

# MENTAL HEALTH IN NEW ZEALAND FROM A PUBLIC HEALTH PERSPECTIVE

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*Published with the permission of the Director-General of Health*

*Published in October 1997 by*

**PUBLIC HEALTH GROUP**

**MINISTRY OF HEALTH**

Wellington, New Zealand

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ISBN 0-478-20849-9 (Book)

ISBN 0-478-20850-2 (Internet)



MANATU HAUORA

## CHAPTER 5: CHILDREN AND ADOLESCENTS

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In recent years there have been growing public concerns expressed about a series of issues relating to childhood and adolescent mental health and psychopathology in New Zealand. These concerns have been reflected in a number of reports that have focused on mental health problems in young New Zealanders. These have included reports on youth suicide (Barwick 1992; Coggan et al 1995), antisocial behaviours and truancy among young people (Report of the Education and Science Committee 1995), cannabis and illicit drug use among the young (Drugs Advisory Committee 1995; Ministry of Health 1996) and the availability of mental health services for young New Zealanders (McGeorge 1995).

All of these reports appear to be symptoms of widespread perceptions that mental health problems among young people are increasing. There is good international evidence to suggest that this perception is well founded. In a recent major review of trends in child and adolescent psychopathology in Europe, Rutter and Smith (1995) found clear evidence to suggest that over the last 50 years there have been clear and non-artefactual increases in a range of childhood and adolescent disorders including conduct problems, depression, substance use behaviours and youth suicide. A comparable analysis has not been conducted for New Zealand but it seems likely that the trends that have been reported for European data also hold for New Zealand data. For example, Deavoll and colleagues (1993) analysed changing patterns of suicidal behaviour in New Zealand over a 100-year period. Their results showed clear evidence of a marked increase in rates of suicide among young people, and particularly young males, with this increase beginning at around the mid-1970s. Although some of this apparent increase may have been due to changes in methods of recording and classifying suicide, it seems likely that rising rates of suicide also reflect an increasing vulnerability to psychiatric disorder among young New Zealanders.

The aims of this review are to provide an overview and synthesis of the evidence on a series of issues relating to mental health during childhood and adolescence including:

- the prevalence of psychiatric disorders among young New Zealanders
- the risk factors for psychiatric disorder
- protective factors that may ameliorate risks of disorder
- the extent to which psychiatric disorders are comorbid
- various programmes and approaches aimed at reducing risks of disorder or leading to improved management.

Wherever possible this review will be based on local research evidence but this will be supported by non-New Zealand-based research to address issues that are not fully covered by the available local evidence. For the purposes of this review, childhood and adolescence is taken to mean the period from birth to the age of 19 years. This definition is consistent with that adopted in other reviews of this topic (McGeorge 1995).

The focus of the review will be largely upon common forms of childhood and adolescent mental disorders and particularly common externalising disorders (conduct disorder, attention-deficit/hyperactivity disorder, oppositional disorders, substance use disorders), and common internalising disorders (affective disorders, anxiety disorders, phobias and related conditions). The reasons for this emphasis are that most of the available research evidence has focused on these disorders and less is known about less common disorders of childhood in New Zealand including autism, childhood schizophrenia and similar conditions.

## THE PREVALENCE OF DISORDER

New Zealand has the advantage of having two large longitudinal studies (the Dunedin Multidisciplinary Health and Development Study and the Christchurch Health and Development Study) that have both conducted extensive prevalence studies of the rate of psychiatric disorder in young people (Anderson et al 1987; McGee et al 1990; Fergusson et al 1993c; Feehan et al 1994). Both of these studies have examined rates of psychiatric disorder in samples of approximately 1000 young people using standardised interviews and diagnostic criteria to estimate the prevalence of disorder at different ages. While these studies are both South Island based, they are likely to provide general guides to the prevalence of disorder among young New Zealanders.

Table 5.1 gives estimates of the prevalence of common psychiatric disorders from both studies using standardised diagnostic criteria set out in the diagnostic and statistical manuals of the American Psychiatric Association including *DSM-III* (APA 1980), *DSM-III-R* (APA 1987) and *DSM-IV* (APA 1994). Table 5.1 shows the following trends:

- Both studies suggest that with increasing age there is evidence of increasing rates of disorder. For example, the Dunedin results suggest that rates of disorder at age 18 years were approximately twice those at age 11 years. Similarly, the Christchurch data show an increase in rates of disorder between ages 15 and 18 years.
- Both studies suggest broadly similar prevalence estimates for specific disorders with the most common disorders being anxiety disorders, mood disorders and conduct disorders.
- Both studies produce quite similar overall prevalence estimates and, in particular, suggest that at age 15 years about a quarter of those studied met diagnostic criteria, whereas by the age of 18 years over a third met these criteria.

**Table 5.1: Prevalence estimates of common psychiatric disorders in childhood and adolescence derived from the Dunedin Multidisciplinary Health and Development Study and the Christchurch Health and Development Study**

	<i>Prevalence at 11 years (%)</i>	<i>Prevalence at 15 years (%)</i>	<i>Prevalence at 18 years (%)</i>
<b>Dunedin study</b>			
Anxiety disorders	7.4	10.7	19.7
Mood disorders	1.8	4.2	18.0
Conduct/oppositional disorders <sup>1</sup>	9.1	9.0	5.5
Attention-deficit disorder	6.7	2.1	*
Substance dependence disorders (excluding nicotine)	*	*	12.2
Any disorder	17.6	22.0	36.6
<b>Christchurch study</b>			
Anxiety disorders	*	13.1	17.1
Mood disorders	*	6.6	22.1
Conduct/oppositional disorders <sup>1</sup>	*	10.8	4.8
Attention-deficit disorder	*	4.8	*
Substance dependence disorders (excluding nicotine)	*	*	8.6
Any disorder	*	24.0	35.0

\* Estimate not available

<sup>1</sup> Estimates at age 18 do not include oppositional defiant disorder (ODD), whereas ODD is included in estimates at ages 11 and 15

It is clear from these estimates that some degree of psychopathology or adjustment problems during adolescence is not uncommon and it is notable that estimates from New Zealand data show broad agreement with prevalence estimates for other societies including the United States (Kashani et al 1987), Canada (Offord et al 1987), the Netherlands (Verhulst et al 1985) and Puerto Rico (Bird et al 1988). This agreement in prevalence estimates across societies clearly suggests that the findings in Table 5.1 are unlikely to be specific to the South Island and probably provide adequate general estimates for other parts of New Zealand.

A limitation of epidemiological estimates of the prevalence of disorder is that they fail to describe the extent to which disorder leads to impairment in the individual's life opportunities and functioning (Bird et al 1988; Weissman et al 1990; Hodges 1993). It is likely that the disorders described in Table 5.1 range from relatively mild and adolescent-limited conditions to severe and chronic conditions. For

these reasons it is perhaps best to consider the estimates in Table 5.1 as providing an upper limit estimate of the number of young New Zealanders with significant psychiatric problems. These points notwithstanding, the findings of both the Christchurch and Dunedin studies clearly show that mental health problems in childhood and adolescence are not uncommon and show a clear tendency to increase up to the age 18 years.

While these studies have documented the likely prevalence of common childhood and adolescent disorders, they are less informative about uncommon but severe problems such as autism, childhood schizophrenia, eating disorders and obsessive-compulsive disorders that may also afflict young people. The evidence on the likely prevalence of these disorders has been ably reviewed by Anderson and Werry (1994) who comment on these matters as follows:

*The prevalence of 'pure' autism in the general population is relatively constant at four-to-five cases per 10 000 children under 14 years, with half being severe cases. There are many more children who show some autistic features in association with brain damage. . . . Childhood schizophrenia is also rare, with similar symptoms to the same disorder presenting later in life. Prior and Werry (1986) assess the prevalence for this disorder as virtually nil before 5 years and around 1 per 1000 until mid adolescence, after which the prevalence climbs 5- to 10-fold into early adult life. . . . Obsessive-compulsive disorder is . . . reported in children in 0.2 percent in clinic populations and 1 percent of inpatients. . . . Anorexia nervosa is a distressing and potentially life-threatening condition that usually only appears at or after puberty and overwhelmingly in girls. . . . Its prevalence has been estimated at 1 percent of 16-year-old girls, with anorexic-type behaviour or unwise dieting being five to six times higher.*

(Anderson and Werry 1994: 323–4).

## ETHNICITY, SEX AND RISKS OF DISORDER IN CHILDHOOD AND ADOLESCENCE

Many public health debates in New Zealand have focused on the role of ethnicity and sex as determinants of risks of disorder and these debates have tended to leave the impression that socially disadvantaged groups including women and Māori are at higher risks of psychiatric disorder. However, examination of the evidence suggests that matters may be more complex than this.

### *Sex and Risks of Disorder*

The relationships between sex and risks of disorder appear to vary over the life course. During middle childhood, males appear to be at greater risk and this is accounted for by higher rates of conduct disorders, attention-deficit disorder and childhood depression in boys. However, during adolescence this sex ratio tends to change, with females having higher rates of disorder than males. For example, in both the Christchurch and Dunedin studies rates of disorder in females 15 years or older were between 1.2 to 1.7 times higher than the rates in males (McGee et al 1990; Fergusson et al 1993c; Feehan et al 1994). The higher rates of disorder in females are explained by increased rates of depression and anxiety disorders among adolescent females.

Over the life-span, these differences appear to even out and the lifetime prevalence of disorder in males and females is similar. For example, the Christchurch Psychiatric Epidemiology Study found that the lifetime prevalence of disorder in males (63 percent) did not differ significantly from that in females (68.5 percent) (Wells et al 1989). The evidence suggests that while over a lifetime both sexes have similar risks of disorder, they differ in the pattern of disorder with females being more prone to internalising disorders, including anxiety and depression, and males being more prone to externalising disorders, including conduct disorders and substance use disorders.

### *Ethnicity*

It might be expected, given the linkages between social disadvantage and risks of mental illness (see later), that membership of a disadvantaged ethnic minority group would be a risk factor for disorder. However, the available research evidence suggests that this is not invariably the case. For example, studies of the prevalence of disorder in the US have shown that African Americans have similar or even lower rates of disorder than Whites despite the highly disadvantaged status of African Americans (Anthony and Helzer 1991; Helzer et al 1991; Weissman et al 1991; Kessler et al 1994).

There have been continued suggestions that the relatively disadvantaged status of Māori and Pacific populations in New Zealand results in these populations being more vulnerable to psychiatric disorders. The evidence for this conclusion is, however, somewhat sparse. A number of authors have examined first admission rate data and have highlighted consistent differences in the patterns of Māori and non-Māori admission rates (Sachdev 1989; Maskill 1991; Ministry of Youth Affairs 1994; Pomare et al 1995). These comparisons show that rates of admission and readmission for psychiatric disorder in adolescence (15–19 years) are higher for Māori than for non-Māori, with Māori males having the highest rates of admission and readmission. However, admission data are a highly fallible and possibly biased method of estimating ethnic differences in risks of disorder and clearly what is required are community-based studies that contrast rates of disorder in Māori and non-Māori groups using standardised diagnostic criteria. To date no such studies appear to have been published.

However, there are some data from the Christchurch Health and Development Study that tend to support the view that risks of adolescent disorders are higher in Māori than in non-Māori (see Table 5.2). It is clear that the overall prevalence of disorder at age 18 years is higher in Māori for all diagnostic groups, with these differences being most marked for conduct disorders and substance use disorders which were between 1.5 to 3.1 times more common among Māori. Ethnic differences in rates of overall disorder appear to be similar to those reported for psychiatric hospital admission data.

However, it should be noted that the apparent associations between ethnicity and risks of disorder may reflect factors, such as socioeconomic status, that are associated with ethnicity and that if such factors were controlled, rates of disorder among Māori may be no higher than among a Pākehā group of comparable socioeconomic status. This view is supported to some extent by findings from the Christchurch Health and Development Study that, when due allowance is made for socioeconomic and related factors, the differences between Māori and non-Māori on measures of educational attainment and juvenile offending tend to be small and frequently statistically non-significant (Fergusson et al 1991, 1993a). Nonetheless, the regional nature of the sample should be borne in mind and it is possible that ethnic differences in the South Island may be less pronounced than ethnic differences in the North Island.

**Table 5.2: Prevalence of common disorders among Māori and non-Māori members of the Christchurch Health and Development Study cohort at age 18 years**

<i>Disorder</i>	<i>Māori prevalence (%)</i>	<i>Non-Māori prevalence (%)</i>	<i>Risk ratio</i>	<i>Significance</i>
Anxiety disorders	24.2	16.1	1.5	< .05
Mood disorders	29.7	20.1	1.5	< .05
Conduct disorder	12.1	3.9	3.1	< .001
Substance dependence disorders (excluding nicotine)	16.5	7.8	2.1	< .005
Any disorder	49.5	33.1	1.5	< .005

An alternative perspective on the origins of the higher rates of mental health and related problems in Māori has emphasised the role of the direct effects of colonisation including dispossession of land, disempowerment and social dislocation in increasing mental health risks among Māori (Jackson 1987; Pomare et al 1995). Collectively, the evidence suggests that there are good grounds for believing that Māori may be at higher risk of childhood and adolescent disorder but to confirm this requires more extensive community surveys of representative samples of Māori and non-Māori. The extent to which ethnic differences in risks of disorder reflect the effects of these different factors remains a matter for debate and further research.

## GENERIC RISK FACTORS FOR CHILDHOOD DISORDERS

While it is clear that the aetiology of childhood disorders varies with the type of disorder being considered, risk factors for different disorders overlap to the extent that these risk factors may be considered as being generic to a given class of disorders (Raphael 1993). To simplify the presentation of risk factors for childhood disorders, this review examines generic factors that have been found to be associated with common externalising disorders (conduct disorder, oppositional defiant disorder, substance use disorders) and common internalising disorders (affective disorders, anxiety disorders). More detailed accounts of the roles of risk factors for specific disorders have been reviewed elsewhere: treatments and risk factors for antisocial and conduct disorders (Patterson et al 1989; Farrington et al 1990; Loeber 1990, 1991); risk factors for attention-deficit/hyperactivity disorders (Hinshaw 1987; Henker and Whalen 1989; Munoz-Millan and Casteel 1989); risk factors for substance use behaviours (Kandel 1980; Newcomb and Bentler 1989; Bucholz 1990; Moncher et al 1991; Conrad et al 1992; Hawkins et al 1992); risk factors for adolescent and childhood depression (Rutter et al 1986; Angold 1988; Kovacs 1989; Fleming and Offord 1990); and risk factors for childhood anxiety disorders (Bernstein and Borchardt 1991; Kashani et al 1991).

## GENERIC RISK FACTORS FOR CONDUCT PROBLEMS AND SUBSTANCE USE BEHAVIOURS

There is a broad consensus in the research literature about the risk factors that may contribute to the development of antisocial and substance use behaviours in young people. These may be described under the following headings:

### *Sociodemographic Factors*

As a general rule, rates of antisocial and substance use behaviours tend to be higher among young people who have low family income, limited parental education and depressed material living standards (Rutter and Madge 1976; Fergusson et al 1990; Williams and McGee 1994). Typically, the greater the degree of sociodemographic disadvantage, the greater the risk of antisocial and substance use disorders.

### *Family Functioning*

The risks of antisocial and substance use behaviours have been linked to a wide variety of indices of family functioning and the adequacy of the family to provide a secure, stable and nurturant child rearing environment. Specific factors include: exposure to family change including parental separation and divorce (Hetherington 1989; Amato and Keith 1991; Wallerstein 1991; Fergusson et al 1994a); marital conflict (Rutter and Giller 1983; Long and Forehand 1987; Fergusson et al 1992); parental psychopathology (Rutter 1989; Downey and Coyne 1990; Connolly et al 1993; Lynskey et al 1994); exposure to abusive experiences in childhood (Browne and Finkelhor 1986; Mullen et al 1988; Bushnell et al 1992; Mullen et al 1993); inadequate parental supervision and discipline (Patterson 1982; Holmes and Robins 1988; Feehan et al 1991); and impaired parent-child relationships (Parker 1979, 1994). The archetypal young person who is at greatest risk of antisocial or substance use behaviours has been reared in a family in which there are multiple problems and difficulties spanning marital functioning, impaired parenting, and exposure to abuse experiences and parental psychopathology throughout childhood (Shaw and Emery 1988; Blanz et al 1991; Fergusson et al 1994b; Shaw et al 1994).

### *Individual Factors*

While both sociodemographic context and family background clearly make contributions to individual risks of antisocial or substance use behaviours, there is increasing evidence to suggest that, independently of these factors, intra-individual variations make clear contributions to the risks of both antisocial and substance use behaviours. In particular, one of the strongest predictors of externalising behaviours during adolescence and young adulthood is the early onset of behaviour problems and difficulties (Loeber 1991; McGee et al 1991; Fergusson and Horwood 1993; Fergusson et al 1995b). The clear continuities that exist between early behaviour and later outcomes may be explained in at least two ways. First, these continuities may reflect common genetic factors that are expressed both as early behavioural difficulties and later psychopathology (Rutter et al 1990; Simonoff et al 1995). The alternative explanation is that these arise because of stable and self-sustaining life course factors that encourage and sustain the development of antisocial behaviours throughout the life course (Quinton et al 1993; Fergusson and Horwood 1996).

### *School Factors*

Much of the individual's social learning occurs at school. Accordingly, it would be expected that school-related factors are likely to play a role in the development of antisocial behaviours. Two lines of evidence support this view. First, there is some evidence that children who show cognitive delays may be at higher risks of later antisocial behaviours (McGee et al 1988; Williams and McGee 1994). However, it has also been argued that cognitive delays may not be a causal factor but rather, merely symptomatic of those at high risk of antisocial behaviours (Wadsworth 1979; Hinshaw 1992; Fergusson et al 1993b; Fergusson and Horwood 1995). Secondly, there is also evidence to suggest that the general culture of the school, including disciplinary practices and related factors, may also influence behavioural outcomes and risks of antisocial behaviours (Rutter et al 1979; Rutter 1983; Figueira-McDonough 1986; Hawkins and Lishner 1987; Kasen et al 1990).

### *Peer Factors*

One of the most striking features of antisocial behaviours, particularly criminal offending and substance use behaviours, is that they show a marked rise in adolescence (Moffitt 1993). One possible explanation for this is clearly that the development of antisocial behaviours in adolescence is encouraged and facilitated by peer-group influences. Although these are among the strongest predictors of both antisocial behaviours and substance use behaviours (Kandel 1980; Farrington et al 1990; Moffitt 1993; Quinton et al 1993; Fergusson et al 1995a; Fergusson and Horwood 1996), it is likely that the causal relationships between individual behaviours and peer-group affiliations are complex. While the peer group may shape an individual's behaviour, an individual's behaviour may shape peer group values and norms (Moffitt 1993).

### *The Media*

The media have frequently been blamed for encouraging antisocial and substance use behaviours in young people. For example, it has been suggested that such factors as television violence (Heath et al 1989), alcohol and cigarette advertising (DiFranza et al 1991; Fischer et al 1991; Connolly et al 1994) and the depiction of suicide in the media (Bollen and Phillips 1982; Kessler and Stipp 1984; Gould and Shaffer 1986) may contribute to substance use, antisocial behaviours and suicide in young people. While media presentations that depict, or act to normalise, antisocial or substance use behaviours may encourage these behaviours and increase risks, it is clear that, given the mix of family, social, individual, school and peer related factors that are likely to contribute to individual behavioural variation, the role of the media in encouraging and supporting antisocial or substance use behaviours is likely to be relatively minor (Rutter and Smith 1995).

## RISK FACTORS FOR INTERNALISING DISORDERS

### DEPRESSION AND AFFECTIVE DISORDERS

There has been a growing literature on risk factors for depression and affective disorders in childhood and adolescence (for reviews of this evidence see Rutter et al 1986; Angold 1988; Kovacs 1989; Fleming and Offord 1990). The major risk factors for these conditions are listed on the following page.

### *Social Disadvantage*

In common with externalising disorders, it has been found that rates of affective disorders in childhood tend to be higher among children from socially disadvantaged backgrounds (Angold 1988). As noted above, evidence from the Christchurch Health and Development Study tends to suggest that rates of depression in Māori at age 18 years were significantly higher than rates in non-Māori.

### *Family History of Depression*

Depression in childhood is frequently associated with a family history of depression (Hammen et al 1987; Klein et al 1988; Orvaschel et al 1988; Downey and Coyne 1990) and this is generally consistent with behavioural genetic studies indicating substantial heritability in tendencies to depressive disorders (Wierzbicki 1987; Rende et al 1993).

### *Exposure to Childhood Adversity*

As with externalising disorders, exposure to adverse or dysfunctional childhood experiences, including parental conflict, childhood abuse and parental adjustment problems is associated with an increased vulnerability to depressive disorders (Rutter et al 1986; Angold 1988; Kovacs 1989; Fleming and Offord 1990).

### *Exposure to Adverse Life Events*

Depression during childhood and adolescence is episodic and is frequently preceded by adverse life events. Common life events that may precipitate depression in adolescents include death and bereavement in the family, relationship breakdown, peer rejection, school failure and associated events (Goodyer 1990; McGee and Stanton 1992).

### *Individual Factors*

While the general social environment may both influence individual vulnerability to disorder and act to precipitate disorder, individual differences in vulnerability to depression, which may be genetically based, also appear to be important (Wierzbicki 1987; Rutter et al 1990; Rende et al 1993). Three lines of evidence support this. First, the results of behavioural genetic studies using twin designs have suggested there is substantial heritability in a tendency to depressive illness (Wierzbicki 1987; Rutter et al 1990; Rende et al 1993). Secondly, there is evidence of clear continuities in depressive conditions, and in many young people depression is a recurrent and ongoing condition rather than being limited to a single acute episode (Kovacs et al 1984; McGee and Williams 1988; Harrington et al 1990). Finally, there has been evidence linking measures of personality (and notably neuroticism) to risks of depressive disorders (Duncan-Jones et al 1990). All three lines of evidence clearly suggest that intra-individual factors may make strong contributions to the individual's risks of affective disorder.

## ANXIETY DISORDERS

In contrast to the literature on risk factors for externalising disorders and depression, the literature on risk factors for childhood anxiety disorders is very sparse. This is well illustrated by a recent review of the epidemiology of anxiety states in which Costello and Angold (1995) were able to describe the known risk factors in only two pages of text. Among the risk factors they noted were: sex (females are more prone to anxiety than males), genetic factors (these appear to overlap with the genetic factors for depression) and a range of familial risks including social disadvantage, poor parental education, parental psychopathology, parental emotional problems, school failure and stressful life events.

## PROTECTIVE FACTORS FOR CHILDHOOD AND ADOLESCENT CONDITIONS

While a large amount of research evidence has focused on the identification of risk factors for childhood and adolescent disorders, there has also been a growing literature on protective factors. Protective factors are factors that may act to ameliorate the risks of disorder in those exposed to adverse risk factors, even though they may not reduce risk among those not exposed to the risk factor (Rutter 1985). The following factors may act to ameliorate or mitigate risk in those exposed to high-risk childhoods.

### *Intelligence and Problem-solving Abilities*

Resilient young people appear to be characterised by higher intelligence or problem-solving skills than their non-resilient peers (Kandel et al 1988; Masten et al 1988; Werner 1989; Seifer et al 1992; Herrenkohl et al 1994).

### *External Interests and Affiliations*

Children from high-risk backgrounds who develop either strong interests outside the family or form attachments with a confiding adult outside their immediate family may be more resilient to the effects of family adversity (Werner 1989; Jenkins and Smith 1990).

### *Parental Attachment and Bonding*

A further factor that may increase resiliency in children from high-risk backgrounds is the nature of parent–child relationships. The presence of warm, nurturant or supportive relationships with at least one parent may act to protect against, or mitigate, the effects of family adversity (Werner 1989; Jenkins and Smith 1990; Wyman et al 1991; Seifer et al 1992; Gribble et al 1993; Bradley et al 1994; Herrenkohl et al 1994).

### *Early Temperament and Behaviour*

There has also been some evidence to suggest that temperamental and behavioural factors may be associated with resilience to adversity (Werner 1989; Wyman et al 1991).

## COMORBIDITY

An important finding that has emerged as a result of the study of the prevalence of disorder in childhood and adult populations has been increasing recognition of the frequent comorbidity of psychiatric disorders. A large number of studies have shown that those who present with one disorder (for example, conduct disorder) are at increased risk of other disorders (for example, substance use or depressive disorders). Table 5.3 shows the pattern of comorbidities that has emerged from the Christchurch and Dunedin studies (and indeed many other studies). The table gives estimates of the odds ratios between diagnostic classifications of disorder at age 18 years in both of these cohorts. The odds ratio measures the increase in the risks (odds) of disorder given the presence of another disorder. Thus, if disorders are not associated, the odds ratio will be one, and odds ratios greater than one show increasing risks of disorder conditional on the presence of another disorder. The data from the Christchurch study are shown on the upper diagonal of the table and the data from the Dunedin study in the lower diagonal. Both studies lead to very similar conclusions, that there are strong tendencies for internalising disorders (anxiety, depression) to be comorbid and for conduct and substance use disorders to be comorbid. The table shows that those with a depressive disorder had over six times the risks of having an anxiety disorder and those with a conduct disorder had over 15 times the risk of having a substance use disorder. There were small odds ratios between internalising (anxiety, depression) and externalising (conduct, substance use) disorders but the table shows in all cases that there were tendencies for those with internalising disorders to have higher rates of externalising disorders.

**Table 5.3: Odds ratios between major disorders for Dunedin and Christchurch samples at age 18 years (Dunedin sample below diagonal, Christchurch sample above diagonal)**

	<i>Anxiety disorders</i>	<i>Mood disorders</i>	<i>Conduct disorders</i>	<i>Substance dependence disorders</i>
Anxiety disorders	–	6.5	2.5	2.8
Mood disorders	6.8	–	3.1	2.7
Conduct disorders	1.4	1.4	–	25.0
Substance dependence disorders	2.6	3.3	15.1	–

The major implication of comorbidity is that many young people who present with disorder meet diagnostic criteria for more than one disorder. For both the Christchurch and Dunedin data, at age 18 years in the region of 40 percent of those who met criteria for a psychiatric disorder met criteria for two or more disorders.

The origins of comorbidities of psychiatric disorders are still largely unexplained. However, Caron and Rutter (1991) have suggested four possible reasons for comorbidity. Firstly, one disorder may act as a risk factor for another disorder. For example, involvement in antisocial behaviours may lead to the development of depression as a result of societal and other responses to antisocial behaviours (Patterson and Capaldi 1990; Rhode et al 1991). Secondly, both disorders may be comorbid as a result of common risk factors that predispose individuals to both disorders. Thus, for example, exposure to abusive experiences during childhood may increase risks of both later conduct problems and later

depression. Thirdly, even though the risk factors for the two disorders may not be in common, these risk factors may themselves be correlated. Thus, for example, cigarette smoking and conduct problems may be correlated because parents in high-risk families may more often smoke, leading to higher rates of both conduct disorder and cigarette smoking in their offspring. Finally, the comorbid condition may represent a distinct entity that differs from either condition in isolation.

Some exploration of the hypothesis that comorbidity arises because the risk factors for disorder are either common or correlated has been conducted as part of the Christchurch study. These analyses suggest that a substantial proportion of the comorbidity of adolescent conditions may arise because the risk factors and life processes that contribute to one disorder also contribute to other disorders (Fergusson and Lynskey 1995, 1996; Fergusson et al 1996a, 1996b, 1996c).

An important issue raised by the comorbidity of disorder concerns the extent to which interventions aimed at reducing childhood and adolescent risks of disorder should be targeted at specific disorders and conditions, or should be targeted at improving the general mental health and adjustment of young people. The available evidence tends to favour the view that interventions targeted at addressing a range of conditions rather than specific disorders may be more successful. Two lines of evidence support this view. First, the evidence on comorbidity reviewed above suggests individuals who are at risk of one disorder are at increased risks of other disorders. Secondly, as noted earlier, there is considerable overlap between the risk factors for different disorders. Both lines of evidence suggest that addressing environmental risk factors that are associated with a range of comorbid disorders is likely to provide the most effective approach to prevention. This conclusion does not, however, preclude the possibility that interventions that are targeted at specific conditions and problems may be needed to supplement more broadly based prevention programmes.

Against this general background, possible strategies for the primary prevention of psychiatric disorders in young people will now be considered.

## OPPORTUNITIES FOR PRIMARY PREVENTION

The above necessarily brief overview of the common risk factors for child and adolescent psychiatric disorder suggests that there are a number of opportunities for intervention methods to reduce risks of childhood psychiatric disorder. These may be thought of as lying on a continuum from interventions that involve macrosocial changes to interventions that may be targeted at high-risk families or individuals. Opportunities for prevention and possible prevention strategies are outlined below.

### MACROSOCIAL (POPULATION) INTERVENTIONS

#### *Increased Social Equity and Social Opportunity*

There is a widespread belief that improvements in social equity issues relating to sex, ethnicity, poverty and similar factors will make positive contributions to reducing risks of childhood and adolescent disorder. This view has been underwritten by the pervasive correlations that have been found between indices of social disadvantage and risks of disorder, and is well illustrated by the comments of Coggan and colleagues (1995) in their recommendations for the prevention of intentional injuries. These authors note that:

*Policymakers and programme managers must also acknowledge that substantial reductions in the level of intentional injury are unlikely to occur without addressing macro-social issues such as poverty, chronic unemployment, racism, sexism, and undereducation.*

*(Coggan et al 1995: xiv)*

Similarly, Raphael (1993) emphasises the need to address social equality issues including poverty and the position of Aboriginal people in Australian society in discussing prevention of childhood disorder in an Australian context.

However, there are reasons for believing that, by themselves, changes in macrosocial conditions relating to social justice, equality and access to opportunity are unlikely to make large contributions to reducing risks of childhood and adolescent psychiatric disorders. Three lines of evidence support this conclusion. Firstly, the findings on rates of disorder for African Americans, when compared to Whites, clearly show that social disadvantage, inequality and discrimination do not invariably lead to increased risks of disorder. These results clearly suggest the effects of disadvantage may be modified by other factors. Secondly, historical evidence does not generally support the view that improvements in general social conditions lead to corresponding improvements in psychiatric disorder. While it is possible to show that, with improving social conditions, there have been general increases in the physical health of children, if anything, the reverse appears to be the case for psychosocial disorders. In particular, in reviewing evidence on historical trends in childhood and adolescent psychosocial disorders in Europe over the last 50 years, Rutter and Smith (1995) note that the largest increases in rates of disorder appear to have occurred during periods when opportunities for young people were increasing, unemployment was declining, physical health improving and affluence increasing. This historical evidence clearly suggests that changes in general social conditions by themselves may not lead to corresponding changes in risks of disorder. Thirdly, the assumption that improved social equity may lead to improved mental health fails to explain the mechanisms by which general social changes are translated into changes at the individual level and implicitly this argument appears to be based on a 'trickle down' theory that assumes that macrosocial change leads to corresponding change in individual vulnerability to disorder.

For all three reasons it is apparent that it is unlikely that general changes in social equity will, by themselves, lead to large changes in the vulnerability of young people to psychosocial disorders. However, such changes may play an important role in providing a social ecology and context that foster the development of programmes designed to reduce risks of psychosocial disorders. For these (and many other good reasons), efforts to reduce sources of social inequality and discrimination including ethnic inequalities, sex inequalities, poverty and related factors may provide an important context for the development of policy efforts to address psychosocial disorders in young people.

### *Full Employment Policies*

Over the past decade there have been increases in unemployment among young people in New Zealand. For example, in 1986 the rate of officially recorded unemployment in those aged 15–19 years was 6.1 percent (Department of Statistics 1986) but this rose to a rate of 10.9 percent in 1992 (Department of Statistics 1992). There have been recurrent public debates about the role of youth unemployment in encouraging rates of psychiatric disorder among young New Zealanders and it has been argued that full employment policies may play a role in reducing risks of psychiatric disorder (Bethwaite et al

1990; Shirley et al 1990). There is a large body of evidence linking unemployment and mental health and other outcomes (for reviews of this evidence see Platt 1984; Warr 1987; Forcier 1988; Bethwaite et al 1990; Shirley et al 1990; Ezzy 1993; Hammarstrom 1994). This evidence has clearly documented the fact that unemployment is associated with increased risks of psychological maladjustment (Stafford et al 1980; Banks and Jackson 1982; Patton and Noller 1984; Tiggeman and Winefield 1984; Hammarstrom et al 1988; Hammarstrom 1990; Winefield and Tiggeman 1990; Morrell et al 1994), substance use behaviours (Layne and Whitehead 1985; Peck and Plant 1986; Power and Estaugh 1990; Janlert and Hammarstrom 1992) and juvenile offending (West and Farrington 1977; Farrington et al 1986). However, the causal role of unemployment in increasing individual vulnerability to psychiatric disorder remains controversial. On the one hand it may be argued that exposure to unemployment may act to provoke the onset of psychiatric disorder by acting as an event that reduces self-esteem, increases feelings of alienation and hopelessness and by excluding the individual from access to full social participation. On the other hand it may be argued that the higher rates of psychiatric disorder among the unemployed may reflect the fact that those predisposed to such disorders may be more likely to become unemployed.

This issue has recently been examined by the Christchurch Health and Development Study in a comparison of rates of mental health problems among the unemployed and non-unemployed at age 18 years (Fergusson et al 1997). This analysis led to three major conclusions. Firstly, those subject to long-term (6+ months) unemployment were at increased risk of a range of adolescent disorders including anxiety disorders, substance use disorders, depression and conduct disorder. Secondly, much of the elevated risk of the unemployed was attributable to personal and social factors that were present prior to the onset of unemployment. Thirdly, even after statistical control for pre-unemployment factors there was evidence that those exposed to long-term unemployment were at increased risk of anxiety disorders and substance use disorders.

Given this evidence it would seem that, like other macrosocial changes, the role of unemployment in creating psychiatric disorder in young people is relatively minor and it is unlikely that changes in youth employment levels will, by themselves, make major contributions to changes in the level of psychiatric disorder in young people. However, such policies may contribute to promoting positive mental health by encouraging a social context that gives all young people access to opportunities and by providing a climate that may support the development of policies more specifically targeted at addressing psychosocial disorders in young people.

### *The Media*

Media influences have often been blamed for rising rates of childhood and adolescent problems, and possible effects of the media have been noted in the areas of television violence (Heath et al 1989), alcohol and cigarette advertising (DiFranza et al 1991; Fischer et al 1991; Connolly et al 1994) and in the encouragement of suicidal behaviours (Bollen and Phillips 1982; Kessler and Stipp 1984; Gould and Shaffer 1986). However, it is unlikely that, by themselves, media influences play a major role in determining individual vulnerability to psychosocial disorders in young people. In general, the effects of the media on individual behaviour have been found to be small and, in a review of the impacts of media influences on vulnerabilities to disorder, Rutter and Smith (1995) have noted that:

*. . . it is most unlikely that adverse effects of mass media largely account for the rise in psychosocial disorders. However, the media do reflect changing attitudes in society and thus, they may augment the effects of societal change.*

*(Rutter and Smith 1995: 784)*

These conclusions suggest that, like changes in social equity, the major role of media influences may be to reinforce and support a social context that may act to encourage or hamper efforts at prevention.

### *Public Awareness of Disorder*

One of the recurrent findings from epidemiological studies of childhood and other disorders is that the majority of those meeting diagnostic criteria fail to seek treatment or support for their condition (Dubow et al 1990; McGee et al 1990; Whitaker et al 1990; Offer et al 1991; Fergusson et al 1993c). For example, both the Christchurch and Dunedin studies suggest that over half of young people meeting diagnostic criteria for disorder at age 15 years had failed to seek treatment (McGee et al 1990; Fergusson et al 1993c). The most commonly given reasons for this appear to be that the young person either did not think they had a problem (and this is particularly the case for antisocial and substance use disorders) or believed that they could manage this problem by themselves. This very large amount of unrecognised or untreated disorder within the community naturally raises the conjecture that, by better education of young people, their parents, teachers and others about the characteristics of psychiatric disorders, more young people could be encouraged to seek treatment. This clearly suggests the need for better population education about psychiatric disorders in childhood and adolescence.

There is, however, one area of mental health in which greater publicity may be contraindicated. Research into the impacts of publicity on suicide has generally suggested that the effects of greater public attention about suicide may be to increase suicide risks. For example, Gould and Schaffer (1986) reported on a US-based series of television programmes depicting suicide. They found that these programmes had the effect of increasing rates of youth suicide. Other studies have also shown that publicity about, and depiction of, suicidal behaviours tends to increase rather than decrease risks of these behaviours (Bollen and Phillips 1982; Kessler and Stipp 1984). For these reasons it may be argued that, as a general rule, publicity and public education about youth suicide should not be encouraged and that treatment of issues relating to youth suicide should be presented in the context of programmes encouraging positive mental health among young people rather than being specifically targeted at the topic of youth suicide (Gould and Schaffer 1986). See also Chapters 23 and 24.

## COMMUNITY-LEVEL INTERVENTIONS

At a more specific level it may be proposed that community-based prevention methods may be effective in reducing risks of psychiatric disorders in young people. Offord and Bennett (1994) describe such methods as aiming to 'strengthen the ability of the community to promote prosocial behaviour and deter antisocial and delinquent behaviour through changing or enhancing existing systems.' The evidence of the effectiveness of community-based interventions is somewhat contradictory. In a review of 29 community-based studies aimed at reducing antisocial behaviour and delinquency, Joffe and Offord (1987) found that there was little evidence of programme effectiveness, although programmes that included direct behaviour modification methods appeared more useful than those that involved community-level interventions without behaviour modification components. On the other hand, Pentz and colleagues (1989) have described a community-based intervention for substance use behaviours that was implemented in 15 US communities and that showed positive effects in reducing adolescent substance use behaviours including tobacco use, alcohol consumption and cannabis use. The apparent discrepancy between the conclusions of Joffe and Offord's (1987) findings and the results reported by Pentz and colleagues (1989) may be explained by the fact that the intervention described by Pentz and colleagues (1989) involved concerted efforts at change at the individual, school and peer level. It may be that the success of this programme was more attributable to its direct impact on the immediate social environment of young people rather than to wider community changes.

## FAMILY-BASED INTERVENTIONS

If there is one theme that unites the literature on common childhood and adolescent psychosocial disorders it is the finding that frequently young people with significant disorder, and particularly multiple comorbid disorders, tend to experience childhood and family environments that are unsatisfactory and compromised in many ways. The profile of the high-risk child or adolescent is characterised by such features as: exposure to marital conflict, impaired parenting behaviours, limited parental discipline and supervision, exposure to child abuse, marital instability, parental psychopathology, disadvantaged material conditions, family violence and associated features. These results strongly suggest that the general nature and quality of childhood experiences may play an important role in determining individual vulnerability to later psychosocial disorders and particularly conduct or substance use disorders. In turn this leads to the conjecture that modification of family and childhood environments may be an important strand in developing prevention programmes to reduce risks of psychosocial disorders in young people.

There have been two approaches to addressing these issues. First, a number of programmes have targeted specific childhood risk factors including marital breakdown (Bloom et al 1982; Stolberg and Garrison 1985), childhood trauma (Pynoos and Nader 1989; Raphael 1993), exposure to child abuse (Gray et al 1977; Merry and Andrews 1994) and parenting skills (Baum and Forehand 1981; Patterson et al 1982; Wahler and Dumas 1987). The alternative approach has been provided by programmes that through methods of home visiting and family support, attempt to improve overall family functioning, child rearing environment and family opportunities. Evidence (Yoshikawa 1994) suggests that to be successful such programmes need to be:

- specifically targeted at high-risk families
- intensive in their treatment of family problems
- conducted over a protracted period of time
- initiated during the preschool years.

The available evidence suggests that such programmes may be successful in encouraging positive changes in outcomes for children in high-risk families, including improved utilisation of health services (Olds and Kitzman 1990; Johnson et al 1993), reduced risks of child abuse (Rosenberg and Reppucci 1985; Garbarino 1986; Olds et al 1986; Hawaii Department of Health 1992) and reduced risks of adolescent adjustment problems (Yoshikawa 1994).

## SCHOOL- AND PEER-BASED APPROACHES

While the family forms an important developmental context that may influence individual vulnerability to disorder, it is clear that school and peer influences are also important. Research into school influences has consistently suggested that school climate may make a contribution to risks of disorder independently of social, community or familial influences (Rutter et al 1979; Rutter 1983; Figueira-McDonough 1986; Hawkins and Lishner 1987; Kasen et al 1990) and these findings have led to the development of school-based interventions that attempt to develop school and classroom environments that minimise risks of adjustment problems during childhood (Gottfredson 1987; Hawkins and Lishner 1987; Farrington et al 1990; Evans and Okifuji 1992; Kellam et al 1994; Walker et al 1995).

Theoretically, peer-based programmes should be highly influential in producing changes in antisocial and substance use behaviours in adolescence, since research has suggested that one of the strongest predictors of these behaviours in adolescence is the nature of the young person's peer affiliations (Kandel 1980; Farrington et al 1990; Moffitt 1993; Quinton et al 1993; Fergusson et al 1995a; Fergusson and Horwood 1996). However there may be difficulties in implementing such programmes since it is clear that peer relationships are likely to be reflections of the prevailing adolescent and youth culture. In their review of these issues, Rutter and Smith (1995) note that one feature of many developed societies has been the development of a youth culture which has become increasingly isolated and insulated from adult influence. Such climates clearly pose barriers to the extent to which peer-based interventions can be effective.

There have been a number of peer-based interventions used to reduce adolescent risks. These approaches have largely been directed at a reduction of substance use or antisocial behaviours. Three general approaches have been employed. The most frequently used method has been peer-based life skills training programmes that teach children and young people specific skills and strategies to avoid social pressures to use drugs. In a review of 143 school-based interventions using a life skills approach, Tobler (1986) concluded that peer teaching was a highly effective approach in deterring substance use in young people. A second approach has been through co-operative peer-based learning. In a review of this approach, Farrington and colleagues (1990) concluded that co-operative learning may have a number of benefits including reduced rates of suspension and expulsion from school. A third approach is through programmes that encourage antisocial young people to form affiliations with prosocial peers. While relatively little research has been conducted on this approach, positive effects of the encouragement of prosocial peer affiliations have been reported in at least one study (Feldman et al 1983).

## INDIVIDUAL-BASED INTERVENTIONS

While societal influences, the community, the family, school and peers are all likely to act in various ways to influence individual vulnerability to psychosocial disorders, it would be misleading to assume that individual psychopathology is merely a reflection of social pathology or family shortcomings. There is now increasing evidence from behavioural genetic studies to suggest the presence of substantial genetic influences in many disorders independently of the effects of environmental factors. These considerations suggest that an effective prevention strategy needs not only to address issues at the levels of society, the community, the family, school and peers, but also to target intervention efforts at high-risk children on the assumption that early intervention with children showing symptoms of disorder is likely to reduce future risks of disorder. The opportunities for such interventions appear to be most clear for conduct and antisocial disorders, which are often of early onset (Loeber 1991; McGee et al 1991; Fergusson and Horwood 1993; Fergusson et al 1995b). It should be noted that in this context, primary prevention tends to merge into secondary prevention since efforts to intervene in disorders during early childhood can be seen as both providing a basis of prevention of disorder in the future and also providing treatment for a developing disorder.

A review of clinical methods for addressing disorders in early childhood is well beyond the scope of this review but various approaches to the clinical treatment of common psychiatric disorder in childhood are summarised by Rutter and colleagues (1994). However, an essential prerequisite for clinical-level intervention with early onset disorders is the availability of childhood and adolescent psychiatric services that are capable of identifying, diagnosing and treating children with early onset disorders. A discussion of the professional and administrative framework needed to accomplish this is also beyond the scope of this review but the foundations for the development of such services in New Zealand have been discussed by McGeorge (1995).

## STRATEGIES FOR REDUCING RISKS OF PSYCHIATRIC DISORDERS

Research into many childhood and adolescent disorders clearly shows that the aetiology of these disorders is strongly multicausal and involves a large number of social, family, school, peer, genetic and related influences. The major implication of the strong multicausality of disorder is that it is unlikely that any single intervention that addresses a restricted set of risk factors will make a major impact on the prevalence of disorder within the community. However, a multifaceted approach to prevention that targets different domains of risk factors is likely to succeed. The above review suggests that this general strategy should include the following.

- Macrosocial changes, including improved social equality, full employment, the development of positive media environments and greater public awareness of psychosocial disorders in young people. As noted above, it is unlikely that, by themselves, these changes will lead to dramatic changes in the risks of childhood disorders but they may play the important role of providing a social ecology that supports and reinforces more targeted approaches to intervention.
- Community-level interventions that build upon the local strengths of the community and are designed to meet sociocultural and other features specific to a given community may provide an important adjunct to macrosocial change. However, the available evidence suggests that such programmes are likely to be effective only if these programmes contain components that are targeted at producing change in the individual's immediate social environment. These components include elements of more targeted family, school, peer and individual programmes designed to produce individual change and to reduce individual risk.
- Family-based interventions targeted at high-risk families are likely to be a very important strand of interventions aimed at reducing risks of disorder. To the extent that the family is likely to provide the strongest socialisation influences, it is clear that interventions that reduce the number of young people exposed to disadvantaged, dysfunctional and difficult family situations are likely to play an important role in the primary prevention of psychiatric disorders.
- Family-based programmes, however, need to be supplemented by school- and peer-based programmes that focus on the development of school and peer cultures and environments that minimise risks of disorder.
- Childhood and adolescent psychiatric services have a major role. To the extent that individual differences, independently of social context, are likely to play an important role in determining vulnerability to disorder, it is important that other interventions at macrosocial, community, family, school and peer levels are supplemented and strengthened by the availability of adequate, well funded and suitably staffed childhood and adolescent psychiatric services that have the capacity to provide diagnosis and clinical treatment for children showing early onset disorders and, of course, the management of psychosocial disorders throughout the period of childhood and adolescence.

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