MENTAL HEALTH IN NEW ZEALAND FROM A PUBLIC HEALTH PERSPECTIVE

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This chapter reviews the evidence concerning the prevention of the two cardinal mood disorders, major depression and bipolar disorder. The chapter has three parts. The first two consider the definition, epidemiology, aetiology, and prevention of major depression and bipolar disorder. The final part presents conclusions and recommendations.

**MAJOR DEPRESSION**

**DEFINITION OF MAJOR DEPRESSION**

A major problem in attempting to define clinically important depression is the ubiquity of depressive feelings. In defining clinically notable depression, a key task is to distinguish normal emotions from abnormal depression. Emotional states like sadness, feeling ‘blue’, or tearfulness are part of normal human experience. Normally, these emotions are relatively transient and not associated with significant distress or functional impairment. On occasion, sadness becomes exaggerated, pervasive, nearly unshakeable, and is accompanied by real impairment in a person’s life.

Since the times of the ancient Greek physicians, many definitions of depression have been presented. These definitions have in common an attempt to define depressive signs and symptoms that are abnormal in degree and to identify those depressive states whose associated suffering would argue for some form of treatment. One recent definition of significant, ‘major’, depression is that of the *DSM-IV* (APA 1994a) that was developed by a task force in the United States and which appears to be an up-to-date, international consensus view that would correspond reasonably closely to what many clinicians would consider to be a condition worthy of some form of treatment. The core symptoms of significant, ‘major’, depression include depressed mood, diminished interest and pleasure in most activities, weight change, change in appetite, insomnia or hypersonnia, fatigue, poor concentration, feelings of worthlessness, and suicidal thinking. Five or more of these must be present for at least two weeks, and represent a change from previous functioning.

**EPIDEMIOLOGY OF MAJOR DEPRESSION**

**Lifetime Prevalence**

The lifetime prevalence of major depression in random community samples is a matter of some contention (Parker 1987). The available estimates vary greatly depending on factors such as geographic location, how a sample was acquired, which diagnostic criteria were used, and how the interviewer
asked about depressive symptoms. Complete coverage of these important technical issues is beyond the scope of this chapter; however, these factors may cause wide variation in the reported lifetime prevalence results.

The lifetime prevalence estimates of major depression in international samples of adults thus vary widely: about 2 percent in Hong Kong (Chen et al 1993); 7.8 percent averaged across five cities in the US (Weissman et al 1991); and 17.1 percent in a national probability sample in the US (Kessler et al 1994). Kendler and colleagues compared nine sets of diagnostic criteria in a large epidemiological sample: the lifetime prevalence estimates for major depression ranged from 12–33 percent depending on the criteria used (Kendler et al 1992).

We are fortunate to have New Zealand data from several different studies. In Christchurch, the lifetime prevalence of major depression in adults selected at random from census data was 12.6 percent (Wells JE et al 1989a) and 33.2 percent in another study of adults randomly selected from the national electoral roll (A Beutrais, P Joyce, R Mulder, personal communication, June 1996). The lifetime prevalence of major depression was 16.7 percent in a cohort of 15-year-olds in Dunedin (Feehan et al 1994) and 4.9 percent in a cohort of 15-year-olds in Christchurch (Fergusson et al 1993).

It is reasonable to suspect that the true lifetime prevalence of major depression is in the range of 10–20 percent. This places major depression among the more common human afflictions. Of note, the lifetime prevalence of major depression in Christchurch was among the highest in 12 similarly conducted studies in nine countries worldwide (Cross-National Collaborative Group 1992).

Current major depression is especially prevalent in primary care medical settings with a point prevalence of about 10 percent (Schulberg et al 1985; Ormel et al 1994).

**Psychiatric Comorbidity**

Major depression often occurs in conjunction with other mental and physical disorders (Maser and Cloninger 1990). The causes of overlap between these disorders are complex (Kendler et al 1995) and not well elucidated (Neale and Kendler 1995). Alcohol dependence, several different anxiety disorders, and eating disorders frequently complicate the clinical portrait of major depression (Depression Guideline Panel 1993a).

**Natural History, Morbidity and Mortality**

The natural history of major depression is highly variable. For depressed individuals seen in psychiatric settings, recovery from the index episode is the norm although persistent depression is found in about 10 percent (Piccinelli and Wilkinson 1994). Notably, 75 percent experienced at least one other episode of depression over a decade of follow-up (Piccinelli and Wilkinson 1994). However, many people who experience a significant depressive syndrome never receive any form of treatment (Regier et al 1978, 1993).

Considerable morbidity is associated with major depression. Epidemiological studies of community samples (Broadhead et al 1990) and primary care medical attendees (Wells KB et al 1989b) have shown that the presence of major depression substantially increases the risk of disability and impaired role functioning. Patients with major depression had similar or worse functioning than those with
eight major chronic medical conditions (Wells KB et al 1989b). Substantial excess mortality is also associated with major depression due to suicide (Berglund and Nilsson 1987; Zilber et al 1989) and other causes (Tsuang and Woolson 1978; Black et al 1987a; Zilber et al 1989). Up to 15 percent of patients hospitalised for psychiatric treatment of depression eventually die by suicide. Despite these findings, major depression is substantially underdiagnosed and undertreated (Regier et al 1978; Klerman 1989; Klerman and Weissman 1992; Regier et al 1993).

**AETIOLOGY OF MAJOR DEPRESSION**

**Aetiological Models**

It is probably useful to conceptualise major depression as a ‘complex disorder’ (Lander and Schork 1994) that is the result of the interplay of genetic, biological, and environmental factors.

Kendler and colleagues have published an interesting paper that attempted to develop an empirical model that combined and ranked genetic and environmental risk factors for major depression (Kendler et al 1993b). It is important to note that this model attempted to predict new episodes of major depression over about a two-year period rather than lifetime major depression. These authors studied 680 pairs of female twins three times over a 30-month period. The model included the interactions among a considerable number of variables that included traumatic experiences, genetic factors, personality variables, and interpersonal stressors. Strong predictors were recent stressful life events, genetic risk factors, past history of major depression, and neuroticism. Predictors of intermediate strength were recent life difficulties, lack of parental warmth, and lifetime traumas. Modest predictors were lack of social support and parental loss.

While this model was clearly preliminary (and only partially successful) it seems to be a clinically useful model that addresses the interaction and relative importance of these variables. It assigned some meaning to the obfuscating terms ‘multifactorial’ and ‘bio-psycho-social’ and added a sense of order to the multitude of variables (ie, stressful life events, family history, past history, personality, etc) thought important in the genesis of major depression.

It is possible that the aetiology of major depression often depends upon the interaction of genetic and environmental risk factors (what has been termed ‘genetic control of sensitivity to the environment’ (Kendler and Eaves 1986)). Under this conceptualisation, the same life events will have markedly different effects depending upon an individual’s genetic complement. For example, stressful life events of similar magnitude may or may not trigger major depression in individuals with different genes.

**Risk Factors**

A number of variables have been found to be statistically associated with major depression in epidemiological studies. These factors include: being female (Wells JE et al 1989a; Weissman et al 1991; Kessler et al 1993; Weissman et al 1993); birth in more recent age cohorts (Klerman and Weissman 1989; Joyce et al 1990; Weissman et al 1991; Cross-National Collaborative Group 1992); being separated, divorced or in an unhappy marriage (Weissman 1987); stressful life events (Kendler et al 1993b); and subsyndromal depressive symptoms (Horwath et al 1992).
**Genetic Factors**

A family history of major depression has consistently been found to be a risk factor for major depression (Weissman 1987; Merikangas and Kupfer 1995). These findings suggest the influence of genetic factors on the liability to develop major depression.

Methodologically rigorous family studies of major depression have demonstrated its familiarity (ie, its tendency ‘to run in families’). First-degree relatives of probands with major depression have increased rates of major depression in comparison to normal controls and to those with non-affective psychiatric illness (Tsuang et al 1980; Gershon et al 1982; Weissman et al 1984; Andreasen et al 1987; McGuffin et al 1988; Heun and Maier 1993).

Twin studies can estimate the proportion of variance in liability to major depression due to genetic and shared and unshared environmental influences. The proportion of variance for developing major depression that can be attributed to additive genetic effects is in the 42–54 percent range (Torgersen 1986; McGuffin et al 1991; Kendler et al 1992). As determination of lifetime major depression is not particularly reliable (Bromet et al 1986; Kendler et al 1993a), when measurement error is taken into account, genetic influences may be as high as 71 percent (Kendler et al 1993a).

There have been three adoption studies of major depression. One study showed evidence supporting significant genetic influences (Wender et al 1986; Ingraham and Wender 1992), one study reported equivocal evidence (Cadoret 1978; Cadoret et al 1985), and one study found no significant genetic effects (von Knorring et al 1983).

**Summary**

Overall, our knowledge of the aetiology of major depression is limited. It is a complex disorder and the available evidence implicates several environmental risk factors and as-yet unspecified genetic risk factors.

**Prevention of Major Depression**

**Primary Prevention**

We are aware of no empirical studies of the primary prevention of major depression in the general population. If we do not know the aetiology of a disorder, preventing its development by interruption of its causal chain is a challenge. Primary prevention of major depression by interventions in the general population is not likely to be possible until we have a better understanding of its aetiology.

Several other factors diminish the possibility that primary preventive interventions can be developed for major depression. Firstly, many of the environmental risk factors implicated in the aetiology of major depression may be impossible or prohibitively expensive to correct. For example, it is difficult to imagine how altering the risk factors of sex and age cohort might be accomplished. Secondly, a number of the environmental risk factors for major depression may well exist in an interconnected matrix; altering one factor (even if possible) might have little influence on the other risks in its matrix and hence have little impact on the incidence of major depression.

Despite these difficulties, a few studies have tested the effects of interventions targeting people at risk. Results have looked promising but methodological problems limit interpretation of the findings. Interventions have included cognitive methods to gain control of mood (Muñoz et al 1995) and mobilisation of protective factors identified in at-risk groups (Vega and Murphy 1990).
Secondary Prevention

The secondary prevention (decreased prevalence through early detection and treatment) of major depression is possible and is the type of prevention for which the greatest body of data exists. There are a number of screening methods that have been developed and tested by which to detect the presence of major depression. Examples of these self-report questionnaires are the CES-D (Radloff 1977) and the Beck Depression Inventory (Beck et al 1961). These questionnaires can be completed in a few minutes and carry reasonable sensitivity for the detection of significant depression (Weissman et al 1977).

There are a number of locations in which screening for major depression could be performed. The Depression Awareness, Recognition, and Treatment programme of the US National Institute of Mental Health provided public education about major depression. In New Zealand, the general practice setting would be an obvious place in which to screen for and treat major depression. The majority of New Zealand adults visit a general practitioner at least once in any two-year period; detection could be readily accomplished in the surgery.

Once detected, there are a variety of methods by which major depression may be effectively treated. Brief psychological therapies such as cognitive-behavioural therapy (Elkin et al 1985) and interpersonal therapy (Klerman et al 1984) are of proven benefit in the treatment of major depression. There exist a sizeable number of antidepressant medications of proven efficacy and safety. A number of these treatments have also been tested in primary care medicine (Schulberg et al 1993).

It is important to note that secondary prevention of major depression is what many general practitioners, psychiatrists, and clinical psychologists do as a matter of course. However, given that only a minority of individuals with major depression receive any form of treatment (Regier et al 1978; Hornblow et al 1990; Regier et al 1993), improvements in detection and treatment are necessary.

Tertiary Prevention

Tertiary prevention entails the reduction of complications of an established condition and the minimisation of the risk of recurrence. The extant literature relevant to the tertiary prevention of major depression contains a paradox. The literature tells us that major depression is often a recurrent and even lifelong affliction. However, the majority of treatment research compares the efficacy or effectiveness of one or several treatments over relatively brief periods (usually less than three months). This important matter has received insufficient attention.

There have been, however, several useful studies. Chief among them have been the Maintenance Therapies in Recurrent Depression Protocol (Frank et al 1990; Frank et al 1991; Kupfer et al 1992). Key findings from these papers are the utility of maintaining individuals with recurrent depression on ‘full’ dosage antidepressant treatment (ie, the amount of medication used to treat the index episode). In addition, there was some evidence to support the use of a specific form of maintenance psychotherapy. These findings have been extended to depression occurring in late life (Reynolds et al 1994).

Despite these studies, there are insufficient data to guide clinicians in the management of a number of important clinical issues. For example, there are limited data concerning the choice of a second treatment when the initial treatment fails; who should and should not be offered maintenance treatment; and which personality and environmental variables might be altered to improve outcome.
Several excellent reviews of the treatment of major depression have been published in Australasia (Andrews 1983; Joyce et al 1995) and in the US (Depression Guideline Panel 1993a, 1993b).

**BIPOLAR DISORDER**

**DEFINITION OF BIPOLAR DISORDER**

Much of what we currently know about bipolar disorder (previously termed manic-depressive disorder or manic-depressive psychosis), systematically and empirically validates astute observations of clinicians from previous generations. In 1921, Emil Kraepelin in *Manic-Depressive Insanity and Paranoia* proposed an understanding of affective illness which has to some extent been upheld in the current diagnostic nomenclature of affective illness. Regarding aetiology, however, there is debate as to whether a single process underlies the various forms of affective illness. From Kraepelin’s time forward until the late 1950s, disorders as varied as ‘circular insanity’, melancholia, and mania were considered to exist under the general rubric of manic-depressive insanity. Based on genetic and personality observations, Leonard (1957; Leonard et al 1962) proposed that a distinction be made between bipolar disorder, in which both depressive and manic episodes are experienced, and monopolar disorder, consisting of either recurrent depressive or recurrent manic episodes.

In the current diagnostic classification, the *DSM-IV* (APA 1994a) upholds the unipolar/bipolar distinction, but brings recurrent mania under the heading of bipolar disorder. The important features of mania are abnormally and persistently elevated, expansive or irritable mood lasting at least one week, with inflated self-esteem, decreased need for sleep, pressure to talk, racing thoughts, distractibility, increased levels of activity, and excessive involvement in pleasurable activities with a high potential for painful consequences, such as sexual indiscretion or unwise business investment.

The hallmark of bipolar I disorder is the presence of at least one manic episode. Although the prototype of mania is often considered to include euphoric mood, expansiveness and grandiosity, this is not the only clinical picture of the manic state. Alternative presentations include irritability, rather than euphoria, and mixed affective states. Mixed states (or dysphoric mania) characterise between 5 and 70 percent of individuals with bipolar I disorder with the mean being approximately one-third of bipolar patients (for review, see McElroy et al 1992).

In addition, ‘a spectrum of ambulatory states alternating with milder, short-lived periods of hypomania’ has been labelled bipolar II (Akiskal 1995). Hypomanic states are defined as persistently elevated, expansive or irritable moods lasting at least four days. Additional symptoms are required, as in mania, and the symptoms must represent an unequivocal change from normal functioning that is observable by others. The exact nature of bipolar II disorder remains controversial. It has been questioned whether it represents an independent disorder or a milder form of bipolar I disorder, and whether individuals with bipolar II disorder are more closely akin to those with bipolar I or unipolar depressive disorders. Where sufficient data exist to clarify the distinction between bipolar I and II, the disorders are considered separately below.
Epidemiology of Bipolar Disorders

Lifetime Prevalence

The lifetime prevalence in community samples is 0.4–1.6 percent for bipolar I disorder and 0.5 percent for bipolar II disorder (APA 1994a). Christchurch data suggest that the lifetime prevalence for a manic episode for individuals between the ages of 18–64 years is 0.7 percent with no significant difference between the prevalence for males (0.5 percent) and females (0.9 percent) (Wells JE et al 1989a). This differs markedly from the 2:1 female: male ratio commonly observed in studies of major depressive disorder. Bipolar II disorder may however be more common in women (APA 1994a). A separate study in Christchurch (n = 1028) found the lifetime prevalence of bipolar I and II to be 1.3 percent (A Beautrais, P Joyce, R Mulder, personal communication, June 1996).

Natural History: Bipolar I

Bipolar I disorder is an episodic and recurrent disorder that often runs a chronic course and requires prophylactic treatment (Angst et al 1978; Post et al 1981). The average age of onset for bipolar I disorder is between the mid-twenties and mid-thirties (Perris 1982; Endicott et al 1985; APA 1994a). There is some suggestion that first presentation differs by sex, with more males presenting with an initial manic episode and more females with an initial major depressive episode. Of the individuals who present with a manic episode, the majority will experience future affective episodes (APA 1994a). In terms of the relation between manic and depressive episodes, the pattern can vary across individuals and within an individual over time. About 65–75 percent of manic episodes occur immediately prior to or following a major depressive episode (APA 1994a). There is some suggestion that, with age, interepisode length decreases (Angst et al 1978; Roy-Byrne et al 1985; Goodwin and Jamison 1990), severity increases (Post et al 1981), and duration of hospitalisation for an episode increases (Keck et al 1995). Between 10 and 15 percent of individuals with bipolar I disorder display a clinical course characterised by ‘rapid cycling’, defined as four or more affective episodes (major depressive, manic, or hypomanic) in a 12-month period. Individuals with a rapid cycling pattern may respond less well to treatment (Dunner et al 1976; Misra and Burns 1977; Goodnick et al 1987). Rarely, individuals with bipolar I disorder display very rapid cycling, experiencing switches from depression to mania in a 48-hour period (Bunney et al 1965; Jenner et al 1968). Season, treatment with tricyclic antidepressants, and sleep deprivation (eg, travel and the post-partum period) may trigger manic episodes in bipolar individuals.

Natural History: Bipolar II

Endicott and colleagues (1985) suggested that the average age of onset of bipolar II is similar to bipolar I and younger than unipolar disorder, and that bipolar II and unipolar individuals display greater chronic affective symptoms than bipolar I individuals. Angst (1986) suggested that bipolar II individuals have earlier age of onset, longer duration of illness, shorter cycles, more episodes, and poorer recovery than individuals with unipolar disorders. These results were not replicated by Kupfer (Kupfer et al 1988) who only found greater suicide attempts and greater psychomotor retardation in bipolar II individuals in comparison to unipolars. The selection criteria for inclusion in the aforementioned studies may have contributed to the differences observed.
Mortality: Bipolar I

Suicide is a serious risk for individuals with bipolar I disorder. The risk of suicide may be somewhat lower in the short term in bipolar I than unipolar individuals (Perris and D’Elia 1966; Angst et al 1979; McGlashan 1984; Weeke and Vaeth 1986) – possibly due to the lower percentage of time spent depressed. In the long term, suicide rates are approximately equal (Tsuang 1978; Black et al 1987b).

Mortality: Bipolar II

The risk of suicide in individuals with bipolar II may be higher than in individuals with bipolar I and unipolar disorder (Dunner et al 1976; Endicott et al 1985; Kupfer et al 1988; Bulik et al 1990; Rihmer et al 1990). It is possible that this elevated suicide risk could be secondary to comorbid psychopathology and diagnostic overlap with alcohol dependence and personality disorders (Rihmer et al 1990), or that individuals with frank bipolar I are more likely to receive adequate treatment because of the intensity of their manic episodes. Indeed, hypomanic episodes are often seen by the individual as enjoyable and unworthy of intervention.

Morbidity: Bipolar I

Considerable morbidity is associated with bipolar I disorder. Although many individuals display no interepisode functional impairment, approximately 20–30 percent of patients display social or occupational impairment at follow-up (Coryell et al 1987). The excesses of behaviour during the manic episode (eg, excessive spending, indiscrete sexuality, and gambling) can continue to impact on the individual and the family long after the temporal confines of the manic episode. Considerable burden can also be seen in family members who are confronted with the extreme oscillations in their loved one’s mood and behaviour.

Psychiatric Comorbidity: Bipolar I

Approximately two-thirds of individuals with bipolar I disorder display at least one Axis I comorbid condition (other psychiatric disorder) (Keck et al 1995). Frequent comorbidity with alcohol or other substance use disorders, anxiety disorders, and impulse control disorders has been noted.

Psychiatric Comorbidity: Bipolar II

Individuals with bipolar II disorder have been noted to have higher comorbidity of alcoholism, personality disorders, premenstrual dysphoria (in women) and antisocial personality disorder (in men) than bipolar I or unipolar patients (Endicott et al 1985). In addition, a history of childhood hyperactivity has been noted to occur frequently in individuals with bipolar II disorder (Endicott et al 1985).

Aetiology of Bipolar I and II Disorders

Genetic Factors

Numerous clinical and genetic investigations have examined the degree to which bipolar illness is heritable and distinguishable from unipolar illness on the basis of family, twin, adoption, and linkage studies. A number of family studies have indicated a higher frequency of affective disorders in the
families of bipolar individuals than unipolar patients (Dupue and Monroe 1978), with a higher prevalence of bipolar illness in the relatives of bipolar patients (Angst 1966; Perris and D'Elia 1966; Winokur and Tanna 1969). Unipolar disorder is more commonly found among relatives of bipolars than relatives of unipolars, yet bipolar disorders are infrequently seen among relatives of clearly diagnosed unipolar depressives (Perris and D’Elia 1966; Gershon et al 1971; Loranger 1975; Gershon and Hamovit 1979). Although unipolar family histories appear to be relatively pure for unipolar disorder, the family pedigrees of bipolar patients indicate a considerable overlap of unipolar and bipolar pathology (Gershon and Hamovit 1979; Mendlewicz 1979).

Twin and adoption studies have yielded more specific information on the transmission of affective disorders. Investigations of monozygotic and dizygotic twins have consistently shown a significantly higher concordance rate for manic-depressive illness in monozygotic than dizygotic twins (Harvald and Hauge 1965; Kringlen 1967; Zerbin-Rudin 1967). More specifically, Allen (1976) has shown significantly different rates of concordance between twins diagnosed with unipolar depression (40 percent) and with bipolar I disorder (72 percent). These rates were much lower among dizygotic twins (11 percent for unipolar and 14 percent for bipolar; that is, very similar rates). A famous adoption study by Mendlewicz and Rainer (1977) identified the importance of genetic factors in the transmission of bipolar I disorder. Thus, it appears that bipolar I disorder is a relatively highly heritable condition and that as yet unknown genetic factors are of aetiological relevance.

**Linkage and Association Studies**

The genetic mapping of the genes that predispose to bipolar I disorder has attracted considerable research interest over the last 30 years, particularly as twin and adoption studies have suggested its high heritability. Unfortunately, a fair summary of these studies in the middle of 1996 is that there are no compelling or conclusive leads. In an excellent summary of these studies, Risch and Botstein (1996) note that ‘the recent history of genetic linkage studies for (bipolar disorder) is rivalled only by the course of the illness itself’ in that there has been intense excitement when a group publishes a positive genetic linkage followed inevitably by dysphoria when non-replications by other groups follow. The history of these studies strongly suggests that great caution is required when evaluating the molecular genetic studies of complex disorders (Lander and Schork 1994) such as bipolar disorder. However, it is the great hope of many in the field that bipolar disorder will one day yield its secrets.

**Prevention of Bipolar I Disorder**

**Primary Prevention**

Similar to major depressive disorder, we are aware of no studies that address primary prevention of bipolar disorder in the general population. Much of what we wrote above about the primary prevention of major depression also holds for bipolar disorder.

**Secondary Prevention**

Any of the secondary preventive approaches for bipolar disorder parallel those outlined for major depression. There are, however, additional facets to secondary prevention of bipolar disorder which include predicting bipolarity in individuals who present with the first affective episode.
Approximately 10–19 percent of depressed adolescents will go on to develop bipolar I disorder (APA 1994a; Rao et al 1995). Individuals with bipolar I disorder tend to display a greater family history of bipolar disorder than individuals with major depressive disorder (odds ratio of 3.8) (Winokur et al 1993). Similarly, elevated rates of hypomania have been detected in family members of those with bipolar II disorder (Endicott et al 1985). When depressed adolescents present for treatment, careful screening for a family history of bipolar disorder or hypomania and ongoing vigilance for the presence of mixed affective states can serve as early warnings for the possible later development of bipolar disorder. These observations could, obviously, influence the clinician’s choice of pharmacological therapy.

**Tertiary Prevention**

According to the Consensus Development Panel (1985), the definition of relapse is ‘the exacerbation of an ongoing episode after an initial suppression of symptoms’ and the definition of recurrence is ‘a new episode following a complete recovery that has lasted for at least several months’. Preventive treatment refers to long-term efforts to prevent completely or reduce the intensity and frequency of recurrence – which we refer to here as tertiary prevention.

Additional effort should be made to improve tertiary prevention of bipolar I disorder. Although lithium has proven to be an invaluable tool in the treatment of this disorder (Angst et al 1970; Bastrup et al 1970; Coppen et al 1971; Hullin et al 1972; Prien et al 1984), a 20–30 percent failure rate of long-term treatment efficacy has been demonstrated (O’Connell et al 1985). Rapid cycling, medication non-compliance, the presence of mood-incongruent psychotic features (Miklowitz 1992), frequent previous hospitalisations, poor social supports, lower social class, current alcohol or drug abuse (O’Connell et al 1985; O’Connell et al 1991), and high familial expressed emotion (Miklowitz et al 1988), have all been associated with treatment failure or increased risk of recurrence. These findings have not been replicated by all investigators (eg, Carlson and Goodwin 1973; Carlson et al 1977; Roy-Byrne et al 1985). Rapid discontinuation of lithium has been associated with an increased risk of recurrence (Faedda et al 1993).

In addition, claims have been made that carbamazapine is of equal efficacy to lithium in the prophylactic management of mania (Post et al 1986, 1991); however, a meta-analysis by Dardennes and colleagues (1995) suggests that this statement is premature and that the data are not yet sufficient to establish therapeutic equivalence. Reviews of pharmacological interventions for bipolar disorder are available (Prien and Gelenberg 1989).

Bauer and Keller (1994) highlight the ‘efficacy effectiveness gap’ in the treatment of bipolar disorder, wherein the success rates seen in randomised clinical trials (‘efficacy’) are not equivalent to the results seen in general clinical practice (‘effectiveness’). Indeed, in a review of the Massachusetts General Hospital psychopharmacological database of all patients being treated for bipolar I or II disorder in a one-year open trial, although 90 percent of the sample met criteria for remission during the course of the year, only 4 percent of patients experienced an episode-free year (Sachs et al 1994).

Bauer and Keller (1994) highlight the importance of delivering high-intensity pharmacotherapy with as few side-effects as possible, efforts to improve medication adherence, patient education, and illness-management skills to improve outcome in general practice.
Psychological interventions such as cognitive-behavioural therapy aimed specifically at developing tools for illness management, such as ways of living with a chronic, recurrent illness, recognising early warning signs of affective episodes, and assisting with adherence to medication are worthy of further investigation. It is also worthwhile to determine whether other focal psychotherapies (eg, interpersonal psychotherapy (Klerman et al 1984)) can assist with decreasing the risk of relapse in individuals with bipolar I disorder (E. Frank, personal communication, June 1992).

Secondary and Tertiary Prevention: Reviews

Excellent reviews of the treatment of bipolar disorder have been published in Australasia (Andrews 1983; Joyce et al 1995) and in the US (APA 1994b).

CONCLUSIONS AND RECOMMENDATIONS

Many authors have criticised the paucity of attention given to primary prevention in psychiatry and, indeed, to the two cardinal mood disorders. Upon closer inspection, however, this matter is considerably more complex. As reviewed above, we have but a slight inkling of what is likely to be a complex causal aetiological chain that results in major depression and bipolar disorder. Until the causes of these conditions are better understood, primary preventive efforts are premature. In contrast, secondary and tertiary preventive efforts for major depression and bipolar disorder are probably feasible. It is critical for any such programmes to be tested empirically prior to more general implementation.

We close with the following recommendations:

1. Primary prevention of major depression and bipolar disorder is not currently possible. Further rigorous investigation of the effectiveness of possible intervention is necessary.

2. The inability to provide primary prevention is in part because the aetiology of major depression and bipolar disorder is not understood. Studies employing the appropriate scientific methodology to clarify the aetiology of these disorders would be useful. We suggest that attempting to elucidate the genetic basis of these conditions would be particularly useful and, should this be accomplished, the relevant environmental factors could then be delineated.

3. New Zealand data concerning the treatment of major depression and bipolar disorder (secondary and tertiary prevention) are needed. In particular, we believe that studies of the detection and treatment of major depression in the general practice setting and of the long-term treatment of major depression and bipolar disorder in the specialist setting are required.

4. Data about mood disorders in Māori and Pacific people are essentially non-existent. We know little about their prevalence in these population groups nor of the impact of culture upon their detection and treatment. Studies employing careful empirical methods to correct these deficiencies are essential.
REFERENCES


