

## How common is comorbidity and why does it occur?

M. TEESSON<sup>1</sup>, L. DEGENHARDT<sup>1</sup>, H. PROUDFOOT<sup>1</sup>, W. HALL<sup>2</sup>, & M. LYNSKEY<sup>3</sup>

<sup>1</sup>National Drug and Alcohol Research Centre, University of New South Wales, Sydney, New South Wales, <sup>2</sup>Office of Public Policy and Ethics, Institute for Molecular Bioscience, University of Queensland, Brisbane, Queensland, Australia, and

<sup>3</sup>Washington University in St Louis, MO, USA

### Abstract

Comorbidity is most generally defined as the co-occurrence of two or more mental health problems. Comorbidity between drug and other psychological disorders has emerged as a major clinical, public health and research issue over the past few decades. The reasons for comorbidity are complex. Furthermore, comorbidity is often associated with poor treatment outcome, severe illness course, and high service utilisation. This presents a significant challenge with respect to the identification, prevention and management of people with comorbid disorders. The unmet need for treatment within this group is considerable and the lack of research is unacceptable. This paper will 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). 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 [APA], 1994; World Health Organization, 1993). Comorbidity between substance use disorders and other mental disorders has gained increasing prominence in psychiatry and psychology within the past few decades and the reasons for this comorbidity are discussed in this paper.

### How common is comorbidity?

It is critically important to study patterns of comorbidity in general population samples because patterns observed in clinical samples will reflect those in the general community. The first large-scale

studies of comorbidity were undertaken in the United States, the first being the Epidemiological Catchment Area Survey (ECA) in which approximately 20,000 respondents aged 18 years and over were surveyed. Among those respondents with a lifetime alcohol use disorder (abuse or dependence), 37% had at least one other mental disorder and 22% had another drug use disorder (Regier et al., 1990). Conversely, among individuals with any lifetime mental disorder, 22% had a lifetime history of an alcohol use disorder and 15% had a lifetime history of a drug use disorder. The highest rates of substance use disorders were found among those with schizophrenia (47%).

The more recent US National Comorbidity Study (NCS) was specifically designed to examine the extent of comorbidity between substance use and mental disorders (Kessler et al., 1994). The NCS was conducted in 1991 with approximately 8,000 respondents aged between 15 and 54 years. That study measured both lifetime and current prevalences of mental disorders. Lifetime comorbidities were found to be comparable to those found in the ECA. The prevalence of any current (12-month) diagnosis was 29.5% (Kessler et al., 1994). Among

those with a 12-month diagnosis of any substance use disorder 36% had at least one anxiety disorder while 25% had at least one affective disorder. Among those with a 12-month diagnosis of any mental disorder, 15% had at least one co-occurring substance use disorder (Kessler et al., 1996).

In 1997, the Australian Bureau of Statistics was funded by the Commonwealth Mental Health Branch to undertake the National Survey of Mental Health and Wellbeing (NSMHWB), a survey of the mental health of the Australian adult population. A nationally representative sample of 10,641 Australians were surveyed (Henderson, Andrews, & Hall, 2000). Eighteen per cent of respondents (aged 18–90 years) met criteria for a *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., *DSM-IV*) mental disorder in the past 12 months. This is somewhat lower than the NCS figure of 29.5%. This difference may be due to the age groups covered (younger ages in the NCS); the omission of certain disorders from the NSMHWB (in particular anti-social personality disorder and specific phobias); and the use of *DSM-IV* diagnoses in the NSMHWB and *DSM-III-R* in the NCS.

In line with the US studies, results of the NSMHWB showed a considerable degree of comorbidity in substance use disorders and other mental disorders (Teesson, Hall, Lynskey, & Degenhardt, 2000). Approximately one in four persons with an anxiety, affective or substance use disorder also had at least one other mental disorder. That is, they had two or more different classes of disorder, such as an anxiety and affective disorder, or an anxiety and a substance use disorder.

Figures 1 and 2 summarise the prevalence and comorbidity data found in the survey. Among those individuals with mental disorders, marginally more women than men had at least one other comorbid

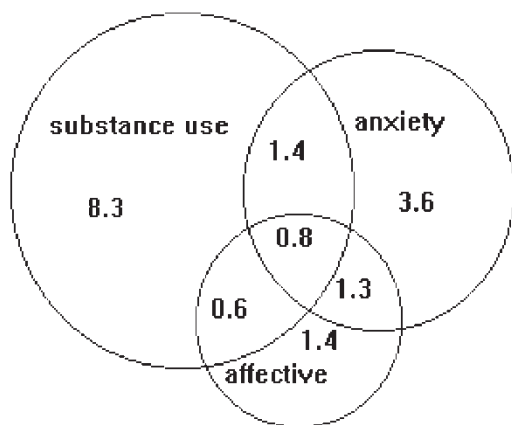


Figure 1. Prevalence (%) of single and comorbid affective, anxiety and substance use disorders among Australian men in the past year.

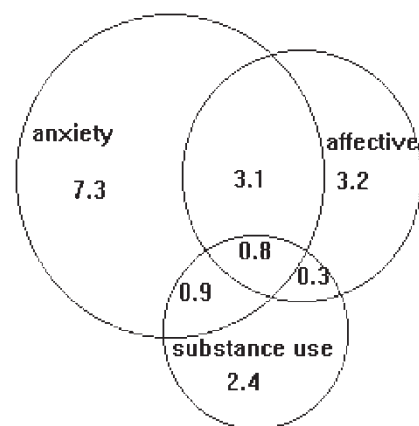


Figure 2. Prevalence (%) of single and comorbid affective, anxiety and substance use disorders among Australian women in the past year.

mental disorder (28% of women as against 24% of men with any of these mental disorders). The patterns of comorbidity differed between men and women, reflecting the differences in prevalence within the sexes for the individual disorders. Among women, affective and anxiety disorders most often occurred together, accounting for three quarters of women who had more than one mental disorder. Among men, comorbid disorders more often involved an anxiety or an affective disorder in combination with a substance use disorder. These combinations of disorders affected two thirds of men who had more than one mental disorder. A small proportion of men (0.8%) and women (0.8%) had all three types of disorder (i.e., an anxiety, affective and substance use disorder) (Andrews, Hall, Teesson, & Henderson, 1999).

The NSMHWB included a low-prevalence study of psychotic disorders. That study demonstrated that drug and alcohol use disorders were highly prevalent in those with psychotic illnesses. Nicotine was the most commonly used drug in that sample, with 67% using nicotine in the previous 12 months. Lifetime diagnoses of alcohol use disorders were found in 30% of the sample and cannabis use disorder in 25%. Jablensky et al. also interviewed a sample of people with psychoses who were marginalised or homeless and not seen by mainstream services. They found more than double the levels of comorbid substance abuse, which contributes to the intensification of psychotic symptoms found in this group.

### 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: (a) that there is a

direct causal relationship between the two, with the presence of one disorder making another more likely to develop; (b) 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 (c) that there are common factors that increase the risk of both disorders. These are discussed in more detail here.

### **Direct causal relationship**

#### *Mental health problems cause substance use problems*

This hypothesis holds that the relationship between substance use disorders and other mental health problems is a result of persons with mental health problems using substances to reduce the symptoms of their illness and then developing problematic use as a result of over-use (Khantzian, 1985; 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. However, the evidence that specific drugs are used to treat specific symptoms is less than compelling (Mueser, Drake, & Wallach, 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). Instead, 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 so-called 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, 1982).

#### *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 (Schuckit, Tipp, & Bergman et al., 1997; Schuckit, Tipp, & Bucholz 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, & Kuyf, 1990), while others have argued that its use might precipitate schizophrenia in vulnerable individuals (Andreasson, Allebeck, & Rydberg, 1987).

Although the direct causal models are appealing in their simplicity, it could also be that other variables account for the association, which have not been considered in research to date. Alternatively, common predisposing factors may play some role in increasing the likelihood of both substance use and mental disorders (Tsuang et al., 1998). These possibilities are considered here.

#### *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.

Similarly, persons who are alcohol dependent may be more likely to lose their job 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 (APA, 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 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 has been examined in a sample of male twins (True et al., 1999; Tsuang et al., 1998). Twin studies have also provided some evidence that there are common genetic influences upon substance use disorders and mental disorders. 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 (Kendler, Neale, MacLean et al., 1993). That 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  $r = +0.56$  (Kendler, Neale, MacLean 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).

This is not surprising, given that there is a wealth of evidence that a number of social and environmental factors are common to both mental disorders and substance use disorders. For example, social disadvantage is more common among persons who (a) are problematic substance users (Institute of Medicine, 1996); (b) meet criteria for mood disorders and anxiety disorders (Blazer, 1995; Kessler et al., 1994; Weissman, Livingston Bruce, Leaf, Floio, & Holzer, 1991); and (c) meet criteria for psychotic disorders. Further, 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.

A longitudinal study of adolescents from Christchurch, New Zealand (Fergusson, Lynskey, & Horwood, 1996) 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 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.

Although this may appear to be a contradiction of the Kendler, Neale, MacLean et al. (1993) study that found that common genetic influences explained the co-occurrence of nicotine dependence and major depression, 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; Kendler, Neale, Kessler, Heath, & Eaves, 1993). Hence, in controlling for a large number of environmental factors, Fergusson et al. 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, is that common risk factors (environmental and/or genetic influences) significantly influence comorbidity and that there was no evidence that major depression caused nicotine dependence or vice versa.

### What are the implications of comorbidity research?

The longitudinal and twin studies reviewed are increasingly finding shared risk factors for a wide range of problem behaviours including mental disorders and substance use disorders. These findings have several implications for the design and implementation of clinical programs and research.

Recognising that much of the association between different problem behaviors arises from the influence of common risk factors, interventions should target and attempt to modify the known risk factors for substance use and mental disorders. One such study has been reported by Tremblay, Pagani-Kurtz, Masse, Vitaro, and Pihl (1995) who implemented a comprehensive intervention program for at-risk kindergarten-aged boys that included both parent training and social skills training for the boys. At follow up in adolescence the intervention group showed significantly reduced rates of aggressive behavior and increased grade retention. Unfortunately, substance use was not assessed at follow up and the impact of such a program on later substance use remains an interesting question.

The research evidence to date suggests that although we should intervene to prevent or delay the onset of substance use behaviors during adolescence, such interventions should not focus solely on substance use. They should target a range of potentially health-threatening behaviors including substance use, sexual risk-taking and problems of personal adjustment because many of these behaviors co-occur due to shared risk factors (Lynskey, 1998).

### Conclusion

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, Fergusson, & Horwood, 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; Kendler, Neale, MacLean et al., 1993). The implications for prevention, although promising, remain to be tested in large-scale intervention studies.

### References

- Allan, C. A. (1995). Alcohol problems and anxiety disorders - A critical review. *Alcohol & Alcoholism*, 145-151.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.) Washington, DC: The Author.
- Andreasson, S., Allebeck, P., & Rydberg, U. (1987). Cannabis and schizophrenia: A longitudinal study of Swedish conscripts. *Lancet*, 2, 1483-1486.
- Andrews, G. (1996). Comorbidity and the general neurotic syndrome. *British Journal of Psychiatry - Supplement*, 76-84.
- Andrews, G., Hall, W., Teesson, M., & Henderson, S. (1999). *The mental health of Australians*. Canberra: Commonwealth Department of Health and Family Services.
- Andrews, G., Stewart, G., Allen, R., & Henderson, A. S. (1990). The genetics of six neurotic disorders: A twin study. *Journal of Affective Disorders*, 19, 23-29.
- Blanchard, J. J., Brown, S. A., Horan, W. P., & Sherwood, A. R. (2000). Substance use disorders in schizophrenia: Review, integration, and a proposed model. *Clinical Psychology Review*, 20, 207-234.
- Blazer, D. (1995). Mood disorders: Epidemiology. In H. Kaplan, & B. Sadock (Eds.), *Comprehensive textbook of psychiatry IV* (pp. 1079-1089). Baltimore, MA: Williams and Wilkins.
- Brown, S., & Schuckit, M. (1988). Changes in depression amongst abstinent alcoholics. *Journal of Studies in Alcohol*, 52, 37-43.
- Cappell, H., & Greeley, J. (1987). Alcohol and tension reduction: An update on research and theory. In H. T. Blane, & K. E. Leonard (Eds.), *Psychological theories of drinking and alcoholism* (pp. 15-54). London: Guilford Press.
- Caron, C., & Rutter, M. (1991). Comorbidity in child psychopathology: Concepts, issues and research strategies. *Journal of Child Psychology and Psychiatry*, 32, 1063-1080.

- Dixon, L., Haas, G., Weiden, P., Sweeney, J., & Frances, A. (1991). Drug abuse in schizophrenic patients: Clinical correlates and reasons for use. *American Journal of Psychiatry*, *148*, 224–230.
- Doris, A., Ebmeier, K., & Shajahan, P. (1999). Depressive illness. *Lancet* *354*, 1369–1375.
- Eysenck, H. J., & Eysenck, S. B. G. (1991). *Manual of the Eysenck personality scales* (EPS Adult). London: Hodder & Stoughton.
- Feinstein, A. R. (1970). The pre-therapeutic classification of comorbidity in chronic disease. *Journal of Chronic Diseases*, *23*, 455–468.
- Fergusson, D., Horwood, J., & Lawton, M. (1990). Vulnerability to childhood problems and family social background. *Journal of Child Psychology and Psychiatry*, *31*, 1145–1160.
- Fergusson, D., Horwood, L., & Lynskey, M. (1994). Parental separation, adolescent psychopathology and problem behaviours. *Journal of the American Academy of Child and Adolescent Psychiatry*, *33*, 1122–1131.
- Fergusson, D. M., Lynskey, M. T., & Horwood, L. J. (1996). Comorbidity between depressive disorders and nicotine dependence in a cohort of 16-year-olds. *Archives of General Psychiatry*, *53*, 1043–1047.
- Francis, L. (1996). The relationship between Eysenck's personality factors and attitude towards substance use among 13–15 year olds. *Personality and Individual Differences*, *21*, 633–640.
- Hall, W. (1998). Cannabis use and psychosis. *Drug and Alcohol Review*, *17*, 433–444.
- Hall, W., & Degenhardt, L. (2000). Cannabis use and psychosis: A review of clinical and epidemiological evidence. *Australian and New Zealand Journal of Psychiatry*, *34*, 26–34.
- Henderson, S., Andrews, G., & Hall, W. (2000). Australia's mental health: An overview of the general population survey. *Australian and New Zealand Journal of Psychiatry*, *34*, 197–205.
- Institute of Medicine. (1996). *Pathways of addiction*. Washington: National Academy Press.
- Iqbal, N., & van Praag, H. M. (1995). The role of serotonin in schizophrenia. *European Neuropsychopharmacology*, *5*, 11–23.
- Jablensky, A., Sartorius, N., & Ernberg, G. (1991). Schizophrenia: Manifestations, incidence and course in different cultures. A World Health Organization Ten-Country Study. *Psychological Medicines Supplement*, *20*.
- Kendler, K., Neale, M., MacLean, C., Heath, A., Eaves, L., & Kessler, R. (1993). Smoking and major depression: A causal analysis. *Archives of General Psychiatry*, *50*, 36–43.
- Kendler, K. S. (1998). Anna-Monika-Prize paper. Major depression and the environment: A psychiatric genetic perspective. *Pharmacopsychiatry*, *31*, 5–9.
- Kendler, K. S., Kessler, R. C., Walters, E. E., MacLean, C., Neale, M. C., Heath, A. C., et al. (1995). Stressful life events, genetic liability, and onset of an episode of major depression in women. *American Journal of Psychiatry*, *152*, 833–842.
- Kendler, K. S., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1993). A twin study of recent life events and difficulties. *Archives of General Psychiatry*, *50*, 789–796.
- Kessler, R. C. (1995). Epidemiology of psychiatric comorbidity. In M. T. Tsuang, M. Tohen, & G. E. P. Zahner (Eds.), *Textbook in psychiatric epidemiology* (pp. 179–197). New York: Wiley and Sons.
- Kessler, R. C., Foster, C. L., Saunders, W. B., & Stang, P. E. (1995). Social consequences of psychiatric disorders, I: Educational attainment. *American Journal of Psychiatry*, *152*, 1026–1032.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *51*, 8–19.
- Kessler, R. C., Nelson, C. B., McGonagle, K. A., Edlund, M. J., Frank, R. G., & Leaf, P. J. (1996). The epidemiology of co-occurring addictive and mental disorders: Implications for prevention and service utilization. *American Journal of Orthopsychiatry*, *66*, 17–31.
- Khantzian, E. J. (1985). The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. *American Journal of Psychiatry*, *142*, 1259–1264.
- Koob, G., & Moal, M. L. (1997). Drug abuse: Hedonic homeostatic dysregulation. *Lancet*, *278*, 52–58.
- Koob, G. F., & Le Moal, M. (2001). Drug addiction, dysregulation of reward, and allostasis. *Neuropsychopharmacology*, *24*, 97–129.
- Lynskey, M. T., Fergusson, D. M., & Horwood, L. J. (1998). The origins of the correlations between tobacco, alcohol, and cannabis use during adolescence. *Journal of Child Psychology and Psychiatry*, *39*, 995–1005.
- Lynskey, M. T. (1998). Broadening the target of drug prevention. *FAS Drug Policy Analysis Bulletin*.
- McKay, D. R., & Tennant, C. C. (2000). Is the grass greener? The link between cannabis and psychosis. *Medical Journal of Australia*, *172*, 284–286.
- Mueser, K. T., Drake, R. E., & Wallach, M. A. (1998). Dual diagnosis: A review of etiological theories. *Addictive Behaviors*, *23*, 717–734.
- Noordsy, D. L., Drake, R. E., Teague, G. B., Osher, F. C., Hurlbut, S. C., Beaudett, M. S., et al. (1991). Subjective experiences related to alcohol use among schizophrenics. *Journal of Nervous & Mental Disease*, *179*, 410–414.
- Pope, H. (1979). Drug abuse and psychopathology. *New England Journal of Medicine*, *301*, 1341–1342.
- Regier, D. A., Farmer, M. E., Rae, D. S., Locke, B. Z., Keith, S. J., Judd, L. L., et al. (1990). Comorbidity of mental disorders with alcohol and other drug abuse: Results from the Epidemiologic Catchment Area (ECA) study. *Journal of the American Medical Association*, *264*, 2511–2518.
- Rutter, M. (1987). Parental mental disorder as a psychiatric risk factor. In R. Hales, & A. Frances (Eds.), *Psychiatric update: American Psychiatric Association: Annual review* (pp. 647–663). Washington, DC: American Psychiatric Press.
- Schuckit, M. A., Tipp, J. E., Bergman, M., Reich, W., Hesselbrock, V. M., & Smith, T. L. (1997). Comparison of induced and independent major depressive disorders in 2,945 alcoholics. *American Journal of Psychiatry*, *154*, 948–957.
- Schuckit, M. A., Tipp, J. E., Bucholz, K. K., Nurnberger, J. I., Hesselbrock, V. M., Crowe, R. R., et al. (1997). The life-time rates of three major mood disorders and four major anxiety disorders in alcoholics and controls. *Addiction*, *92*, 1289–1304.
- Solomons, K., Neppe, V. M., & Kuyl, J. M. (1990). Toxic cannabis psychosis is a valid entity. *South African Medical Journal*, *78*, 476–481.
- Stockwell, T., & Bolderston, H. (1987). Alcohol and phobias. *British Journal of Addiction*, *82*, 971–979.
- Stockwell, T., Hodgson, R., & Rankin, H. (1982). Tension reduction and the effects of prolonged alcohol consumption. *British Journal of Addiction*, *77*, 65–73.
- Tamb, K., Harris, J. R., & Magnus, P. (1997). Genetic and environmental contributions to the correlation between alcohol consumption and symptoms of anxiety and depression: Results from a bivariate analysis of Norwegian twin data. *Behavior Genetics*, *27*, 241–250.
- Teesson, M., Hall, W., Lynskey, M., & Degenhardt, L. (2000). Alcohol- and drug-use disorders in Australia: Implications of the National Survey of Mental Health and Wellbeing. *Australian and New Zealand Journal of Psychiatry*, *34*, 206–213.

- Thornicroft, G. (1990). Cannabis and psychosis. Is there epidemiological evidence for an association? [Erratum appears in *Br J Psychiatry* 1990; 157: 460.]. *British Journal of Psychiatry*, 157, 25–33.
- Thornicroft, G., Meadows, G., & Politi, P. (1992). Is “cannabis psychosis” a distinct category? *European Psychiatry*, 7, 277–282.
- Tollefson, G. D. (1991). Anxiety and alcoholism: A serotonin link. *British Journal of Psychiatry - Supplement*, 34–39.
- Tremblay, R. E., Pagani-Kurtz, L., Masse, L. C., Vitaro, F. & Pihl, R. O. (1995). A bimodal preventive intervention for disruptive kindergarten boys: Its impact through mid-adolescence. *Journal of Consulting and Clinical Psychology*, 63, 560–568.
- True, W., Xian, H., Scherrer, J., Madden, P., Bucholz, K., Heath, A. et al. (1999). Common genetic vulnerability for nicotine and alcohol dependence. *Archives of General Psychiatry*, 56, 655–661.
- Tsuang, M. T., Lyons, M. J., Meyer, J. M., Doyle, T., Eisen, S. A., Goldberg, J., et al. (1998). Co-occurrence of abuse of different drugs in men: The role of drug-specific and shared vulnerabilities [see comments]. *Archives of General Psychiatry*, 55, 967–972.
- Velez, C., Johnson, J., & Cohen, P. (1989). A longitudinal analysis of selected risk factors for childhood psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 861–864.
- Weissman, M., Livingston Bruce, M., Leaf, P., Floio, L., & Holzer, C. (1991). Affective disorders. In L. Robins, & D. Regier (Eds.), *Psychiatric disorders in America* (pp. 53–80). New York: MacMillan.
- World Health Organization (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva: The Author.

Copyright of Australian Psychologist is the property of Taylor & Francis Ltd and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.

Copyright of Australian Psychologist is the property of Taylor & Francis Ltd. The copyright in an individual article may be maintained by the author in certain cases. Content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.