD-11 in 2015. Around 20 years will then have passed since the previous revision of each system (Sartorius, 2010). This is a much longer gap than between previous editions, and represents an improvement: science is not well served by too frequent revision, which needlessly obstructs the possibility of comparison between studies. Some of the delay was inevitable, given the increasing complexity of the process of revision. It involves very many stakeholders, and the establishment by WHO and APA, respectively, of taskforces, advisory groups, and subgroups. The obligation to consult widely involves enormous amounts of work and demands complex processes of integration.

Three very appropriate criteria have been set out for the removal or introduction of categories. These relate to public health, practical utility, and empirical evidence (Sartorius, 2010). However, the precise application of the criteria is likely to lead to disagreement. The gathering and evaluation of the relevant empirical evidence is extremely time-consuming. Moreover, despite the rigor of the procedures involved, political considerations may sometimes trump these three criteria. This is not surprising: classification can have consequences for the way particular university departments and disciplines are funded, the licensed use of drugs is related to diagnostic entities, and, in the United States particularly, so is insurance cover for specific treatments.

A harmonization group has been set up, tasked with the work of ensuring that DSM-V and ICD-11 are as closely compatible as possible in their classificatory procedures. First (2009) has provided an impressively exhaustive article (particularly in its online version) on the potential for harmonization. Of the 176 sets of criteria that correspond in the ICD-10 and DSM-IV systems, only one was identical. Twenty-one percent of sets had conceptually based differences, while 78% had deficiencies that

appeared unintentional. Thus, in the case of major depressive episode, eight items are in common, three are not. There are also algorithmic differences, and DSM-IV has a bereavement exclusion. In this context, there are proposals to include a formal category of mixed anxiety/depression in DSM-V. This category already exists in the appendix of DSM-IV, and is an acknowledged (albeit undefined) disorder in ICD-10.

There have been intense arguments about whether categories should actually be replaced by dimensions (bipolar or unipolar). As we have seen, dimensional considerations clearly apply not only to affective disorders, but also to personality disturbance, and even to psychotic disorder. Combinations of categories and dimensions are in any case feasible. In fact, some dimensions are expressed in the form of categories: for example, ICD-10 has three severity categories of depressive disorder, wherein the dimensional element is captured. Categories are probably more practicable for clinical purposes, but not necessarily for scientific ones.

Case Identification in Research

The basis of epidemiology is case identification. The process of diagnosis involves allocating symptom patterns to a diagnostic class according to given rules. In recent years, these rules have been set out explicitly in diagnostic criteria for research (DCRs) serving the dominant classifications of ICD and DSM. These are so precise that they can be incorporated into computer algorithms like CATEGO (Wing et al., 1990) and OPCRIT (McGuffin, Farmer, & Harvey, 1991).

Once the presence of symptoms has been established, the information can be entered into one of these computer programs in order to provide a diagnostic classification. Human idiosyncrasy is reduced to an absolute minimum in this process. However, researchers must still decide how carefully the underlying symptoms should be identified. The choices include unstructured clinical assessment, responses to questionnaires, and semi-structured research interviews.

The first option, unstructured clinical judgment, introduces variability into the process of case allocation, since researchers are relying for consistency merely on their devotion to a common educational tradition. This situation is made worse when the judgments of an unspecified number of others (e.g., the treating physician) are used, as with the diagnostic information recorded in case registers or in national statistics.

In order to be practicable, questionnaires should seek simple responses to unelaborated questions. However, symptoms are traditionally recognized through an assessment of mental experiences, the subtlety of whose formulation demands quite elaborate inquiry (Brugha, Bebbington, & Jenkins, 1999). They are usually established by a process of clinical cross-examination. This is rather complicated, since it requires the questioner to frame further questions in a flexible way in the light of the answers given by the subject. While it might be possible to encapsulate this procedure in a standard questionnaire by using a branching algorithm, it would be exhaustive and exhausting—it might require paths comprising over a dozen questions just to establish, say, the presence of pathologically depressed mood. In these circumstances there are clearly practical limits to the process of standardization, and it is probably better to rely on the shortcuts available from using the skills of trained clinicians. Since diagnosis

is built around symptoms defined and elicited in this manner, redefinition in terms of answers to much more limited questions would involve changing the concept of the diagnosis itself. No one has seriously suggested that the way psychiatric symptoms are conceptualized should be changed, so if a questionnaire is used, phenomena may be recorded as present or absent when subsequent clinical inquiry might reveal otherwise. Nevertheless, structured questionnaires do allow lay interviewers to be used, with considerable cost savings. The Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981) and the Composite International Diagnostic Interview (CIDI; Robins et al., 1988) are examples of fully structured questionnaires that have been widely used and have good reliability.

Semi-structured research interviews are costly in clinical time, and the way in which symptoms are established makes it impossible to standardize the procedure entirely (Robins, 1995). Because of the reliance on clinical judgment and the effect this has on the choice of follow-up questions, some variability will remain. This is the price paid for greater validity, that is, the closer approximation to the clinical consensus about the nature of given symptoms. The SCAN (Wing et al., 1990) are based on a semi-structured interview, and have been quite widely used in epidemiological research studies (Ayuso-Mateos et al., 2001; Bebbington, Marsden, & Brewin, 1997; McConnell et al., 2002; McManus, Meltzer, Brugha, Bebbington, & Jenkins, 2009; Meltzer, Gill, Petticrew, & Hinds, 1995; Singleton, Bumpstead, O'Brien, Lee, & Meltzer, 2001). SCAN has good inter-rater reliability despite its semi-structured format.

Questionnaires and Interviews

If, as I have argued, there are doubts in principle about the validity of structured questionnaires, it is worth knowing how their performance compares with semi-standardized interviews. One head-to-head comparison has been made between SCAN and CIDI (Brugha, Jenkins, Taub, Meltzer, & Bebbington, 2001). This permits two separate questions: does the questionnaire provide a similar frequency of disorder to that established by the semi-structured interview? And to what extent are the same cases identified by the two instruments? Differences in frequencies would, at the very least, indicate some systematic biases separating the instruments. However, even if, for example, CIDI recognized more cases than SCAN, it could still be the case that CIDI picked up most or all of the cases identified by SCAN. This would imply that the constraints of a rigid questionnaire tended to lower the threshold of case identification, as might be the case if the rigidity, and the paucity of elaborative questions, led to over-recognition of specific symptoms. If on the other hand, in addition to over-recognition of cases, there were little overlap between the cases found by the two systems, it would indicate a more general failure of rigid questioning to establish symptoms properly.

Brugha et al. (2001) found that the coefficients of concordance for the various ICD-10 diagnoses varied between poor and fair. They calculated that using CIDI would give prevalences about 50% greater than those obtained from SCAN. The index of agreement for any depressive episode was poor (0.14). As expected, the discrepancies arose particularly from cases around the threshold for recognition.

However, we must also take into account the fact that the criteria for depressive disorder (DSM-IV) are more restrictive than those of ICD-10 major depressive episode. This should result in lower prevalence, perhaps 20% lower. There are therefore two influences on prevalence, of opposite effect, whose interaction will be responsible for a methodologically based discrepancy in prevalence. Thus, epidemiological studies reporting DSM-IV major depressive disorder often use CIDI, and this combination probably results in prevalences of depression around 20% above the output from a SCAN/ICD-10 combination. The short form of the CIDI, as used in the Finnish study (Lindeman et al., 2000), may result in particularly high prevalence (Patten, 1997, 2000).

The good news is that as most cases in dispute will lie around the threshold, their attributes are likely to be similar and hence the demographic and social characteristics of the disorder in question are likely to be identified with a fair degree of consistency and accuracy.

Bottom-Up and Top-Down Case Identification

The other way in which instruments differ is whether they are diagnosis driven or symptom driven. Instruments that are diagnosis driven do not require eliciting the same set of symptoms in each case in order to establish the appropriate diagnostic category. All they have to do is to confirm that the required diagnostic criteria are met. The DIS and CIDI are examples of such instruments. The advantage is that they can cut corners by not having to check out all symptoms once a diagnosis has been made: this is often the way clinicians work in their ordinary practice.

Symptom-driven instruments however are exhaustive in their coverage of symptoms, and only then do they use the symptomatic information to check if diagnostic criteria have been met (e.g., SCAN, CIS-R). This has several advantages. The first is that in theory it should be possible to use the symptom information to serve a new algorithm if the diagnostic criteria were changed. This might be extremely arduous in practice, although attempts of this sort have been made. A further advantage is of particular relevance to the study of the common affective disorders. Establishing whether or not a set range of symptoms is present allows an overall symptom count to be made, and this is useful when it is appropriate to study the distributions of symptoms in the general population, as in the study by Melzer et al. (2002) mentioned above. In principle, it could also be used to identify more severe disorder, without invoking extrinsic attributes like social performance. Finally, the establishment of individual symptoms in epidemiological samples allows them to be studied in their own right as reflections of psychological processes.

The Frequency of Depressive Disorder

In their seminal report on the Global Burden of Disease, Murray and Lopez (1996) projected that by 2020 depression would rank as the second leading cause of disability worldwide. This prediction however relies on the assumption that reasonably accurate

statistics are available, and that they can be integrated across jurisdictions. At the time the estimates were made, these requirements had been met only in the most tenuous way. While things have definitely improved, particularly in the past 10 years, it does remain difficult to calculate the burden of depressive illness in different countries. Differences in the frequency of depression between countries may in part be substantive, but will inevitably be clouded by measurement issues. These include local constraints on the conceptualization and acknowledgement of depression, and variation in the performance of instruments in local hands. Effective quality control will reduce, but not eliminate, such methodological “noise.”

In epidemiological studies, frequency can be measured in a variety of ways: incidence; point, period, and lifetime prevalence; and morbid risk. Box 1.2 defines commonly used rates in epidemiology. General population surveys usually report period or lifetime prevalence rates, while investigations of clinical series often use first contact or admission as a proxy for incidence. In this chapter, I shall rely largely on community studies of prevalence, as the characteristics of clinical series are distorted by *nosocomial* factors, such as the determinants of, and barriers to, referral to services.

The earliest community psychiatric surveys date back a century, but standardized methods of assessment allowing the comparison of research from different locations have been used only in the past 30 years. The earlier studies have been reviewed elsewhere (Bebbington, 1997, 2004; Weissman et al., 1996). The range of values for prevalence was appreciable, and somewhat greater than in more recent surveys. Moreover, the detailed results did not lend themselves to simple explanation, and there were considerable differences in the information gathered, and in the way it was gathered and combined.

Community psychiatric surveys based on standardized instruments were initially carried out in small areas, as this was relatively easy to organize, even when there were large numbers of subjects. The Epidemiologic Catchment Area surveys used the DIS to interview nearly 20,000 subjects, but were restricted to five localities in the United States (Robins & Regier, 1991). The overall lifetime prevalence of major depression was 4.9%, ranging from 3% to 5.9% in the different centers.

However, it is quite difficult to make sense of differences in prevalence in different locations in psychiatric community surveys, unless the geographical coverage is large.

Box 1.2 Epidemiological rates

Incidence rate: the number of new cases in a given period as a proportion of a population at risk.

Point prevalence rate: the number of cases identified at a point in time as a proportion of a total population.

Period prevalence rate: the number of cases identified as in existence during a specified period as a proportion of a total population.

Lifetime prevalence rate: a variant of period prevalence where the period for case identification comprises the entire lifetime of each subject at the point of ascertainment.

This is one argument for having surveys of representative national populations. The first of these occurred in Britain (Jenkins et al., 1997), and there have now been three British National Surveys of Psychiatry Morbidity (Jenkins et al., 1997; McManus et al., 2009; Singleton et al., 2001), and two in Australia (Andrews, Hall, Teesson, & Henderson, 1999; Henderson, Andrews, & Hall, 2000; Slade, Johnston, Oakley Browne, Andrews, & Whiteford, 2009). They each involved interviews carried out by nonclinical interviewers with several thousand subjects selected at random from the whole national population. The British surveys were all based on the revised version of the Clinical Interview Schedule (CIS-R; Lewis, Pelosi, Araya, & Dunn, 1992), an interview that provides ICD-10 diagnoses (WHO, 1992a), while the Australian surveys used variants of CIDI (Robins et al., 1988). CIDI allows both DSM-IV and ICD-10 diagnoses, and the Australian series reported the latter.

The last decade has seen an ambitious attempt to improve the validity of international comparison. The WHO World Mental Health Initiative sought to minimize methodological noise by adopting a common instrument and common methods. The initiative is enormous: at the last count, surveys were being carried out in 28 countries. Some of these surveys were truly national, while others were of specific regions within a given country. The expectation is that over 154,000 people will eventually have been interviewed. The six European national ESEMeD samples are included in the World Mental Health Survey reports (Alonso et al., 2002).

The sheer number of the constituent surveys leads to inherent problems of interpretation. Frequencies may vary because of differential success in engaging participants, different population structures, and the lexical consequences of differences in the transcultural interpretation of emotion (Bebbington & Cooper, 2007). International variations in the ecological context of the population may be important: economic performance, the level of inequality, the degree of urbanization, and the extent of democratic freedoms. Some jurisdictions will be subject, intermittently or persistently, to the effects of war. These influences, identifiable at the national level, will have idiosyncratic impacts on individuals. Ideally, the analysis of international differences in social and other environmental influences should inform our understanding of the nature of psychiatric disorder, but complex results will often elude easy interpretation. However, if associations are observed consistently across different jurisdictions, this does add strength to any conclusions we might draw.

Bromet et al. (2011) provide a relevant example of an attempt to derive synthetic conclusions from the mass of data that has emerged from the World Mental Health Survey Initiative (see Table 1.2). They integrated the findings regarding the prevalence of major depressive episode in 10 high-income and 8 low- to middle-income countries. (The authors did not include participating surveys from Nigeria and Ethiopia because they were suspicious that the low rates of depression in those countries may have resulted from particular difficulties with the interview.) While some response rates were lower than would be regarded as necessary for a representative sample (46% in France), response rates were unrelated to the reported prevalence of depressive disorder.

Lifetime prevalence was significantly greater in the high-income countries, averaging 14.6%, compared to 11.1% in the low- to middle-income countries. In contrast, the range of values seen for *12-month prevalence* was much less, and very similar in the

Table 1.2.

Twelve-month and Lifetime Prevalence Percentage of Major Depressive Episode in 18 Countries: World Mental Health Survey

	<i>12-month prevalence (%)</i>	<i>Lifetime prevalence (%)</i>	<i>12-month/ lifetime (%)</i>	<i>Age of onset (years)</i>
High-income countries				
Belgium	5.0	14.1	35.2	29.4
France	5.9	21.0	27.9	28.4
Germany	3.0	9.9	30.1	27.6
Israel	6.1	10.2	59.6	25.5
Italy	3.0	9.9	30.2	27.7
Japan	2.2	6.6	33.3	30.1
Netherlands	4.9	17.9	27.3	27.2
New Zealand	6.6	17.8	37.0	24.2
Spain	4.0	10.6	37.5	30.0
US	8.3	19.2	43.1	22.7
Average	5.5	14.6	37.7	25.7
Low- to middle-income countries				
Brazil	10.4	18.4	56.7	24.3
Colombia	6.2	13.3	46.7	23.5
India	4.5	9.0	50.0	31.9
Lebanon	5.5	10.9	50.0	23.8
Mexico	4.0	8.0	50.0	23.5
China	3.8	6.5	58.0	18.8
South Africa	4.9	9.8	49.6	22.3
Ukraine	8.4	14.6	57.8	27.8
Average	5.9	11.1	53.3	24.0

Source: Data tabulated by the author from Bromet et al. (2011).

high-income and low- to middle-income countries (5.5% cf. 5.9%, respectively). The ratio of 12-month to lifetime prevalence can be taken as an indication of persistence, and therefore suggests less persistence in high-income countries. However, this result could also be due to reduced recall in low- and middle-income countries. The findings are probably not due to differential international usage of the standardized instruments, and are therefore likely to be substantive. However, interpretation in relation to local contexts is difficult.

The age of onset was really quite similar in high- and in low- to middle-income countries. However, there was an interesting and probably substantive finding in relation to age. Generally, people over 65 had the lowest rates of major depressive episode in high-income countries, but not in low- to middle-income countries, where the rates are uniform across the age groups. Kessler, Birnbaum, et al. (2010b) have shown convincingly that the decline in major depressive disorder with age cannot be due to the misattribution of affective symptoms to concomitant physical disorder. However, differential survivor bias is likely to effect comparisons between high-income countries and low- to middle-income countries. The relationship of age with prevalence is discussed further below.

Women had uniformly higher prevalences of major depressive episode, a difference that was significant in the large majority of countries. The mean ratio was around two (range 1.6 to 2.7). This finding is consistent with earlier surveys, and a detailed interpretation was included in the first edition of this chapter (Bebbington, 2004). Single, divorced, and widowed people had increased rates of major depressive episode, although variably so, probably dependent on the local status of people in these categories. The frequency of depression in single people was only increased in high-income countries.

The effect of educational level was very variable, and probably reflects the different social context in individual countries. Personal income seemed more important in high-income countries than in low- to middle-income countries, possibly the effect of a wider spread of income inequality.

The level of impairment associated with depression was substantial everywhere, but was particularly high in high-income countries (where impairment may have more apparent impact). The impairment reported by those with major depressive episodes was greater in countries with higher prevalence, suggesting that high prevalence does not merely reflect a relative readiness to acknowledge affective symptoms. Finally, impairment was greater in more recent onset conditions, implying progressive adaptation to persistent disorder.

These results from the World Mental Health Initiative clearly emphasize the serious and universal nature of the problem of depression.

Depression and Age

There are clear general statements that can be made about the relationship between age and depression. First, the propensity for depression is rare before adolescence (Birmaher et al., 1996). Secondly, as we have seen from the World Mental Health (WMH) surveys described above, in developed countries the prevalence of depression declines in late middle age or early old age. This is also clearly apparent in the data on depressive episode from the three British national surveys of psychiatric morbidity (see Figure 1.2).

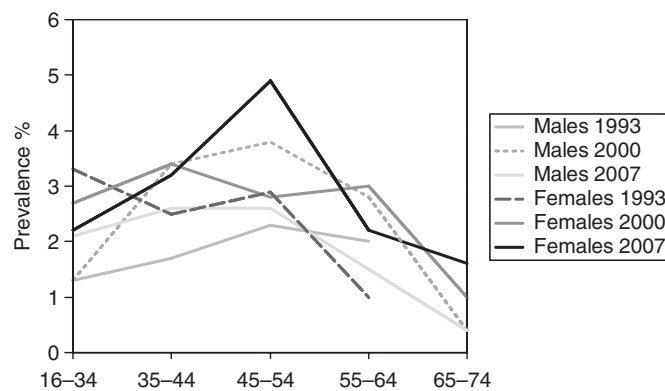


Figure 1.2. Prevalence Percentage by Age of Major Depressive Episode in British National Surveys of Residents in England

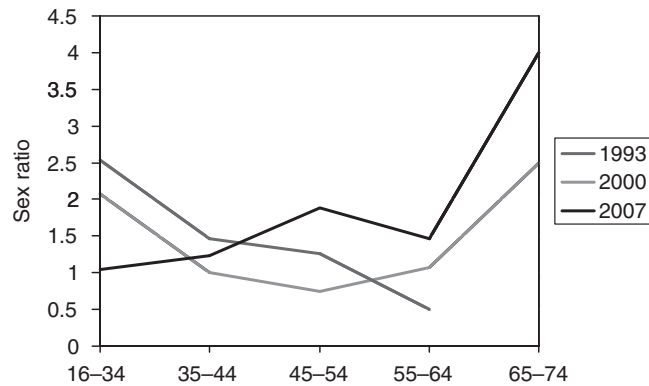


Figure 1.3. Sex Ratio of Prevalence Percentage by Age of Major Depressive Episode in British National Surveys of Residents in England

It has been said that the female-to-male sex ratio for depression is not constant over the life span, being around unity in childhood, rising during adulthood, and declining once again in elderly groups (Jorm, 1987). This reduction of the sex ratio in late middle age has been attributed to a reduction in female rates following the menopause (Bebbington et al., 1998). However, the change in the sex ratio with age shown in Figure 1.3 indicates a considerable degree of inconsistency, such that an impact of the menopause cannot be discerned.

The analysis of depression by age in different surveys makes explicit the temporal element in studies of prevalence, and therefore leads on to an issue that has been engaging epidemiologists since the 1970s. This is whether the prevalence of depression has been increasing (Compton, Conway, Stinson, & Grant, 2006; Klerman, 1988; Marcus & Olfson, 2010; Murphy, 1986).

Is the Prevalence of Depression Increasing?

Three separate mechanisms may influence the apparent variation over time of rates of depression: age, period, and cohort effects. (A cohort is a group of people with birth dates occurring within a specified period.) People may be exposed to risk of disorder because they are passing through an *age of risk* (an age effect). They may also be exposed to disorder because they are passing through a *time of risk* (for instance, a period of economic turmoil). Different cohorts will then suffer the disorder at different ages, corresponding to their age at that time (a period effect). Finally, individual cohorts may have a differing overall propensity to develop the disorder. This arises because of a conflation of date and age, and the cohort's relative vulnerability or resilience results from some shared biological or social experience. In consequence, their contribution to total prevalence will then be independent of age and period, and there is a true cohort effect. The effect on prevalence is like the surge of water down a river after a storm, an effect on overall flow removed only by arrival at the sea. It is

however extremely difficult to distinguish statistically between age, period, and cohort effects, and, as a result, changes in the prevalence of disorders are hard to interpret.

One of the strands of evidence used to argue for increasing rates of depression involves the use of cross-sectional data relating to lifetime prevalence and the date of onset of the first episode of depression. Survey participants can be divided by age group. It is generally found that younger age groups display a steeper curve in the rate of onset, leading to the inference that rates of depression are increasing (Kessler, McLaughlin, et al., 1994; Klerman & Weissman, 1989; Lewinsohn, Rohde, Seeley, & Fischer, 1993). However, it is possible that the age group effect is not one of increasing depression, but one of greater forgetfulness in older groups (Giuffra & Risch, 1994; Hasin & Link, 1988). Recall bias of this sort is supported by a 40-year study of a Canadian community (Murphy, Laird, Monson, Sobol, & Leighton, 2000).

Another way of deriving evidence for changing rates of depression is to compare national surveys repeated after an interval, using the same or similar methods of assessment. A number of these are listed in Table 1.3. It will be seen that there is appreciable variation in the recorded prevalence of major depressive disorder and depressive episode. The US National Comorbidity Surveys (Kessler et al., 2003; Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993) actually show a decline, although this may be due to the fact that the earlier sample only extended to age 54, together with other methodological changes, particularly the shift from DSM-III-R to DSM-IV criteria. However, there was also a decline in the Australian national surveys, in which case finding was more consistent. This contrasts with the sizable increase seen in the two US National Longitudinal Alcohol Epidemiologic Surveys, which used identical case findings. The three British national surveys are characterized by lower prevalences of depressive episode, but there is little variation over time. Taken together, these surveys do not provide evidence for increasing rates of depression.

Recently, the technique of pseudo-cohort analysis² has been used with the British National Surveys of Psychiatric Morbidity (Spiers et al., 2012). This involves the identification in each survey of groups of people defined by the same range of birth dates. This was done in order to investigate the age effect rather than the possibility of increasing rates, in other words whether the apparent age effect was best explained through a lasting association of depression with middle adult life, or by the effect of higher rates in certain cohorts.

Successive cohorts of men born since 1950 had experienced a similar prevalence of depression as they aged through adulthood. However, men born between 1950 and 1956 had a higher prevalence than those born between 1943 and 1949. The results for women were less consistent, with some significant increases and decreases in depression between pairs of earlier cohorts, but with stability or a decline in rates in those born since 1963.

Women born between 1957 and 1963 had a very high prevalence of depression when surveyed aged 44–50 years in 2007. This single-age sex group had unremarkable levels of depression when sampled in 1993 and 2000, so the high prevalence in 2007 was clearly unusual. This increase in a single group seems very unlikely to be due to improved recognition or a lower diagnostic threshold. While a change in

² Pseudo-cohort because given cohorts are made up of different individuals in successive surveys.

Table 1.3.
Period Prevalence of Depression in Repeated Surveys

Survey	Number of participants	Prevalence (%)			Notes
		All	Male	Female	
National Comorbidity Survey (Kessler et al., 1993)	8,098				Age 15–54. 1-year prevalence. University of Michigan version of CIDI DSM-III-R major depressive disorder
National Comorbidity Survey Replication (Kessler et al., 2003)	9,282	6.8	4.9	8.6	1-year prevalence DSM-IV depressive disorder. WMH-CIDI
First Australian National Survey (Andrews et al., 1999)	10,600	5.1	3.4	6.8	1-year prevalence ICD-10 depressive episode. Automated presentation of CIDI
Second Australian National Survey (Slade et al., 2009)	8,841	4.1	3.1	5.1	1-year prevalence ICD-10 depressive episode. WMH-CIDI
National Longitudinal Alcohol Epidemiologic Survey 1991–1992	42,862	3.3	2.7	3.9	As reported by Compton et al. (2006). Alcohol Use Disorder and Associated Disabilities Interview Schedule for DSM-IV (AUDADIS-IV). Major depressive episode.
National Longitudinal Alcohol Epidemiologic Survey and Related Conditions 2001–2002	43,093	7.1	4.9	9.1	
First British National Survey (Meltzer et al., 1995)	8,903	2.2	1.7	2.7	1-week prevalence – CIS-R. ICD-10 depressive disorder. Analysis presented here restricted to residents of England aged 16–64
Second British National Survey (Singleton et al., 2001)	6,175	2.8	2.6	2.9	
Third British National Survey (McManus et al., 2009)	5,425	2.2	2.2	3.0	

prevalence of this magnitude within 7 years is surprising, it is impossible to determine the extent to which it is a quirk of sampling, or represents some unique experience of this female birth cohort as they reached middle age. Overall, however, the analyses in this study are incompatible with an increasing overall trend toward depressive disorder.

It is clearly possible for rates of depression to vary with time, just as they may vary geographically. Like investments, prevalences may fall as well as rise, and fluctuation in both directions is likely to be the norm. For instance, general statements that say prevalence is continually and uniformly increasing, are almost certainly false. Sometimes the changes will be due to methodological vagaries, and in other cases due to small alterations in the risk and duration of recurrent or relapsing disease. Substantive changes over periods commensurate with human lifetimes are almost certain to be environmental, whether biological or social. Finally, differences in prevalence between studies are more likely to be the result of short-lived fluctuations picked up by chance than major trends. By the same token, they will mainly represent responses to short-lived contextual change. Paid employment is a frequent source of these fluctuations.

Employment and Depression

Structured employment is a feature of developed societies, and many of us spend a sizable portion of our waking lives at work. Employment generally has beneficial effects on psychological health: it brings interest, income, fulfillment, social contacts, and status, and provides structure and a sense of control (Jahoda, 1982; Krause & Geyer-Pestello, 1985). The strong and persistent link between unemployment and mental ill-health, particularly depressive disorder, is long-established (Meltzer et al., 1995; Talala, Huurre, Aro, Martelin, & Prattala, 2009). Thus, in longitudinal studies, losing employment is associated with a deterioration in mental health, just as reestablishing it leads to improvement (Murphy & Athanasou, 1999).

The benefits of employment are likely to differ, both between men and women, and among women. In developed economies, the advantages of employment may be weaker in married women (Roberts, Roberts, & Stevenson, 1982; Roberts & O'Keefe, 1981; Warr & Parry, 1982), more so if they have children (McGee, Williams, Kashani, & Silva, 1983; Parry, 1986), most so when the children are of preschool age (Haw, 1995). Not surprisingly, full-time employment seems to be particularly demanding (Cleary & Mechanic, 1983; Elliott & Huppert, 1991). The most likely explanation for these findings is role conflict and overload. Thus, part of the excess of depressive disorders in women may be related both to their reduced involvement in employment and to the particular strains they are exposed to if they do work.

Depression and the Characteristics of Work

The circumstances of employment are not always beneficial. Researchers have accordingly linked the specific attributes of employment to mental health outcomes. For example, in a recent longitudinal study, work with poor psychosocial characteristics (adverse levels of control, demands and complexity, job insecurity, and unfair pay) was as strongly associated with mental ill-health as unemployment (Butterworth et al., 2011). Moreover, moving from unemployment to poor quality work actually led to a decrease in mental health. The consequences of adverse work conditions appear to be relatively nonspecific, covering common mental disorders in general, but including

depressive disorders. This has been established in prospective research over the past decade (Siegrist, 2008; Stansfeld & Candy, 2006).

Two main models have been developed to evaluate this putative link. They postulate effects that operate through the experience of strains and imbalances. Although these might be mediated through biological, psychological, or behavioral pathways (Melchior, Berkman, Niedhammer, Zins, & Goldberg, 2007), both models can be seen as primarily cognitive. Under the Job Demand–Control model (JDC; Karasek, 1979), job strain ensues when high job demands are combined with low job control. The model has been augmented by incorporating the beneficial effect of the social support obtainable through work (thus the Job Demand–Control–Support model, or JDSC; Johnson & Hall, 1988). The second model is the Effort–Reward Imbalance model (ERI; Siegrist, 2008). This asserts that adverse health is the consequence of circumstances in which the efforts of the work are high and the rewards are low, with a resulting sense of inequity. This in turn leads to poor health. The situation is exacerbated if the individual becomes overcommitted in the process of dealing with the situation (Siegrist, 1996).

Both models have been used to guide research, and both have been substantiated, although they have rarely been tested head to head. Thus, the adverse features postulated as important in each model have been linked to anxiety, and mood disorders (de Lange, Taris, Kompier, Houtman, & Bongers, 2003; Netterstrom et al., 2008; Siegrist, 2008; Stansfeld & Candy, 2006). Clark et al. (2012) analyzed data from a large population survey in England (the Adult Psychiatric Morbidity Survey of 2007): their results suggested that the characteristics of work associated with depression and other common mental disorders were actually broader, and revealed more complex interactions, than would have been predicted from the models. Disorder was, as expected, associated with high-demand/low-control jobs and low social support at work (JDSC) and with ERI. However, it was also associated with high-demand/high-control and low-reward/low-effort jobs. Interestingly, the impact of the work environment seemed insulated from the rest of people's lives: nonwork stressors did not appear to increase susceptibility to work-related stressors. The authors also found no support for a hypothesized moderating effect of nonwork social support, debt, and overcommitment on the impact of work stressors on common mental disorders.

Job Security and Depression

Another attribute of employment is the degree of job security. Many industrialized economies have increasingly adopted policies of labor flexibility, the obverse of which is increased job insecurity. A considerable number of studies have now investigated the relationship between job insecurity and mental health. A meta-analysis of 37 surveys in the years to 1999, covering around 15,000 respondents confirmed that job insecurity was strongly associated with poor mental health (Sverke, Hellgren, & Näswall, 2002). Stansfeld and Candy (2006), in a meta-analysis of longitudinal studies from 1994 to 2005, found that job insecurity was one of several occupational stressors predictive of common mental disorders. Bonde (2008) reviewed 16 studies covering 63,000 employees, and concluded that the perception of adverse psychosocial conditions in

the work place was linked to an increased risk of depressive symptoms and major depressive episode. The fear of job loss itself seems to be more important than the loss of specific features of the job (Hellgren, Sverke, & Isaksson, 1999).

Job insecurity is likely to have differing effects on different sorts of employee. Simmons and Swanberg (2009) showed that, for poor employees in the United States, job insecurity was the single, significant correlate of depressive symptoms even after controlling for other demographic and work environment characteristics. In contrast, in more affluent employees, depressive symptoms were associated with high psychological demands and low supervisor and coworker support.

When the current economic situation of an organization is perceived as poor, job insecurity is high (Mauno & Kinnunen, 2002). Future reorganization plans contribute to the rise in job insecurity (Ashford, Lee, & Bobko, 1989), as do nonpermanent job contracts (Mauno & Kinnunen, 2002). The stress of uncertainty may be more a cause of anxiety and depression than coming to terms with redundancy.

Financial Strain and Depression

Unemployment, underemployment, and poorly rewarded employment create financial strain, and easily lead people into debt, with direct mental health consequences (Theodossiou, 1998). Problems with financial indebtedness and the impact that financial stress has on family well-being have been well documented in the media (Bridges & Disney, 2010). Moreover, at the time of writing, many developed economies are witnessing increasing rates of unemployment, cuts in pensions and benefits, and price inflation. These will inevitably contribute to the financial stress faced by individuals and families, and there is already evidence at the population level of increasing rates of depression (Lee et al., 2010; Madianos, Economou, Alexiou, & Stefanis, 2011).

Research at the individual level on the relationship between debt and mental disorder has been limited by the difficulties of measuring debt in large epidemiological surveys. It has rarely been possible to include questions about specific amounts of money relating to individual items of expenditure (housing costs, heating costs, weekly shopping bills). The topic is sensitive, and the information is time-consuming to collect, increasing both cost and respondent burden. In many cases, respondents are merely asked whether they can make ends meet or details of their budgeting strategies. These subjective assessments are then used as proxies for actual indebtedness and for underlying household budgetary problems. Moreover, many studies have relied on limited questionnaire measures, and few have used structured interviews and standardized diagnostic criteria (Hintikka et al., 1998; Roberts, Golding, Towell, & Weinreb, 1999). Hence very few population-based epidemiological studies have been able to conduct persuasive studies of debt and mental disorder (Eaton, Muntaner, Bovasso, & Smith, 2001; Muntaner, Eaton, Miech, & O'Campo, 2004).

Recent studies have used improved methods and report interesting findings. So, financial stress may be central to the association between lower socioeconomic status and depression (Butterworth, Olesen, & Leach, 2012). Jenkins et al. (2008) found that, although people with low income were more likely to have mental disorder, this relationship was attenuated after adjustment for debt. Conversely, even after

adjustment for confounders, people in debt remained more than twice as likely to have depressive disorder than those who were not (Meltzer, Bebbington, Brugha, Farrell, & Jenkins, 2012). Although the source of debt had little effect, the *number* of debts were associated with an increasing likelihood of depressive disorder.

It should be acknowledged that the problem of causal direction is particularly acute in this field. Thus people may get into debt for a variety of reasons (gambling, substance abuse, compulsive shopping, marital or relationship breakdown, and redundancy), and these factors, alone or in combination, then increase the risk of anxiety and depression. Individual behavioral responses come in to play. When faced with a worsening financial situation, some people will cope by looking for opportunities to reduce its impact, while others may fall (or fall further) into debt. Moreover, people with mental disorders are less likely than others to obtain or maintain employment, and may also find it difficult to deal with indebtedness by budgeting effectively and applying for benefits. Their debt may also be exacerbated by a failure to appreciate its degree. Both these mechanisms probably apply—people with debts are more likely to have mental health problems and people with mental health problems are more likely to be in debt (Fitch et al., 2009).

Clark et al. (2012) found in a national sample of employed people that job insecurity and debt were independent correlates of depression. Job insecurity, being in debt, and working at the bottom of the occupation hierarchy were all independently associated with depression.

As with most life stresses, the effect of job strain and financial strain remains very nonspecific. Thus, debtors have high rates of all common mental disorders, including depressive episode (Meltzer et al., 2012).

The Childhood Antecedents of Later Depression

There is a long history of research into the childhood antecedents of depressive disorder. Early studies looked at the effects of separation from, and loss of, parents (Brown & Harris, 1978; Tennant, Bebbington, & Hurry, 1980; Tennant, Hurry, & Bebbington, 1980). More recently, the focus has moved to specific unpleasant events and circumstances, such as bullying, witnessing marital violence, and exposure to sexual and physical abuse.

Childhood adversity may take the form of a repetition of minor upsets and constraints, or of major dramatic events, also sometimes repeated. The traumatic nature of some of these experiences is immediately obvious, and long-term effects are exceedingly plausible. However, damaging effects following the extended experience of parental child-rearing behavior are less self-evident. There has therefore been some debate about whether parenting style in itself is sufficient to account for much of the variance in adult depression. One school of thought is that children have a built-in plasticity in the face of quite considerable disparities in levels of care.

However, it is now established that parenting style does have an appreciable impact on later mental health. The Parental Bonding Instrument (PBI; Parker, 1990; Parker, Wilhelm, & Asghari, 1979) is a self-report inventory designed to measure perceived parental care. It divides parenting style into the aspects of *care* and *overprotection*.

Optimal parenting is reflected in high scores on care and low scores on overprotection (Parker, 1990). While adult depression is consistently (if not very strongly) related to lack of care, the association with overprotection is less consistent (Enns, Cox, & Clara, 2002; Parker, Hadzi-Pavlovic, Greenwald, & Weissman, 1995). These relationships have been found in a number of countries (Heider et al., 2006).

Clearly, the PBI is a self-report measure, and might merely represent a querulous response set in people whose mood is depressed. In fact there is little evidence of this. Parker (1981) demonstrated the validity of the PBI by examining the correspondence between sibling ratings of the subject's parenting with the subject's own. Moreover these ratings appear reasonably stable over a 20-year follow-up period (Wilhelm, Niven, Parker, & Hadzi-Pavlovic, 2005). It is quite possible that the apparent relationship between parenting and depression might be revealed as spurious by a third variable (neuroticism, for instance). It is equally possible that neuroticism mediates between the experience of poor parenting and depression (Kendler, Kessler, Neale, Heath, & Eaves, 1993). However, Duggan, Sham, Minne, Lee, and Murray (1998) found that the effects of poor parenting and neuroticism on later depression were independent of each other.

Childhood abuse, whether physical, emotional, or sexual, is associated with later psychopathology (Bifulco, Brown, & Adler, 1991; Fergusson, Horwood, & Lynskey, 1996; Mullen, Martin, Anderson, Romans, & Herbison, 1996). Adult depressive episodes are associated with a variety of childhood traumas (De Marco, 2000). One of these is bullying, in which there has been a persistent media interest. It can be defined as vicious, aggressive behavior directed toward people who cannot defend themselves effectively. While bullying used to be thought of as a school-related experience, it is now acknowledged to be prevalent in other settings, including the electronic media (Smith & Monks, 2008). It is a phenomenon in which victim characteristics are fairly consistent. These include certain personality traits—being shy, silent, fearful, anxious, physically weak, insecure, crying easily, having low self-esteem (Egan & Perry, 1998; Kumpulainen et al., 1998; Olweus, 1994) and being different from other children in some way—stuttering (Blood & Blood, 2007), being overweight (Robinson, 2006), or having learning difficulties (Reiter & Lapido-Lefler, 2007).

The persistent effects of bullying are abundantly clear. Sourander et al. (2009) reported that boys who had been bullied at the age of 8 were, 15 years later, considerably more likely than their nonbullied peers to suffer a variety of mental health consequences. These included depressive and anxiety disorders. This finding was confirmed by Kumpulainen (2008), who reported bullying was such a continuously distressing experience that it predicts both concurrent and future psychiatric symptoms and disorders. Being bullied in childhood and adolescence has sufficient effect on mood to increase appreciably the risk of suicidal ideation and behavior (Kim & Leventhal, 2008).

The mental health consequences of child sexual abuse have recently been subject to considerable scrutiny, in reaction to a long period in which reports of its occurrence were often discounted. It is now well established that sexual abuse in childhood (CSA) is common (Bebbington et al., 2011, Dinwiddie et al., 2000; Friedman et al., 2002; May-Chahal & Cawson, 2005; Pereda, Guilera, Forns, & Gomez-Benito, 2009). Rates of CSA do not differ in relation to most sociodemographic attributes, although

it is commoner in women and in those who have not been brought up by both biological parents until the age of 16 (Bebbington et al., 2011). It may also be related to suboptimal parenting style. Low care appears to be associated with sexual abuse, not only by relatives, but also by nonrelatives (Hill et al., 2001).

There is consistent evidence of deleterious psychiatric sequelae in adulthood, and there are certainly enhanced risks of depression (Weiss, Longhurst, & Mazure, 1999). However, the mental health consequences are relatively nonspecific, as they include a whole range of other disorders (Bebbington et al., 2004, 2009; Coxell, King, Mezey, & Gordon, 1999; Dinwiddie et al., 2000; Janssen et al., 2004; Jonas et al., 2011; Kendler et al., 2000; King, Coxell, & Mezey, 2002; Nelson et al., 2006; Putnam, 2003; Read, van Os, Morrison, & Ross, 2005). The effects seem to be proportionate to the severity and persistence of the abuse (Anda et al., 2006; Bulik, Prescott, & Kendler, 2001; Kendler et al., 2000; Kendler, Kuhn, & Prescott, 2004; Molnar, Buka, & Kessler, 2001; Mullen, Martin, Anderson, Romans, & Herbison, 1993), and *may* be greater in women (MacMillan et al., ?; Molnar et al., 2001; Weiss et al., 1999; but see below).

Jonas et al. (2011) report data from the English Adult Psychiatric Morbidity Survey of 2007, in which detailed information on sexual abuse was elicited. Sexual abuse in childhood (before the age of 16) was strongly associated with a wide range of psychiatric disorders. However, their published analyses relate to the amalgamated category of *common mental disorders* (a mixed bag of six anxiety and depressive disorders). I therefore provide the equivalent results for major depressive episode, in relation to nonconsensual sexual intercourse, to all forms of abuse involving contact, and finally to all forms of abuse including uncomfortable sexual talk (see Table 1.4). The odds ratios are roughly equal in males and females, and tend to be higher for the more severe forms of abuse. However, because severe forms of abuse are relatively rare, the population attributable fraction (PAF) is greater for the category covering the whole range of abuse. PAFs provide an upper bound limit of the amount of disorder that can be attributed to the given factor. They do involve an assumption of causality and the presumption that the results are not confounded. Both assumptions are unlikely to be

Table 1.4.
Sexual Abuse and Depressive Episode

<i>Abuse type</i>	<i>Statistics</i>	<i>Males</i>	<i>Females</i>
Nonconsensual sexual intercourse	Odds ratio (95% CI)	4.1 (0.9–17.6)	4.4 (2.4–7.8)
	Adjusted odds ratio	2.4	4.3
	Population attributable fraction	2.1%	7.8%
Contact abuse	Odds ratio (95% CI)	3.9 (2.1–7.3)	3.0 (2.0–4.5)
	Adjusted odds ratio	4.6	2.7
	Population attributable fraction	13.2%	17.3%
All forms of abuse	Odds ratio (95% CI)	2.9 (1.6–5.3)	2.7 (1.9–3.9)
	Adjusted odds ratio	3.2	2.6
	Population attributable fraction	13.5%	20.8%

Source: Data from the Adult Psychiatric Morbidity Survey of England (2007).

more than partially true, but PAFs do give a rough idea of public health impact. In relation to all forms of sexual abuse, the PAF for depression in females was over 20%, and in males 13%. The lower value in males was largely due to the relative infrequency of their experience of abuse, rather than to reduced sensitivity.

The various forms of childhood disadvantage show an understandable but unfortunate tendency to cluster in the lives of given individuals (Bebbington et al., 2004). Green et al. (2010) and McLaughlin et al. (2010) used data from the National Comorbidity Survey Replication in the United States to provide analyses of 12 types of childhood disadvantage. Seven of these types formed a maladaptive family functioning cluster, within which were three forms of abuse: physical abuse, sexual abuse, and exposure to family violence. They examined childhood disadvantage in relation to the onset and persistence of a range of different disorders. Sizable relationships were apparent with all the disorders tested, with relatively little specificity. Links were generally stronger with onset than with persistence. One of the categories they examined was mood disorders, a catch-all in which the majority of cases were very probably of major depressive episode. Childhood adversities were associated with a quarter of cases of mood disorder. Family violence, physical abuse, and sexual abuse were significantly associated with the onset of mood disorder, and also with its persistence, albeit less strongly. Kessler, McLaughlin, et al. (2010) reported similar results from 21 high-, middle- and low-income countries in the World Mental Health Initiative. The impact of childhood adversity was apparent in each of these national groupings, and overall it was associated with onset in 23% of cases of mood disorders, very similar indeed to the US studies described above.

It is of interest to speculate how childhood traumas have their effects on the emergence of depression in adulthood. It is generally not because they cause childhood depressive disorder, as this is a rare condition. The links must therefore usually be indirect—the causal connection appears to operate over a gap of years. This suggests some enduring change that mediates the later propensity to depression. Candidates include mentally intrusive reminders of the abusive experience, psychological processes involving attitudes and beliefs, propensities toward mood disturbance in the face of subsequent experience, and styles of coping that may impair the processing of the original abuse. CSA certainly has extreme adverse effects on self-esteem, self-blame, and psychological well-being (Banyard, Williams, & Siegel, 2001; Kamsner & McCabe, 2000; Mannarino & Cohen, 1996; Murthi & Espelage, 2005). People who have been sexually abused often display avoidant coping, which is also seen in the various psychiatric disorders that have been linked to abuse (Cortes & Justicia, 2008; O'Leary, 2009). Abuse may also modulate the physiological stress response in deleterious ways (Driessen et al., 2000; Heim, Newport, Miller, & Nemeroff, 2000; Read et al., 2005; Spauwen, Krabbendam, Lieb, Wittchen, & van Os, 2006). Finally, it may create a vulnerability to later damaging exploitation. Thus, CSA seems to be followed by a significant increase in the risk of adult sexual abuse: in one study, 50% of those who had experienced abuse under 16 also reported an episode over the age of 16 (Jonas et al., 2011).

We must also ask ourselves why so many different disorders are associated with the same putative etiological agent? The explanation may merely reflect the nonexclusive nature of psychiatric classification, which results in the frequent comorbidity seen in

practice. If not, the specific psychiatric consequences may arise from the particular context and attributes of the abuse, or a tendency in the individual, inhering from other causes, of responding in particular ways.

Conclusions

In this chapter, I have considered the practical difficulties facing the epidemiological study of depression. Epidemiology is a medical approach that relies initially on the conceptualization of impaired functions as disorders, followed by a requirement to identify these disorders in a reliable way. So conceived, depression shades both into normal experience and into other affective disorders. Distinguishing it in a way at once useful and consistent is thus difficult, as I have argued in some detail. In particular, the comparability of studies is jeopardized by differences between classifications and instruments and in the way these are applied. The consequence is that no two research teams are likely to identify the same sets of respondents as cases; indeed, the overlap is in practice small and there may be systematic over- or under-identification, resulting in different prevalence rates. To a degree, these obstacles to precise case identification are probably insuperable, although the arguments remain strong for doing the best we can.

Two things alleviate this rather miserable conclusion. Because in general populations most cases identified are around the threshold that distinguishes them from noncases, different studies are likely to end up with case groups that have similar characteristics. Robust associations, for example, the association of depression with life events or with poverty, will therefore survive the inadequacies of our instrumentation. The second way around these inadequacies is to supplement the medical case approach with studies that look at the correlates of total symptom score. In this way, important findings can be triangulated, as they are in the study of blood pressure.

Finally, epidemiology provides more interpretable results when it is theory driven, with firm a priori hypotheses about how results might be mediated by social, psychological, and biological factors.

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