Chapter 2

What is comorbidity and why does it occur?

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Introduction

Comorbidity can be defined most generally as the co-occurrence of two or more mental health problems. It has emerged as a major clinical, public health and research issue over the past few decades. This is due in part to changes in psychiatric nomenclature, in which there is a greater focus upon elucidating any number of mental health problems with which an individual might present, rather than diagnosing one problem to the exclusion of others.

Currently, mental health problems are conceptualised as patterns of behaviour or thought that are associated with significant disability, distress, loss of individual freedom, or adverse events such as death; and which arise from dysfunction within the individual (Neugebauer, 1999). These problems can encompass a wide range of behaviours including substance use, mood disturbances, anxiety, and disturbances in thought and perception.

According to current classification systems in psychology and psychiatry, mental disorders are diagnosed according to operationalised diagnostic criteria, and the diagnosis of one disorder does not necessarily preclude the diagnosis of another (American Psychiatric Association, 1994; World Health Organisation, 1993). In some cases, more than one mental disorder is diagnosed — such comorbidity is examined in this chapter. Specifically, this chapter will define the concept of comorbidity; discuss the implications of comorbidity for theories of mental health, treatment and prevention; give a brief overview of epidemiological research into comorbidity; and examine the reasons why comorbidity might occur.

Definitions

‘Comorbidity’ was defined by Feinstein (1970) as “any distinct clinical entity that has co-existed or that may occur during the clinical course of a patient who has the index disease under study” (pp. 456–7). Within psychiatry, comorbidity is commonly used to refer to the overlap of two or more psychiatric disorders (Boyd, Burke, Gruenberg, et al., 1984). Comorbidity between substance use disorders and other mental disorders has gained increasing prominence in psychiatry and psychology within the past few decades (Wittchen, 1996). Angold and colleagues have recently drawn a distinction between two types of comorbidity (Angold, Costello, & Erkanli, 1999). Homotypic comorbidity refers to the co-occurrence of mental disorders within a diagnostic grouping (Angold et al., 1999). The co-occurrence of two different substance use disorders (e.g. cannabis and alcohol) is an example of homotypic comorbidity. Heterotypic comorbidity refers to the co-occurrence of two disorders from different diagnostic groupings (Angold et al., 1999). This might include, for example, the co-occurrence of a substance use disorder and an anxiety disorder.
Why study comorbidity?

Comorbidity potentially has implications for theories of aetiology, prevention and treatment of mental health problems.

Importance for theory

If mental health problems are more likely to occur among those with substance use disorders, this raises important questions about the aetiology of mental disorders (and vice versa). Several hypotheses exist concerning the reasons why comorbidity might occur, including that: (a) there is a causal relationship between the two; (b) that common factors increase the likelihood of both disorders; and (c) that the relationship is spurious (artefactual), resulting from factors such as the methods with which the sample was selected (Caron & Rutter, 1991; Kessler, 1995; Mueser, Drake, & Wallach, 1998). Before we can begin to unravel the reasons behind any ‘comorbidity’, we need to carefully document the nature of any associations. This will give some insight into possible mechanisms underlying the association.

Importance for treatment

If people who are problematic substance users are more likely to have other mental health problems, this needs to be taken into account both in the assessment of a client, and in determining the most appropriate treatment. Comorbidity is particularly relevant if co-occurring disorders predict a differential clinical outcome, which has been suggested by previous research (e.g. Carey, Carey, & Meisler, 1991; Haywood et al., 1995; Pristach & Smith, 1990; Rouillon, 1996). Attention to comorbid problems may also improve treatment outcome. The efficacy of treatment for alcohol and nicotine dependence, for example, may be improved if treatment for depression is also provided (Hall et al., 1998; Lynskey, 1998).

Importance for prevention

Prevention programs have traditionally operated in isolation from each other. For example, it is often the case that programs addressing suicide prevention, substance use prevention and sex education/sexual risk taking occur separately. There is rarely an attempt to conduct programs aimed at addressing multiple problems in an integrated fashion. Furthermore, there is an increased interest in psychiatry on prevention. The concept of comorbidity has two broad implications for prevention: a) if ‘comorbidity’ is real, then prevention efforts should be broad in their target; and b) an understanding of the nature of comorbidity will help dictate the targets of prevention. If comorbidity arises because different problems or disorders share the same risk factors, then interventions addressing these risk factors should reduce the prevalence of these multiple problems.

The importance of general population research on comorbidity

It is critically important to study patterns of comorbidity between different mental disorders in general population samples. It is not possible to know that patterns observed in clinical samples will reflect those in the general community, because significant biases may be present (Berkson, 1946; Galbaud Du Fort, Newman, & Bland, 1993). There are a variety of reasons why comorbidity might be more common in clinical samples. It is also likely that skewed patterns of comorbidity will exist because of factors such as areas of particular interest, or expertise of clinicians.
in a given treatment centre, or alternatively, exclusionary policies of a treatment centre, or factors that may differentially influence a person’s decision to seek help.

These factors are impediments to making accurate decisions about treatment needs of the general population from clinical samples. It is also difficult to make advances in theories about comorbidity since we do not know whether comorbidity observed in clinical samples is due to sampling, or referral biases. Only by studying representative samples of the general population can we ensure that our findings reflect general patterns of co-occurrence of different mental health problems in the community.

**General studies of comorbidity**

Most epidemiological research on comorbidity is relatively recent. In order to understand the development of research into comorbidity on a general population level, it will be useful to outline a brief summary of the history of epidemiological research into comorbidity.

**The US Epidemiological Catchment Area study**

In 1978, the US President’s Commission on Mental Health decided to conduct epidemiological research to estimate the prevalence of mental disorders in the general community and the extent of health service use among persons with such disorders (President’s Commission on Mental Health, 1978; Regier & Kaelber, 1995). The project was undertaken by the US National Institute of Mental Health (NIMH), and the resulting study was the Epidemiological Catchment Area study (ECA). The ECA aimed to provide estimates of the prevalence and incidence of the following major DSM-III disorders: mood disorders, substance use disorders, anxiety disorders, and psychotic disorders.

Researchers involved in the ECA decided to develop a diagnostic interview that incorporated the newly defined DSM-III diagnostic criteria, since no such DSM-III-based interview existed at that point (Regier & Kaelber, 1995). The NIMH Diagnostic Interview Schedule (DIS) (Robins, Helzer, Croughan, & Ratcliff, 1981; Robins, Helzer, Croughan, Williams, & Spitzer, 1981) was highly structured, designed to be administered by trained lay interviewers, and would identify persons who met operationalised criteria for specific DSM-III mental disorders (Regier & Kaelber, 1995). It was validated against existing diagnostic interviews, clinicians’ diagnoses, and physicians’ diagnoses (Folstein et al., 1985; Helzer et al., 1985; Orvaschel et al., 1985).

In the ECA study, samples were taken from five ‘catchment area’ sites with a total population of at least 200,000 persons. They were chosen by the NIMH from applications from the following institutions: Yale University, Johns Hopkins University, Washington University, Duke University, and the University of California in Los Angeles, which surveyed New Haven, Baltimore, St. Louis, Durham, and Los Angeles, respectively (Robins & Regier, 1991). Both community and institutional facilities (such as prisons, nursing homes, and psychiatric facilities) were sampled.

The ECA’s response rate was 76%, with an overall sample size of 19,640 (Robins & Regier, 1991). Sample sizes of approximately 3,000 household residents and 500 institutional residents per site had been targeted to ensure that risk factors for...
schizophrenia (which affects around 1% of the population) could be studied (Regier & Kaelber, 1995; Robins & Regier, 1991). The research groups were required by the NIMH to obtain representative samples of the population in the five sites (Holzer et al., 1985). The estimates obtained were weighted to project estimates for the entire United States (Robins & Regier, 1991). Lay interviewers, all trained at Washington University to ensure comparability of interview administration, conducted the interviews (Regier & Robins, 1991). Each site conducted its own survey and data collection.

The ECA has been called a “landmark study in psychiatric epidemiology” (p. 81 Kessler, 1994a) in that: (a) it was the largest general population survey of mental disorders carried out to that date; (b) it was the first to administer a structured diagnostic interview; and (c) it was the first to estimate total population prevalence estimates, since institutionalised and non-institutionalised samples were obtained (Kessler, 1994a).

The ECA stimulated a number of epidemiological surveys in other countries, which used similar sampling methods, the same DSM-III diagnostic criteria, and the same survey instrument (the DIS). Studies were carried out in Munich, Germany (Fichter et al., 1996; Wittchen, Essau, von Zerssen, Krieg, & Zaudig, 1992); Edmonton, Canada (Bland, Newman, & Orn, 1988); Christchurch, New Zealand (Oakley-Browne, Joyce, Wells, Bushnell, & Hornblow, 1989; Wells, Bushnell, Hornblow, Joyce, & Oakley-Browne, 1989); Shanghai, China (Wang et al., 1992); Korea (Lee, 1992); and Taiwan (Hwu, Yeh, & Chang, 1989).

**The US National Comorbidity Survey**

The design of the ECA was improved upon by researchers who designed and conducted the US National Comorbidity Survey (NCS) in 1992 (Kessler, 1994a; 1994b). The NCS extended the ECA in the following ways:

1. the NCS used DSM-III-R diagnostic criteria, with some allowance for comparisons with DSM-IV when it was released, in contrast to the DSM-III criteria used in the ECA;
2. the NCS was designed not only as a study of the prevalence of mental disorders, but also as a study of the risk factors for such disorders;
3. it was a nationally representative sample of US adults, as opposed to the five catchment areas that were used in the ECA; and
4. as the title suggests, one of the primary aims of the NCS was to explore the patterns of comorbidity between different mental disorders that had been observed in the ECA.

The NCS was designed to explore the prevalence, causes and consequences of comorbidity. The age range (18 to 54 years) used in the survey was chosen because comorbidity was found to be most prevalent among this age group in the ECA (Kessler, 1994a, 1994b). The NCS was a national survey. Participants were selected from the non-institutionalised civil population in the 48 contiguous US States, with an additional sample of students from university campus housing. Institutional samples were not selected since the inclusion of such samples in the ECA had not been found to make a substantial difference to prevalence rates of mental disorders (Robins & Regier, 1991). Experienced field interviewers were used in the data collection.
collection to ensure that interviews were conducted by competent staff. A special feature of the NCS was that non-responders to initial interviews were re-targeted for interview to ensure that prevalence estimates were not affected by non-response rates. This was because research had suggested that those who refused to participate in surveys had higher rates of mental disorders (Kessler, 1994b).

The NCS had a response rate of 83%, with a final sample size of 8,098. The psychiatric diagnoses assessed were DSM-III-R diagnoses of anxiety disorders, mood disorders, substance use disorders and psychotic disorders. The diagnostic interview was the Composite International Diagnostic Interview (CIDI), which was designed for administration by trained interviewers who are not clinicians (Kessler, 1994b). It was administered by staff at the Survey Research Centre at the University of Michigan between September 1990 and February 1992 (Kessler et al., 1994).

Other epidemiological studies

Since the conduct of the ECA and NCS, a number of epidemiological studies have been carried out using DSM-III-R or DSM-IV criteria with representative samples of persons from countries such as the US (Grant & Pickering, 1998), Canada (Ross, 1995), and the Netherlands (Bijl, Ravelli, & van Zessen, 1998).

The Australian National Survey of Mental Health and Wellbeing

Australian researchers planned and conducted the Australian National Survey of Mental Health and Wellbeing (NSMHWB) in 1997. It involved a modified version of the CIDI (which is a more recent version of the DIS) and used DSM-IV criteria. The nationally representative NSMHWB sample involved the assessment of ICD-10 and DSM-IV substance use disorders, mood disorders, anxiety disorders, and it also screened for likely cases of psychosis (Hall, Teesson, Lynskey, & Degenhardt, 1999; Henderson, Andrews, & Hall, 2000). The NSMHWB was conducted to provide representative information on the mental health of Australian adults aged 18 years and over. There were three major aims of the survey (Henderson et al., 2000): (a) to estimate the prevalence of mental disorders in the general population; (b) to estimate the amount of disability associated with such disorders; and (c) to estimate the use of health and other treatment services by persons with such disorders. The NSMHWB was consistent with the findings of other general population studies in finding that mental disorders are prevalent in the general population (Andrews, Henderson, & Hall, 2001). There were notable similarities in the socio-demographic correlates of the disorders examined (substance use disorders, mood disorders, anxiety disorders and screening positively for psychosis).

The UK conducted the National Psychiatric Morbidity Survey (1997; Jenkins, Lewis et al., 1997), using an adapted DIS interview for assessing ICD-10 substance dependence. They assessed mental health problems using the Clinical Interview Schedule-Revised (CIS-R) (Farrell et al., 1998), which may be used to estimate ICD-10 mental disorders, although this has subsequently been shown to have poor agreement when compared with semi structured clinical interviews using the SCAN (Brugha et al., 1999).

These studies were consistent in that they found mental disorders to be common in the adult population, and to be associated with disability and social disadvantage. They also found that comorbidity does occur in the general population.
Explanations of comorbidity

There are several reasons why two disorders might co-occur — that is, be truly comorbid (Caron & Rutter, 1991; Kessler, 1995). These are: (1) that there is a direct causal relationship between the two, with the presence of one disorder making another more likely to develop; (2) that there is an indirect causal relationship between the two, with one disorder affecting a third variable in a way that increases the likelihood of the second disorder; and (3) that there are common factors that increase the risk of both disorders. These are discussed in more detail below.

Direct causal relationship

There is a range of causal relationships that have been used to explain specific types of comorbidity between substance use problems and other mental health problems. Mental disorders have been argued to cause substance use disorders, and vice versa.

Mental health problems cause substance use problems

A plausible hypothesis of the relationship between substance use disorders and other mental health problems is that persons with mental health problems who begin to use substances to alleviate the symptoms of their illness develop problematic use as a result of over-use (Khantzian, 1985, 1997; Pope, 1979). A central assumption of this ‘self-medication’ hypothesis is that substances are used to alleviate symptoms and that specific substances will be selected for their specific effects upon mood and cognition. For example, it has been suggested that persons who are heroin dependent use heroin to ameliorate aggression and rage, while persons who are cocaine dependent use it to alleviate symptoms of depression (Khantzian, 1985).

A variation of the self-medication hypothesis has also been used to explain the relationship between schizophrenia and substance use. One such hypothesis is that persons with schizophrenia use tobacco to reduce positive symptoms such as hallucinations and delusions (Gilbert & Gilbert, 1995), and also to reduce negative symptoms such as blunted affect, apathy and anhedonia (Gilbert & Gilbert, 1995; McEvoy & Brown, 1999).

However, the evidence that specific drugs are used to ‘treat’ specific symptoms is less than compelling (Mueser et al., 1998). For example, self-report studies of persons with schizophrenia and substance use disorder have found very little evidence that different substances are used to alleviate specific mood states or symptoms (Dixon, Haas, Weiden, Sweeney, & Frances, 1991; Noordsy et al., 1991). Furthermore, patterns of substance use among persons with psychotic disorders tend to reflect substance availability and hence show the same patterns of substance use as are found in the general population (Hall, 1998).

The common co-occurrence of alcohol use and anxiety disorders has suggested the ‘tension reduction’ hypothesis (Cappell & Greeley, 1987). This hypothesis proposes that persons with anxiety disorders use alcohol to relieve anxiety or distress, and that problematic use becomes more likely (being reinforced) because alcohol becomes the means to control these negative mood states (Cappell & Greeley, 1987). This hypothesis is consistent with the acute anxiolytic effects of alcohol (Allan, 1995). However, it is less consistent with what is known about the longer-term effects of alcohol consumption. The effects of chronic alcohol use in high doses include increased anxiety (Stockwell & Bolderston, 1987; Stockwell, Hodgson, & Rankin,
Studies of phobic disorders have also found that phobic anxiety is not alleviated by alcohol use (Marshall, 1997). A more general form of the self-medication hypothesis proposes that substances are used in an attempt to relieve a variety of dysphoric moods, such as depression and anxiety, general malaise and boredom (Mueser et al., 1998). Research on self-reported reasons for substance use has provided some support for this notion (e.g. Warner et al., 1994); but it can be argued that alleviating dysphoria is simply one among many risk factors — such as poor social skills, poor social functioning and peer group influences — that increase the likelihood of both substance use and mental disorders (Mueser et al., 1998).

**Substance use problems cause mental health problems**

A different type of direct causal hypothesis is that substance use problems precipitate mental health problems. For example, there is evidence that some persons may develop depression that is secondary to alcohol dependence (Marc A. Schuckit et al., 1997) in the sense that it develops after alcohol dependence and is likely to remit with abstinence from alcohol (Brown & Schuckit, 1988).

There has also been considerable debate over whether cannabis use is causally related to schizophrenia (Blanchard, Brown, Horan, & Sherwood, 2000; Hall, 1998; Hall & Degenhardt, 2000; McKay & Tennant, 2000; Mueser et al., 1998; Thornicroft, 1990; Thornicroft, Meadows, & Politi, 1992). Some have argued that cannabis use can trigger a ‘cannabis psychosis’ (Solomons, Neppe, & Kuyl, 1990), while others have argued that its use might precipitate schizophrenia in vulnerable individuals (Andreasen, Allebeck, & Rydberg, 1987).

Comorbidity between different substance use problems has also been explained in causal terms. For instance, it has been hypothesised that the use of cannabis leads to the later use of other illicit drugs (O'Donnell & Clayton, 1982). There has been a great deal of debate about this ‘gateway hypothesis’. A strong relationship exists between the use of cannabis and the later use of other illicit substances (Fergusson & Horwood, 1997; Fergusson & Horwood, 2000; Kandel & Faust, 1975; Kandel, Yamaguchi, & Chen, 1992), and it persists after statistical control for a wide range of personal, family background and environmental factors (Fergusson & Horwood, 2000). Nevertheless, it could be that other variables account for the association, which have not been considered in research to date. Alternatively, common genetic factors may play some role in increasing the likelihood of both cannabis use and other substance use, a possibility that has been given some support by twin studies (Tsuang et al., 1998). These possibilities are considered below.

**Indirect causal relationship**

An indirect causal relationship would exist between two comorbid disorders if one disorder had an effect upon another factor that, in turn, increased the likelihood of developing the second disorder. For example, research has shown that the presence of early-onset substance use disorders reduces the likelihood of completing high school, entering tertiary education, and completing tertiary education (Kessler, Foster, Saunders, & Stang, 1995). Difficulties encountered because of poor educational achievement might subsequently increase the likelihood of other problems, such as depression and continued substance use problems.
Similarly, persons who are alcohol dependent may be more likely to lose their jobs because of poor work performance or absenteeism. Indeed, one of the criteria for DSM-IV substance use disorders is disruption to, or failure to, complete roles such as occupational requirements (American Psychiatric Association, 1994). Unemployment could then lead to depression because of the lack of a regular income and perceived damage to their career.

Common factors
Common risk factors may well explain an association between two disorders (Caron & Rutter, 1991; Kessler, 1995; Mueser et al., 1998). If disorders are predominantly the result of a set of risk factors and these sets are the same or similar for two disorders, it may well be the case that ‘comorbidity’ reflects the fact that the pathways by which persons develop one disorder are the same as those by which they develop another. These common factors might be biological, personality, social and environmental, or a combination of these factors.

Biological factors

Neurotransmitter function
There is suggestive evidence that common physiological factors may explain the co-occurrence of different substance use disorders (homotypic comorbidity). This is plausible given that different substances act upon similar brain loci and upon the same neurotransmitter systems (Koob & Moal, 1997; Krishnan-Sarin, Rosen, & O’Malley, 1999; Nutt, 1997). Furthermore, some of the underlying neural substrates of mental disorders and substance use disorders are similar. There is considerable evidence that both substance use disorders and mental disorders are characterised by disturbances in monoamine neurotransmitter function (Doris, Ebmeier, & Shajahan, 1999; Iqbal & van Praag, 1995; Koob & Moal, 1997; Koob & Le Moal, 2001). Some have argued that one reason for comorbidity between alcohol use disorders and anxiety disorders may be reduced serotonin function (Tollefson, 1991).

Genetic factors
The possibility of a common genetic vulnerability to problematic use of different substances was examined in a sample of male twins (True et al., 1999; Tsuang et al., 1998). One of these studies examined the genetic and environmental contributions to illicit substance abuse of, and dependence on, cannabis, stimulants, sedatives, opiates and psychedelics (Tsuang et al., 1998). It found that while the vulnerability to dependence upon different substance types had some unique (drug-specific) genetic effects (0% for psychedelics, 5% sedatives, 9% stimulants, 11% cannabis and 38% heroin) there was a significant common genetic component. This comprised 6% of the variance for heroin use disorders, 22% for cannabis, stimulants, sedatives, and 26% for psychedelic use disorders. Analysis revealed that a ‘common vulnerability’ model provided the simplest explanation of the data, with around one third of the variance of this common vulnerability caused by genetic effects.

A similar analysis of alcohol and nicotine dependence (True et al., 1999) found that there was a significant common genetic vulnerability (r = 0.68) to both nicotine and alcohol dependence among male twins, with 26% of the variance in the risk for alcohol dependence shared with the genetic risk of nicotine dependence. This research needs to be replicated among female twins.
Twin studies have also provided some evidence that there are common genetic influences upon substance use disorders and mental disorders (i.e. for heterotypic comorbidity). For example, research has suggested that common genetic factors increase the risk of alcohol dependence, anxiety symptoms, and affective symptoms (Tambs, Harris, & Magnus, 1997).

A twin study of women also found that there were significant common genetic factors implicated in the comorbidity between major depression and tobacco smoking (K. Kendler et al., 1993). This study found that the heritability of liability to tobacco smoking and major depression was 55% and 48%, respectively. Analyses were conducted to examine whether there was a causal relationship between tobacco smoking in major depression, or whether common factors accounted for the association that was observed between the two. The best explanation of the co-occurrence of tobacco smoking and major depression in this sample was a common genetic factor. There was no evidence of common environmental factors. The correlation between smoking and major depression due to these genetic factors was estimated at + 0.56 (K. Kendler et al., 1993).

**Individual factors**
Temperament is commonly associated with substance use and mental health, particularly the trait of neuroticism. Persons scoring high on neuroticism have been characterised as more anxious, worrying, depressed and moody (Eysenck & Eysenck, 1991). Persons who are heavy substance users score higher on neuroticism than those who are not (Francis, 1996). Persons who suffer from mood and anxiety disturbances also have higher levels of trait neuroticism, and a considerable part of the liability to both mood and anxiety disorders is explained by higher levels of trait neuroticism (Andrews, 1996; Andrews, Stewart, Allen, & Henderson, 1990).

**Social and environmental factors**
Common genetic influences or individual factors play an incomplete part in explaining comorbidity. Twin studies have also shown that shared environmental factors increase the likelihood of both alcohol dependence and major depression among women (Tambs et al., 1997; True et al., 1999; Tsuang et al., 1998). Tsuang (1998) and colleagues found that two thirds of the common vulnerability to different types of illicit drug use disorders was explained by shared environmental factors. This is not surprising, given that there is a wealth of evidence that a number of factors are common to both mental disorders and substance use disorders. For example, social disadvantage is more common among persons who are problematic substance users (Institute of Medicine, 1996); who meet criteria for mood disorders and anxiety disorders (Blazer, 1995; Kessler et al., 1994; Weissman, Livingston Bruce, Leaf, Flroio, & Holzer, 1991); and who meet criteria for psychotic disorders, and there is evidence to suggest that this is not merely because of social drift after developing the disorder (Mueser et al., 1998). For all these groups of disorders, studies have shown that there are higher rates of separation and divorce, and a lower likelihood that persons will be married or in a defacto relationship (Blazer, 1995; Jablensky, Sartorius, & Ernberg, 1991; Kessler et al., 1994; Weissman et al., 1991). There is also a number of other factors that have been similarly associated with substance use disorders and with mental disorders, such as parental psychiatric illness and family dysfunction (Fergusson, Horwood, & Lawton, 1990; Fergusson,
Horwood, & Lynskey, 1994; Rutter, 1987; Velez, Johnson, & Cohen, 1989). It is possible that these social factors serve to increase the apparent ‘comorbidity’ of mental disorders.

Kendler and colleagues (1993) also found that common genetic influences explained the co-occurrence of nicotine dependence and major depression. Another study examined this issue using data from a longitudinal study of adolescents from Christchurch, New Zealand (Fergusson, Lynskey, & Horwood, 1996). It examined the association between nicotine dependence and major depression while controlling for a large number of demographic variables, family background characteristics, and personal characteristics. It found — in apparent contrast to the Kendler study — that the co-occurrence of the two could be almost completely explained by common environmental factors, and that the most parsimonious explanation of the relationships between the two did not include a causal relationship.

While this may appear to be a contradiction of the Kendler study, it must be borne in mind that genetic and environmental factors are not independent. There is evidence, for example, of a genetic influence both upon exposure to stressful life events, and in responses to them (Kendler, 1998; Kendler et al., 1995; K. S. Kendler et al., 1993). Hence, in controlling for a large number of environmental factors, Fergusson and colleagues may well have been controlling for some of the genetic influences upon both nicotine dependence and major depression. What is clear from both of these studies, regardless of which sort of influence accounted for the comorbidity (environmental and/or genetic influences), both studies agreed in that there was no evidence that major depression caused nicotine dependence or vice versa.

A similar conclusion was reached by Lynskey and colleagues (1998) in an examination of liability to alcohol, tobacco and cannabis use using the same New Zealand cohort. This study found that the simplest explanation of the relationship between alcohol, tobacco and cannabis use was a ‘common vulnerability’ model of increased liability to the use of the three substances, which could be completely explained by a large number of environmental factors included in the analyses (Lynskey et al., 1998).

Summary

While at present there remains much that is not known about the causes of comorbidity, there is increasing evidence to suggest that simple causal hypotheses may not easily explain the association. There is a broad convergence of risk factors for both problematic substance use and mental disorders; a plausible hypothesis for the comorbidity between these disorders is that substance use and mental disorders (mood disorders, anxiety disorders, personality disorders and psychotic disorders) share common risk factors and life pathways. A number of longitudinal cohort and twin studies have explicitly examined this hypothesis and have concluded that common factors explain the comorbidity between alcohol, tobacco and cannabis use (Lynskey et al., 1998); dependence on different illicit drugs (Tsuang et al., 1998); alcohol and nicotine dependence (True et al., 1999); and nicotine dependence and major depression (Fergusson et al., 1996; K. Kendler et al., 1993).
Conclusions

Interest in, and research on, comorbidity has been increasing over past decades. It has emerged as one of the complex issues facing theorists, clinicians and policymakers who are responsible for providing funding for mental health problems. This chapter has discussed the concept of comorbidity and discussed the importance of community samples in documenting the extent of comorbidity in the community. As will be documented in later chapters, these surveys have established that comorbidity occurs. This has implications for theory, prevention and treatment of mental health problems. There are a number of potential explanations for comorbidity.

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