2 Profile of depression in Australia

This chapter gives a profile of depression in Australia. The major systems used to diagnose depression are described and definitions for the terms used throughout the report to refer to disorders are presented. Disorders that are strongly associated with depression and the links between depression and the other NHPA are also covered. The course of depression across the lifespan is outlined along with risk and protective factors, including those that are more common to specific population groups. The impact of depression on the community is considered before concluding with an examination of depression research in Australia.

2.1 Definitions and diagnosis of depression

The term depression is used in many different ways: to describe transient states of low mood experienced by all people at some time in their life through to severe psychiatric disorders. Depression is understood to be a condition that generally comes and goes, that is more likely at certain stages of the life cycle, and with some types driven by genetic and biological factors and other types being more a response to major life events.

The clinical diagnosis of depression is made on the basis of the existence of a collection of signs and symptoms, also called a syndrome. Currently, the most widely used classification systems for depressive disorders are the *Diagnostic and Statistical Manual of Mental Disorders, fourth edition* (DSM-IV) (APA 1994) and the *International Classification of Diseases, tenth edition* (ICD-10) (WHO 1992), which has replaced ICD-9. The DSM-IV system underpins much clinical practice in Australia and is both a dimensional and categorical subtyping system. It allows a continuum of severity, but also includes three major depression subtypes:

- mild, moderate or severe major depression without psychotic symptoms;
- severe major depression with psychotic symptoms; and
- melancholia.

The ICD-10 system forms the basis of much research and international comparisons. It subdivides depression along a severity continuum into:

- mild;
- moderate; and
- severe, with or without psychotic features.

Depressive symptoms can be measured in the community and in research populations by a number of self-report inventories and checklists such as the Beck Depression Inventory (Beck & Steer 1987), the Centre for Epidemiologic Studies Depression Scale (Radloff 1977), and the Self-Rating Depression Scale (Zung 1965). Specialised scales are available for measuring depressive symptoms in children (eg Children's Depression Scale, Lang & Tisher 1983) and older adults (eg Geriatric Depression Scale, Yesavage et al 1983).

Profile of depression in Australia

To ensure clarity and consistency throughout the report, the following definitions of depression have been adopted. While these definitions generally reflect current clinical and research practice, they are not prescriptive. Clinical diagnostic systems such as the DSM-IV and ICD-10 provide more detail regarding appropriate diagnoses and definitions of disorders.

A *depressed mood* is ubiquitous, common, and generally lasts minutes to days. The individual feels 'down', hopeless, helpless, pessimistic, self-critical, and has lowered self-esteem. Such moods may be quite severe, but by themselves are generally brief.

DSM-IV *depressive symptoms* are listed in Box 2.1, and represent a reasonable list of common features. People may have depressive symptoms but not meet the criteria for one of the depressive disorders, where certain numbers of symptoms are stipulated. An 'intermediate' condition between depressed mood states and depressive disorders and where there is the presence of depressive symptoms, has been termed subsyndromal or subclinical depression.

Recent studies have shown that the presence of depressive symptoms may predict and follow episodes of major depression, continue between episodes, and form a spectrum of depressive psychopathology (Judd et al 1997). When depressive symptoms are included in depression prevalence studies, rates are not only high, but depression patterns appear also to be more chronic. Depressive symptoms are also associated with an increased risk for suicide attempts (Howarth et al 1992).

Box 2.1: DSM-IV major depressive symptoms

1. *Depressed mood most of the day.*
2. *Loss of interest or pleasure (in all or most activities, most of the day).*
3. *Large increases or decreases in appetite (significant weight loss or gain).*
4. *Insomnia or excessive sleeping (hypersomnia).*
5. *Restlessness as evident by hand wringing and similar other activities (psychomotor agitation) or slowness of movement (psychomotor retardation).*
6. *Fatigue or loss of energy.*
7. *Feelings of worthlessness, or excessive or inappropriate guilt.*
8. *Diminished ability to concentrate or indecisiveness.*
9. *Recurrent thoughts of death or suicide.*

A *major depressive disorder* is characterised by episodes of more persistent and pervasive disturbances in mood and accompanying features. It is formally diagnosed by the presence of at least five out of the nine symptoms listed in Box 2.1, including depressed mood and loss of interest or pleasure for most of the time over the past two weeks. Over time, the person may also withdraw from social contact and show impairment in performing usual social roles.

Major depressive disorder is generally categorised into *bipolar* and *unipolar* subtypes, a distinction based on the different courses of the disorders and indicating differing approaches to treatment.

A *bipolar disorder* typically involves a longitudinal pattern of manic or hypomanic episodes, usually interspersed with depressive states. Manic episodes are hedonic states, with the individual exhibiting confidence, optimism, grandiosity, increased energy, talkativeness, increased libido, and being drawn to pleasurable activities. Hypomanic symptoms show an above-normal elevation of mood that is not as extreme as mania. Such states can also be accompanied by psychotic symptoms, such as delusions and hallucinations, where the person is seriously out of contact with reality.

Unipolar disorders represent a larger residual group of disorders where an individual experiences depressive episodes only. A group of Australian researchers (Parker & Hadzi-Pavlovic 1996) has produced findings that support clinical distinction between two depressive subtypes:

1. *melancholic*, or *endogenous*, *depression* is associated with specific clinical features, particularly disturbance of psychomotor function. Although melancholic depression is rare in the community, it is an important condition in specialist treatment settings, as it responds best to physical treatments such as antidepressant drugs and electroconvulsive therapy; and

2. a *residual*, quite heterogeneous group of disorders, including 'reactive depression', 'adjustment disorder with depressed mood', and depressions secondary to anxiety and personality style. It also includes DSM-IV disorders such as *dysthymia* and *cyclothymia*. Both of the latter are characterised by either fewer depressive symptoms, or less severe expression of depressive symptoms, than the major depressive disorders, but the symptoms are persistent, lasting two or more years.

Postnatal depression describes the expression of depression associated with childbirth and post-partum mood disorder. These include brief episodes of depressed mood, major depressive disorder, and post-partum psychosis in which psychotic symptoms are also present.

Other disorders discussed throughout the report that require clarification are:

Affective disorders, or *mood disorders*, are terms that can be used to describe all those disorders that are characterised by mood disturbance. Disturbances can be in the direction of elevated expansive emotional state or, in the opposite direction, a depressed emotional state.

Seasonal affective disorder is a subtype of mood disorder where there is a seasonal pattern of mood variation. There is a regular pattern of onset and remission of depressive symptoms and episodes, which usually have onset in autumn/winter and remission in spring/summer. The symptoms of seasonal affective disorder are atypical of depression, often comprising hypersomnia, carbohydrate craving, as well as increased appetite and weight gain.

Anxiety is an unpleasant feeling of fear and apprehension accompanied by increased physiological arousal. *Anxiety disorders* are those in which fear or tension is the primary disturbance, and include *phobic disorders*, *panic disorder*, *generalised anxiety disorder*, *obsessive disorder* and *post-traumatic stress disorder*.

2.2 Risk and protective factors

The aetiology of depression is complex and multifactorial and requires a biopsychosocial model that takes into account the interaction of many diverse factors. Biochemical changes in the brain give rise to the major symptoms of depression (Nemeroff 1998), while the precipitants of these changes are often psychosocial. Genetic factors increase vulnerability to depression, but the specific genes involved are not known.

It is difficult for research to determine what risk and protective factors are paramount, because depression appears to be affected by such a wide variety of factors, from those at the biological level of the individual to social structural factors such as unemployment and low socioeconomic status. For many risk and protective factors, supporting research evidence is lacking or unclear. The experience of multiple risk factors may also greatly increase vulnerability, and, as a consequence, it is thought that specific population groups, such as refugees and Aboriginal peoples, are at increased risk of depression; however, supporting research evidence is needed. The following text, while not a review of risk and protective factors, presents some of the factors widely held to be important. Box 2.2 summarises the main risk and protective factors.

Box 2.2: Summary of risk and protective factors associated with depression

Risk factors	Protective factors
Environmental and social	
Social disadvantage (eg poverty, unemployment)	*Good interpersonal relationships (eg supportive relationship with at least one person/parent, perceived social support)*
Family discord (eg relationship break-up, conflict, poor parenting practices)	*Family cohesion (eg positive parent-child relations)*
Parental mental illness	*Social connectedness*
Child abuse (eg physical and sexual abuse, neglect)	*Academic/sporting achievements*
Exposure to adverse life events (eg bereavements, family separation, trauma, family illness)	
Caring for someone with a chronic physical or mental disorder	
For older adults, being in residential care	
Biological and psychological	
Parental mental disorder and family history of depression	*Easy-going temperament*
Being a female adolescent	*Optimistic thought patterns*
High trait anxiety and pre-existing anxiety disorders, substance misuse, conduct disorder	*Effective coping skills repertoire (eg social skills, problem-solving skills)*
Temperament—reacting negatively to stressors, and personality trait of neuroticism	
Negative thought patterns (pessimism, learned helplessness)	
Avoidant coping style	

Adverse environmental influences that may increase vulnerability to depression across the lifespan include recent life events and experiences of loss and failure such as: bereavement; relationship break-up; school failure; social isolation; lack of social connectedness or sense of community ties; socioeconomic factors such as poverty and unemployment (particularly long-term unemployment) (Winefield 1995; Rutter 1985, 1987; Schofield & Bloch 1998) and caring for a person with chronic physical or mental disorder. Negative thoughts and evaluations of the self are considered to be significant psychological factors that trigger depression (Beck 1967). Lack of effective coping responses to life's problems is also likely to contribute to the onset of depressive disorder, particularly if the preferred coping style is to avoid problem solving and reinforces feelings of helplessness and failure (Folkman et al 1986, Peterson et al 1993).

Protective factors across the lifespan include having an easy-going temperament and good perceived social support, especially having a relationship with a supportive adult (Werner 1992). A coping style that favours problem solving is also protective (Folkman et al 1986).

Major risk factors contributing to vulnerability in childhood, as well as later in life, include one or both parents suffering from depression (particularly the mother), the loss of a caring parent, physical abuse, neglect, and sexual abuse (Perry et al 1995, NHMRC 1997). For disorders in childhood, family influence is paramount. Poor parenting practices (such as a cold, controlling and affectionless parenting style), severe marital discord, divorce, and other family disruptions are potential risk factors (Rey 1995). Parenting is usually conceptualised as a two-fold process, involving contributions from both parent and child. Although it had previously been mainly viewed as a complex environmental factor, Kendler's (1996) large multi-generational study of female twins has provided more evidence that genetic factors in both parent and child contribute to parenting style and interactions, at least in females. Behavioural problems in childhood are also a known risk factor for depression (Angold & Costello 1993). The negative effects of loss or abuse in childhood can be mitigated, however, by experiences of academic or sporting achievement in adolescence, as well as by a supportive relationship outside the home and good interpersonal relationships (Rutter 1985, 1987; Luthar & Ziegler 1991).

Risk and protective factors in adolescence have been reviewed to produce the *NHMRC Clinical Guidelines for Depression in Young People* (NHMRC 1997). Confirmed risk factors for depression in adolescence are symptoms of anxiety, conduct disorder or substance use disorder; being female; being an older adolescent; having a depressed parent; and having a previous history of depressive disorder or symptoms. Probable risk factors are having a close biological relative with depression and exposure to stressful life events. Possible risk factors that need further investigation are: poor self-esteem or vulnerability because of negative thinking; neuroticism or vulnerable personality; parental divorce or conflict; uncaring or over-controlling parental style in childhood; early childhood physical or sexual abuse; being of Aboriginal or Torres Strait Islander descent[3]; residing in rural areas; having sleep dysfunction; low socioeconomic status; poor peer relationships; decreasing school performance; having co-existing medical problems; being homeless or in custody; having learning difficulties; prior history of suicide attempt; being a refugee; hormonal changes during puberty; and parental

[3] According to the *Bringing them Home* (1996) report, family separation policies have caused Aboriginal and Torres Strait Islander parenting skills to be undermined, leading directly to risks for children and future generations.

death. Possible protective factors needing further investigation are good peer relationships, good relationships with parents, and being employed. In families where a parent has a depressive disorder, resilience in adolescence is associated with having a good understanding of the parent's illness, good interpersonal relationships, and a strong sense of self (Beardslee & Podorefsky 1988).

For adults, having a temperamental style with a propensity to react negatively to environmental stressors is a major risk factor for depression (Rutter 1997). Negative thought patterns, such as learned pessimism or helplessness, are further risk factors, while an optimistic thought pattern is likely to be a protective trait (Peterson et al 1993, Seligman 1975). For older adults, factors associated with being placed in residential care contribute to the development of depressive disorder (Ames 1993).

2.3 Depression and related disorders

Depressive disorder is associated with high rates of comorbidity (ABS 1998; Kessler et al 1994, 1996). When depression occurs with another disorder, it is likely to cause more disability than when it occurs alone (Sartorius et al 1996). The frequent occurrence of mixed patterns of disorder is important for recognition and management in primary care and other settings.

Depression and anxiety disorders

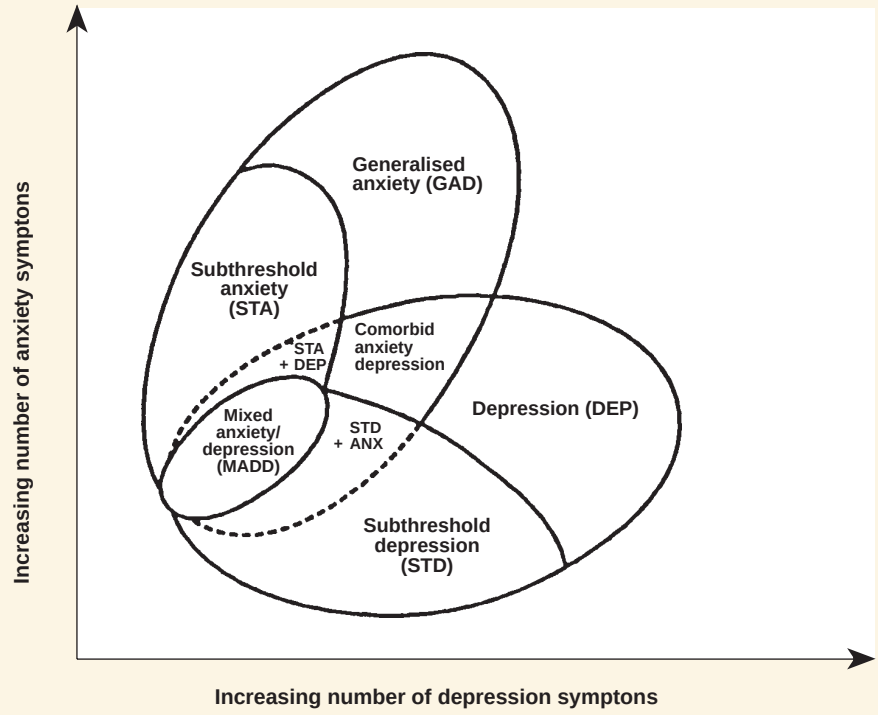
Depression is more likely to be associated with anxiety than any other disorder. Just over half the people with an affective disorder or depressive disorder also reported an anxiety disorder in the 1997 SMHWB. The comorbidity of depression and anxiety is evident in both primary care and specialised mental health settings.

In an Australian primary care study, it has been found that 36 per cent of those attending primary care settings have symptoms of psychological disorder and 20.5 per cent report both anxiety and depressive disorders (Harris et al 1996). Figure 2.1 shows that comorbidity between major depressive disorder and anxiety occurs in nearly half the people with an affective disorder, as determined by a WHO study of primary care at 15 diverse international sites (Sartorius et al 1996). Symptoms of both depression and anxiety are found in nine per cent of those who, even though they were below the threshold of symptoms to meet diagnostic criteria, nevertheless, had clinically significant symptoms and functional impairment (Harris et al 1996).

When the comorbidity of anxiety and depression is examined in greater depth, as in the US National Comorbidity Study (Kessler et al 1996), it is found that anxiety disorders frequently precedes major depressive disorder and the co-occurrence of both is common. The occurrence of an anxiety disorder also predicts subsequent risk of a major depressive disorder. The authors suggest that reported increases in the prevalence of depression may be attributable to increases in depression that is secondary to an anxiety disorder, and that such depressive disorders are likely to be more severe.

Local studies confirms these international findings, both for the general community (Parker et al 1997b) and for people being treated in a hospital setting for depression (Parker et al 1998). These studies indicate that a first episode of

Figure 2.1: Comorbidity of depression and anxiety in primary care



Source: Satorius et al (1996).

unipolar major depressive disorder is more likely in people who have a primary anxiety disorder, with generalised anxiety disorder, panic disorder and social phobia being the most likely pre-existing conditions. A pre-existing anxiety disorder also increases the likelihood of longer and more frequent episodes of depression. The finding that certain expressions of anxiety increase the chance of depression occurring and also prolong episodes suggests a key point of intervention and treatment—specifically, persons with a pre-existing anxiety condition may benefit more from having the anxiety treated than by treating only the depressive disorder.

Depression and health-related risk behaviour

While depression is more likely to co-occur with anxiety disorders, it might also do so with a range of health risk behaviours (Sartorius et al 1996). Depression has links with health risk states which include tobacco use, illicit drug use, alcohol misuse and dependence, eating disorder and obesity.

Smoking has been shown in a number of epidemiological studies to be associated with either depressive symptoms or mixed anxiety and depression. One longitudinal study reports that adolescent depression may predict subsequent smoking in adulthood (Kandel & Davies 1986) and an Australian study has shown an association between smoking and both depression and anxiety in teenagers (Patton et al 1996b, Patton et al 1998). Furthermore, depressed smokers are less likely to give up smoking than those who are not depressed and may have taken up smoking to self-medicate their depressive symptoms (Anda et al 1990). While causation is not established, there are a number of common risk factors for both

depression and smoking, including alcohol use, the personality trait of neuroticism, exposure to adversity, and socioeconomic disadvantage (Jorm et al 1999b).

The 1997 SMHWB found that nearly one in four people who had an affective or depressive disorder also had a related substance use disorder. However, the likelihood of depression being comorbid with alcohol dependence was half the likelihood of it being comorbid with anxiety. There is accumulating evidence that depression predicts progression to both alcohol misuse and dependence in younger drinkers (Nelson et al 1996). Similarly, in dieters, it predicts the development of symptoms of bulimia nervosa (Patton et al 1999).

Depression and physical illness

Depression is also common in people with physical illnesses. The 1997 SMHWB found that nearly half the people who had an affective or depressive disorder also had a related physical problem. Conversely, for people with physical disorders the prevalence of depression may also be as high as 50 per cent (Lamberg 1996). Depression is a contributing factor particularly for people contacting medical services with symptoms of pain and fatigue, especially chronic fatigue (Simon & von Korff 1991, Hickie et al 1996).

Suffering from depression also may affect physical health; several types of immune dysfunctions have been reported in people suffering from depressive disorder (Seidel et al 1996).

The diagnosis of depression in people who are physically ill is not always evident. The indicators of depressed mood, such as loss of interest and fatigue, may be attributable to the physical condition or to current medications. This leads to difficulty in recognising depression or, alternatively, to over-identification of depression when the symptoms are linked to the physical illness.

Depression and other National Health Priority Areas

Cardiovascular health

A recent review highlights the considerable prevalence of depression in people diagnosed with cardiovascular disease, with rates ranging from 10–23 per cent for major depressive disorder (Musselman et al 1998). Depression has also been shown to predict future cardiac events in people with coronary artery disease. A study of persons treated for depression showed that these individuals are 2.3 times more likely to have a heart attack. Although the risk is also increased for smokers and those with high cholesterol, it was concluded that depression was the biggest contributor to heart attacks (Cohen 1997). Depression increases cardiovascular risk through a number of mechanisms that are both physical (such as autonomic dysregulation and platelet dysfunctioning promoting thrombosis) and psychological in nature (such as non-compliance with the treatment regime) (Shapiro et al 1997).

Depression also impacts negatively on survival following a cardiac event, and hastens mortality in the ensuing 18 months. A study examining the 18 months post myocardial infarct survival has found that people who are depressed have an almost fourfold risk of death compared to those not depressed (Frassure-Smith & Lesperance 1995). Depression has also, in prospective epidemiological studies and a large clinical study, been shown to independently predict mortality from cardiovascular disease (Musselman et al 1998).

If unrecognised and untreated, depression is known to adversely affect compliance with prescribed exercise therapy, as well as rehabilitation and recovery, in people recovering from myocardial infarction. Antidepressant medication, specifically selective serotonin reuptake inhibitors (SSRIs) (see Table 4.1), may play a role in treating depression in this population while also reducing the risk of thrombosis (Shapiro et al 1997).

Diabetes

There are relatively few recent studies examining the prevalence of depression in people with diabetes. Karlson and Agardh (1997) have found a moderate elevation of depressive symptoms in a group of people with insulin-dependent diabetes. The degree of depression is not related to disease severity, but to perceived daily burden of living with the disease. Rajala et al (1997) confirm that psychosocial factors related to the disease cause depression. Goodnick (1997) found that pre-existing depression has been associated with an increased risk of developing diabetes and comorbid depression is associated with poorer diabetic control. The nature of the association remains unclear, but is thought to have a biochemical basis. In terms of antidepressant treatment (see Table 4.1), the tricyclic antidepressants are associated with worsening diabetes control because of the predominant influence on noradrenalin, whereas the SSRIs are associated with significant reductions in blood glucose levels related to the predominant effect on serotonin over noradrenalin reuptake (Goodnick 1997).

Cancer

Spiegel (1996) reports that about half of all people under medical treatment for cancer have a psychiatric disorder, usually with depressive symptoms. However, depression in people with cancer is frequently under-diagnosed and under-treated (McDaniel et al 1995). The diagnosis of depression is clouded by symptoms such as loss of appetite, weight loss, insomnia, loss of energy and loss of interest, all of which may be secondary to either cancer or depression. Some cancers may cause an organically-based depressive disorder (such as central nervous involvement by tumor); some anticancer drugs (such as the corticosteroids, vinblastine, vincristine, and interferon) can also cause depression (Massie et al 1994).

Depressed individuals with cancer need a thorough medical, endocrinological and neurological assessment. Treatment of depression for people with cancer improves their depressed mood and other depressive symptoms, improves their quality of life and may also improve their immune function and survival time (McDaniel et al 1995).

Spiegel (1996) notes that while earlier studies reported people with depression at higher risk of developing cancer, later studies have not confirmed a predictive relationship. However, anxiety about the possibility of having cancer may delay seeking medical diagnosis and thereby reduce prospects of long-term survival by 10–20 per cent.

Injury

Suicide is the largest single cause of injury-related death in Australia (DHFS & AIHW 1998a). Depression is a factor commonly associated with suicide in all age groups; the majority of people who die from suicide meet criteria for depressive disorder in the weeks before death (Barraclough et al 1974, Shaffer et al 1996). After a previous suicide attempt, depression is the next highest risk factor for youth suicide (Zubrick & Silburn 1996).

Through its relationship with drug-related harm, which was noted earlier, depression is also indirectly related to injury. Alcohol misuse, in particular, has now been well documented as a contributing factor to the frequency and severity of injury (DHFS & AIHW 1998a). The influence of alcohol is particularly notable in suicide (Hayward et al 1992), interpersonal violence, road injury, injury to young males, drowning, sport and leisure injury, and occupational injury.

2.4 The course of depression across the lifespan

Depression is often a recurrent disorder and a whole of lifespan perspective helps to understand the onset and evolution of the disorder. It manifests somewhat differently at different ages, and is associated with varying precipitating factors.

Childhood

Depression and anxiety symptoms occur in about 4–6 per cent of children screened for mental health problems, with about half of these children sustaining significant functional impairments and being in need of treatment (Zubrick et al 1995, 1997, Silburn et al 1995, Moon et al 1998). Depression and anxiety in childhood are likely to persist with age, if not effectively treated (Offord & Bennett 1994). A follow-up of a group of 80 children and adolescents receiving psychiatric treatment for depressive disorders has reported a fourfold risk of subsequent major depressive disorder in adulthood (Harrington et al 1990).

The presence of *any* mental disorder in childhood increases the risk for a mental disorder in adult life. In a four-year follow-up of children in the Ontario Child Health Survey, over one-quarter (26 per cent) of 4–12 year olds with emotional disorders continued in the same category when they were aged 8–16 years. The British National Child Development Study on the other hand has reported that childhood conduct disorders were associated with a higher risk of depressive disorder at the age of 23 years and later (Rutter 1991).

The Dunedin (New Zealand) Longitudinal Study provides some data on the development of depression from pre-adolescence to young adulthood in members of a birth cohort (Anderson et al 1987, McGee et al 1990). According to this study, depressive disorders, in terms of clinical diagnosis, are relatively rare in childhood but increase in the pre-adolescent years when gender differences emerge. It is clear that some vulnerabilities, possibly related to the risk factors identified in Section 2.2, are established in childhood.

Adolescence

The first onset of major depressive disorder and dysthymia often occurs in mid-to-late adolescence and this is the life stage of peak incidence. Figure 2.2 reveals that most new cases develop between the ages of 15 and 18 years (Hankin et al 1998). This period may be a critical time for examining vulnerability to depression because of the high rates and high risk of onset and also the emergence of a gender difference, with females exhibiting more depression than males.

Around 20 per cent of young people in the community suffer from depressed mood, with up to 43 per cent reporting feeling sad for at least two weeks in the past year (Cubis 1994). Five per cent of young people suffer from a depressive disorder and the prevalence of current major depressive disorder is 2.7 per cent, a considerable rise from less than one per cent in pre-pubertal children (NHMRC 1997). Incidence rates are much higher than those for adults (Lewinsohn et al 1993, Garrison et al 1997, Eaton et al 1997, Giaconia et al 1994).

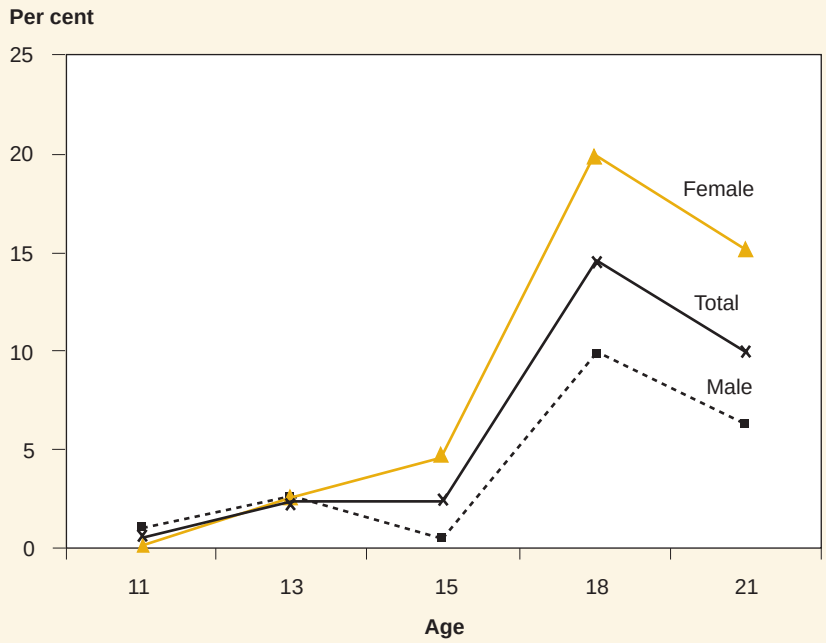
Community studies show that, for girls, there is a progressive rise in depressive symptoms from menarche, so that by the mid-teens girls exhibit at least twice the prevalence rate of males (Patton et al 1996a, Angold et al 1998). The cause of this striking rise in the incidence of depressive symptoms in adolescent females is as yet unknown, but hypotheses include the influence of female gonadal hormones, psychological changes that accompany puberty and changes in social roles. The rise appears to be the reason for the gender difference that persists through the reproductive years until the menopause (Kessler et al 1994). Jorm (1987) notes that the gender difference is not as evident in either childhood or old age.

Hunter (1992) states that Aboriginal children and young people experience at least the level of mental health problems evident in the general youth population, but are even further disadvantaged because they have less access to services. He also notes that some depressive symptoms may be reactions to disadvantage, racism and perceived oppression. There are, however, no epidemiological data comparing Aboriginal and Torres Strait Islander peoples with non-Indigenous populations.

Data on the natural history of adolescent depression are sparse, and while the majority of episodes appear to resolve spontaneously within around six months, the duration of adolescent depression varies considerably between individuals, from two weeks to many years. An episode is a risk factor for further episodes of disorder; the risks for subsequent episodes are increased sixfold for males and fourfold for females (Nolen-Hoeksema et al 1992). At least 50 per cent of adolescents who experience a depressive disorder subsequently suffer one or more occurrences (NHMRC 1997). These findings indicate the importance of effective intervention to ensure prevention of relapse in adolescents who have experienced an initial depressive disorder.

Table 2.1 presents the results from follow-up studies of a group of clinically referred 8–13 year olds for the first episode of major depressive disorder or dysthymia (Kovacs et al 1997). The authors report that the mean duration of first episode is likely to be up to four times longer in such clinically referred young people compared to young people in community settings. For dysthymia, however, mean duration could be equally long in both community and referred settings. Compared with major depressive disorder, dysthymia is more likely to be prolonged and to have comorbidity with behavioural disorders

Figure 2.2: Development of new cases of clinical depression, by age and sex



Source: Hankin et al (1998).

Table 2.1: Outcomes for a group of clinically referred 8–13 year olds for first episode of major depression or dysthymia

Indicator	Major depression	Dysthymia
Onset	7.75 to 14.01 years (mean 10.98 years)	5.14 – 12.8 years (mean 8.71 years)
Median duration	9 months	3.9 years
Recovery rates	86%	7%

Source: Kovacs et al (1997).

Bipolar depressive disorder commonly onsets in adolescence, but its recognition as bipolar disorder is generally delayed until the explicit emergence of hypomanic symptoms. In comparison with unipolar depressive disorder, bipolar depression is relatively uncommon, affecting under one per cent of adolescents (NHMRC 1997).

Adulthood

The onset of depressive disorders also occurs for the first time in early adult life, and in half of these cases there may be a prior, external stressor (Judd 1997). It is suggested that 20 per cent of cases meet criteria for diagnosis as a depressive disorder for the first time before the age of 25 years (with symptoms developing during the previous six years) and 50 per cent before the age of 39 years (with symptoms developing during the previous 10 years) (Mrazek & Haggerty 1994).

Prevalence rates for depressive disorder have been estimated as 3.4 per cent for men and 6.8 per cent for women, over a 12-month period, for Australian adults. The rates for dysthymia are considerably lower, at one per cent for men and 1.3 per cent

for women (ABS 1998). Lifetime estimates of unipolar depressive disorder are variable and have been particularly prone to methodological problems (Parker et al 1997a, Kessler et al 1994). Bipolar depressive disorders are estimated to have a lifetime prevalence in Australia of about 1.5 per cent (Parker et al 1997b). Depressed mood is much more common and ranges from 9–20 per cent for current prevalence (Boyd & Weissmann 1981).

According to the US National Comorbidity Study the lifetime prevalence of major depression with a seasonal pattern is 0.4 per cent and depression with a seasonal pattern is one per cent.

The duration of the first episode of major depressive disorder varies by gender, with an average of 181 weeks for males and 114 weeks for females (Simpson et al 1997). This study reports that while most people recover from their first episode with a median recovery time of three years, the majority will have at least one more episode of major depressive disorder in the following five years, with recurrence being the highest in earlier years.

Studies of remission from depression have generally taken place in clinical rather than community settings. A follow-up study of people treated for depressive disorder suggests that 50 per cent recover within six months, but in about 10 per cent of people the disorder has a chronic course, defined as being ill for a five-year period (Keller et al 1992). There is also a common pattern of depressive symptoms between episodes of major depression that may be recurrences of either the original depressive episode or a different form of depression. Predictors of persisting symptoms include the initial severity of symptoms, earlier age of onset, family history of depression, and inadequacy of treatment during the acute episode.

Between 30 and 50 per cent of people who initially recover from a depressive disorder, following treatment, relapse in the short term when maintenance treatments are not used (Shea et al 1992). Factors associated with relapse appear to include persisting depressive symptoms, number of previous episodes of the disorder, and psychological risk factors (see section 2.2).

Postnatal depression

The 'blues', or brief episodes of depressed mood and tearfulness, occur in 50–70 per cent of women within one to ten days of childbirth (NSW Health 1994). The presence of the 'blues' in the immediate post-partum period is related to the subsequent development of postnatal depression (Cooper & Murray 1998). Ten to 15 per cent of women will suffer a major depressive episode within the first 3–6 months after childbirth (O'Hara 1987). Post-partum psychosis affects about two women per thousand deliveries and the risk of recurrence in subsequent deliveries is very high (Boyce & Stubbs 1994).

Risk factors for a depressive disorder following childbirth are predominantly psychosocial, such as marital conflict, the absence of personal support from spouse, family and friends, difficulties with the infant (pre-term, reflux, physical problems) and stressful life events. A previous psychiatric history, especially a previous depressive episode, is also a risk factor, particularly if there are obstetric complications during delivery (Cooper & Murray 1998).

Depression in the older years

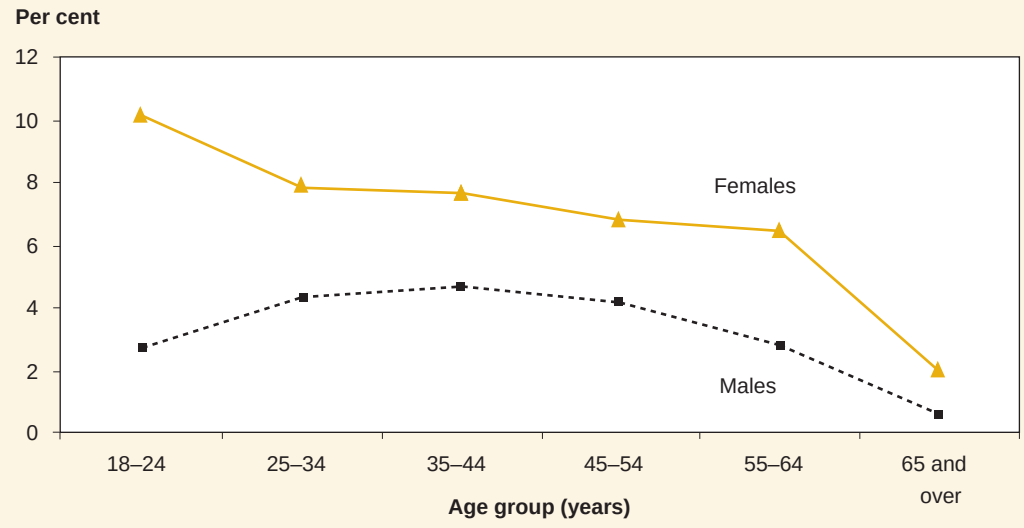
The prevalence of depressive disorders decreases with age among those who live in the community (Figure 2.3). However, some depressive episodes appear for the first time in later life and depression in older people has been shown to be more likely to persist if untreated. Depression in association with other disorders in older people may be a predictor of premature death (Henderson et al 1997, Ames et al 1988). Depression is often underdiagnosed in the older years (Snowdon 1998).

The prevalence of depressive symptoms is strongly related to living arrangements for older people (Phillips & Henderson 1991). Rates are lower among those who live in the community than among those in residential care. The 1997 SMHWB found that depressive disorder is experienced by less than one per cent of men and two per cent of women, aged 65 years and over, who were living in the community. Older people in residential care experience about twice the level of depressive symptoms as those in the community, and have a 20 times greater risk for major depressive disorders (Ames 1993). Between 15 and 42 per cent of residents in hostels and nursing homes experience a substantial level of depressive symptoms and between six and 18 per cent exhibit depressive disorders.

Depressive symptoms are also strongly related to physical health (Prince et al 1997a, Evans et al 1991) and loss of dear ones (Prince et al 1997b) in older people.

There is a link between depression and suicide at all ages. Traditionally, rates of suicide increased with age, particularly for men. Before the 1960s, rates among older men were high. However, over the past 100 years there has been a general reduction in the suicide rates among older people (Goldney & Harrison 1998). Currently, suicide rates tend to fall with age, except for men aged 80 and over, who continue to have the highest rates of all the age/sex groups (Figure 1.6).

Figure 2.3: Age-specific prevalence of depression, 1997



Source: 1997 National Survey of Mental Health and Wellbeing: Adult Component (ABS 1998).

2.5 Depression in specific population groups

The experience of depression varies across population groups. There are factors unique to Aboriginal peoples and Torres Strait Islanders, people from culturally and linguistically diverse backgrounds, rural and remote communities, defence services personnel and veterans, and children of parents with mental disorders that are of particular importance in terms of the expression, recognition and prevalence of depression.

Aboriginal peoples and Torres Strait Islanders

Depression, anxiety, substance use disorders, and high-risk behaviours are believed to be highly prevalent in Aboriginal and Torres Strait Islander communities (McKendrick et al 1992, Swan & Fagan 1991). It is clear from the work of McKendrick (1992, 1993, 1994) that a high proportion of people presenting to Aboriginal Medical Services have mental disorders, or are psychologically distressed. Studies have reported that more than 63 per cent of people in such settings have a significant level of distress, principally depression (Swan & Raphael 1995).

The *Ways Forward* report highlighted that the most significant and frequent problems identified by Aboriginal peoples and Torres Strait Islanders are grief, trauma and loss (Swan & Raphael 1995). These are identified in Section 2.2 of this report as risk factors for depression. Trauma, loss and grief derive from the history of invasion; the ongoing impact of colonisation; loss of land and culture; high rates of premature mortality; high levels of incarceration; high levels of family separations, particularly those involving the forced separation of children from parents; and Aboriginal deaths in custody. Domestic violence, sexual and physical abuse, and a whole range of other traumas also contribute (Swan & Raphael 1995). In studies of non-Indigenous communities, the extent of such traumatic separation, loss, abuse, dislocation, and dehumanisation can only be found in populations subjected to systematic torture, genocide, concentration camps, or urban or family violence.

Furthermore, the *Burdekin Report* highlighted that every Aboriginal witness discussed the long-term psychological effects that colonisation has had on Aboriginal peoples since 1788. 'As one witness said, "the pain and bitterness of these memories are passed on from generation to generation and results in feelings of hate, anger, frustration, grief, depression and alienation". These memories are reinforced by the continuing economic and social disadvantage experienced by Aboriginal peoples' (Human Rights and Equal Opportunity Commission 1993, p 693).

Some transcultural psychiatric surveys have suggested a low prevalence of anxiety and depression in Indigenous people. This often reflects the limitations of the observers. A key study by Morice (1978), who learned the Pintupi dialect, reported an extraordinarily rich description of terms to describe grief, depression, fear and anxiety among the Pintupi people. The glossary of terms ranged across varying grades of severity, from grief and disappointment through to loneliness and depression, and serious mood disorder.

People from culturally and linguistically diverse backgrounds

People from culturally and linguistically diverse backgrounds comprise a substantial proportion of the Australian population, increasing from just over 19 per cent in 1947 to more than 59 per cent in 1993 (Minas et al 1996). They are a diverse group from many different ethnic communities. Size, length of establishment in Australia, nature of migration experiences, English language competency, degree of assimilation and level of specific ethnic community support are factors which differentiate communities. Determining prevalence is complicated by such issues as sampling and also by cultural differences in the definition, conceptualisation, experience and reporting of depression.

The ABS has produced prevalence rates for affective disorders in people born in non-English-speaking countries showing that the rate is lower than for people born in Australia (ABS 1998). However, it should be noted that respondents with poor English-language competency were not included in the sample.

Refugees, who comprise 10 per cent of the annual migrant intake, are a distinct group with a very different profile to other migrants. In particular, they meet the humanitarian criteria of having suffered persecution and gross violation of human rights. Culturally sensitive individual assessments of torture survivors have shown that 80 per cent of those accepting a referral for rehabilitation have a high level of depressive symptoms (Victorian Foundation for Survivors of Torture 1997).

People living in rural and remote regions

There are limited comparative data available on depression in rural areas in Australia. However, suicide death rates for men living in rural areas have been consistently higher than for men living in urban areas, particularly for young men aged 15–24 years. Given the association between depression and suicide this may indicate increased levels of depressive symptoms for rural men (see Figure 1.8).

A major disadvantage experienced by people in rural and remote regions is the lack of access to health services, including specialised mental health services, for depression.

Veterans and defence services personnel

Veterans and defence services personnel are specifically noted here, not only because they are particularly at risk of depression, but also because they may be served by separate health and entitlement systems through the Department of Veterans Affairs.

Most studies have been of United States Veterans, particularly Vietnam Veterans. In an analysis of the national Vietnam Veterans Readjustment Study, Keane et al (1998) has shown that exposure to war zone stress contributed to the development of not only PTSD, but also a wide range of psychiatric disorders, including depression. According to this study, women veterans are particularly likely to react to these stressors with depressive symptoms, whereas men are more likely to develop PTSD and substance use disorders.

In an Australian sample, O'Toole et al (1996) have revealed a clear, close relationship between dysthymia and combat exposure. Veterans are also at

increased risk of depression through more generic risk factors, such as marital breakdown (DVA 1998).

Little research has been carried out on the mental health of veterans of World War II, although a study of former prisoners of war of the Japanese has found that they had very high rates of depression in the decades following the war compared to a control group of ex-servicemen (Dent in press).

Children of parents with a mental illness

Children who grow up with a parent who has a mental disorder are at a higher risk of depression either through increased genetic susceptibility to mood disorder or schizophrenia or because there may be gaps in parenting because of mental illness. Marriages where one partner has a mental disorder tend to have a higher rate of separation and divorce. If the parents do not separate, there are likely to be lengthy periods where one parent cares for the children while the other parent is in hospital (Goodwin & Jamison 1990).

Children of parents with a mental disorder report feelings of powerlessness to help the parent, feelings of abandonment and neglect while the parent is ill, and fears of becoming like the parent or developing a similar illness to the parent. It may take some time for the child to understand what is happening, and parents may be unwilling or unable to explain to children what depression is and what is happening to their mother or father. Depression in a parent can be experienced by children as rejection or something caused by the child's behaviour (Duke & Hochman 1992).

Children with younger siblings often take on increased family responsibility while the parent is ill or hospitalised. Mothers who are depressed and who have dependent children are often not hospitalised because of difficulties in finding care for the children or because the severity of the depression is not recognised. Few mental health facilities offer childcare or assistance with care of dependent children. Community support services for people with disabilities often do not cater for adults with a depressive disorder or other mental disorders who have dependent children. Consequently, many children live with a severely depressed parent who may be unable to adequately care for them. Adult children of parents who suffered from depression have reported that they received little or no counselling or information about what was happening to their parent(s) and that this lack of information and understanding added to their distress (Human Rights and Equal Opportunity Commission 1993). Children in this situation may find themselves taking responsibility for getting the parent into medical care or coping with the aftermath of an attempted suicide by the parent (Cronkite 1994).

2.6 Impact of depression

Depression causes a substantial burden of morbidity, disability and mortality. Some of these disability outcomes are summarised in Box 2.3. In direct monetary terms, it is estimated that in 1993–94, $521m was spent in health system costs associated with depression (Mathers 1998). However, the true burden in terms of health resources, personal suffering and detriment to quality of life, including stigma and possible discrimination, is not possible to quantify. Depression impacts not only on the individual, but also on their family, their friends and colleagues, and society in general.

Profile of depression in Australia

The 1997 SMHWB confirmed high rates of disability associated with depressive and other common mental disorders. Those with affective disorders (which include major depressive disorder and dysthymia) had close to three times the number of disability days of those who were well. This rose to six times the number of days of not fulfilling normal role obligations where affective disorders were associated with physical illness.

Box 2.3: Disability outcomes associated with depressive disorder

High levels of disability are reflected in:

- *impairment in work productivity;*
- *days lost from work;*
- *educational failure;*
- *poor family functioning;*
- *poor social functioning;*
- *diminished sense of wellbeing;*
- *utilisation of medical services; and*
- *visits to medical clinics.*

Even more, disability at a population level may be associated with depressive symptoms due to their high prevalence. With as few as two depressive symptoms, high levels of household strain, social irritability, financial strain, limitations in physical or job functioning, restricted activity days, bed days and poor health status are reported (Judd et al 1996). From a population, societal or 'service burden' perspective, more medical service utilisation, suicide attempts, and other indicators of impairment arise from individuals with depressive symptoms, because of their high prevalence in the community, than from individuals with major depressive disorders (Johnson et al 1992).

When depression co-occurs with any other psychological or physical condition, it produces more disability than if it occurs on its own. For example, one study has reported that depression alone causes occupational dysfunction in 39 per cent of people with that disorder, but if depression is comorbid with another disorder, the disability is present in up to 48 per cent of all such people (Sartorius et al 1996).

Another impact of depression can be illustrated in the relationship between suicide and depression. People suffering from depressive disorders have a risk of suicide 30 times that of the general population (Chipps et al 1995). The highest risk is experienced by males with a diagnosis of major depressive disorder who have been discharged from hospital in the last four weeks. Using estimates of population attributable risk, the elimination of depression and related disorders could reduce the incidence of serious suicide attempts by 80 per cent (Beautrais et al 1996).

2.7 Depression research in Australia

Australia has a significant history of research on depression. Notable early achievements were Cade's work on lithium as a treatment for bipolar disorder (Cade 1949) and Kiloh's work on the classification of depressive disorders (Kiloh & Garside 1963).

Depression research in Australia

In recent years, Australia has contributed around 2–3 per cent of the world's scientific publications on depression, which is commensurate with its efforts in other areas of health research. The major themes dominating recent publications have been: classification, assessment and diagnosis; depression comorbid with other health problems; causes of depression; and biological treatments. Most published research has been carried out with people receiving specialist treatment for depression. Consequently, there is a need for research based in the community and in primary care, on depression in children and adolescents, and on psychosocial treatments and prevention.

Pharmaceutical companies are major sponsors of trials on antidepressant medication. As a result, this research focus is relatively well supported. In contrast, research into population-based epidemiology and prevention and early intervention techniques does not have a strong funding base. Research on the effectiveness of non-pharmacological interventions, alone and in conjunction with drug treatments, needs to be supported.

The major source of public funding for depression research is allocated through the NHMRC. The NHMRC allocates funds to a large program on depression based at the School of Psychiatry at the University of New South Wales, as well as five project grants. They also allocate funds to one network grant and one unit grant, both of which are only partly on depression. Some of the work of the NHMRC Psychiatric Epidemiology Research Centre and the Australian Neuroscience and Mental Illness Research Network is also devoted to depression. It is estimated that the NHMRC allocated $1.25 million to depression research in 1998, which represented 0.8 per cent of its total funding for research and 26 per cent of its funding in the disciplines of psychiatry and psychology. The Australian Research Council (ARC) also allocates for research in psychology. In 1998 there was one Large Grant on depression, worth $30,000.

As well as the research funded by the NHMRC and the ARC there is an important contribution from the Commonwealth, State and Territory health departments. A notable achievement in 1997 was the National Survey of Mental Health and Wellbeing, conducted by the Australian Bureau of Statistics with funding from the Commonwealth Department of Health and Aged Care. This survey provided the first national data on the prevalence of depressive disorders and on the effects of depression on service use and disability in Australia. Other contributions to depression research are made by State governments, for example, by the Victorian government through its funding of the Mental Health Research Institute of Victoria.