MENTAL HEALTH IN NEW ZEALAND FROM A PUBLIC HEALTH PERSPECTIVE

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Within psychiatry, the amount of attention paid to eating disorders has varied over time, although both of the two main variants – anorexia nervosa and bulimia nervosa – have been recognised for a long time (Gull 1874; Blachez 1896). The central features of these related disorders are overvalued ideas about body size and shape, which are often accompanied by dissatisfaction with the body, and an attempt to change body size and shape by dieting and other weight control measures (eg, Cooper and Fairburn 1993). These are disorders that predominantly affect women, and the peak age of onset is in adolescence (Beumont 1970; Bushnell et al 1990). These disorders tend to have a chronic fluctuating course and may persist for many years (Morgan and Russell 1975; Ratnasuriya et al 1991; Collings and King 1994).

The three main features of anorexia nervosa are self-induced weight loss, fear of becoming fat, and amenorrhoea (or, in men, loss of libido). It has been graphically described as ‘the relentless pursuit of thinness’ (Bruch 1974). Restriction of food intake, and strategies for weight control such as over-exercising or use of purgatives are commonly used by those with anorexia nervosa. The anorexic patient typically describes being afraid of gaining weight, even when she or he is significantly underweight, and this is often associated with anxiety and odd eating practices such as hoarding and hiding food, cutting food into very small pieces, eating slowly and drinking large amounts of water. The most widely used criterion for what is significantly underweight is a body weight more than 15 percent below the standard for height and age.

After several decades of neglect within psychiatry, bulimia nervosa became a focus of attention about 15 years ago with the publication of Russell’s criteria for bulimia nervosa (Russell 1979), and the inclusion of bulimia nervosa in the American Psychiatric Association’s (APA) DSM-III manual of mental disorders (APA 1980). The essential features of the disorder are recurrent episodes of over-eating that are subjectively uncontrolled, fear of gaining weight, and attempts to limit weight gain through extreme weight-control strategies such as fasting, vomiting, use of purgatives or diuretics. The disorder often begins when dieting produces intense urges to eat. A feeling of compulsion to give in to these urges produces a binge in which a large amount of food is eaten in a short period of time, followed by vomiting or use of purgatives to counteract the binge.

When treatment is sought for these disorders, it is often long term and expensive, requiring extensive therapeutic input, and commonly, in the case of anorexia nervosa, in-patient care. Both bulimia nervosa and anorexia nervosa have an adverse effect on the social and occupational functioning of the individual, and cause significant distress not only to the sufferers but also to their families. Both disorders can also cause a range of adverse health outcomes. Even when normal body weight is maintained, bulimia nervosa can cause erosion of tooth enamel and tooth decay, parotid gland enlargement, oesophageal and peptic ulceration, haematemesis, disturbance of electrolyte balance, impaired autonomic response, suppressed thyroid functioning and cardiac arrhythmia. Where body weight is low, amenorrhoea and
osteoarthritis can develop, and with extreme weight loss hypothermia, hypoglycaemia, and multiorgan failure can result (Russell and Beumont 1995). Long-term follow-up studies of hospitalised patients have found alarmingly high mortality rates for anorexia nervosa (almost one in five in some studies (Crisp et al 1992)), although findings from tertiary referral centres are unlikely to generalise to all those with the disorder.

PREVALENCE AND INCIDENCE

PREVALENCE OF ANOREXIA NERVOSA AND BULIMIA NERVOSA

Early reports from the United States based on unvalidated research interview or self-report questionnaires found an alarmingly high prevalence of bulimia nervosa (eg, Halmi et al 1981), but later work with better research methodology has generally found between 3 and 5 percent of young women have had bulimia during their lifetime (Pyle et al 1986; Schotte and Stunkard 1987). The main published New Zealand work on the prevalence of eating disorders in the general population has been the Christchurch Psychiatric Epidemiology Study (CPES) (Wells et al 1989; Bushnell et al 1990, 1993, 1994), which included an assessment of anorexia nervosa and bulimia nervosa. Consistent with other studies from European countries which have shown that anorexia nervosa occurs in less than 1 percent of women during their lifetime (Rastam et al 1989; Rathner and Messner 1993), the Christchurch study found only three women per thousand in the general population had, during their lifetime, experienced this disorder (see Table 16.1). The rate was so low as to preclude detailed further analysis, and this study was unable to address risk factors for the development of anorexia nervosa.

Table 16.1: Lifetime prevalence of eating disorder syndromes in New Zealand

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
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<tbody>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td></td>
<td>(SE)</td>
<td>(SE)</td>
<td>(SE)</td>
</tr>
<tr>
<td>Anorexia nervosa</td>
<td>0.0</td>
<td>0.3</td>
<td>0.1</td>
</tr>
<tr>
<td>(0.0)</td>
<td>(0.3)</td>
<td>(0.2)</td>
<td></td>
</tr>
<tr>
<td>Bulimia nervosa</td>
<td>0.2</td>
<td>1.9</td>
<td>1.0</td>
</tr>
<tr>
<td>(0.5)</td>
<td>(0.5)</td>
<td>(0.3)</td>
<td></td>
</tr>
</tbody>
</table>

Source: Wells et al 1989

The CPES found that bulimia nervosa had affected 2 percent of women and was rare in men. It was more common among younger women (see Table 16.2), with nearly one in 20 18–24-year-olds having had the disorder during their lifetime. During the age period 18–44 years, in which women were at greatest risk of having the disorder, 2.6 percent (95% CI 1.7–4.0) had the disorder during their lifetime, 1.5 percent (95% CI 0.9–2.6) in the last six months, and 1.0 percent (95% CI 0.5–2.0) currently had the disorder. Putting aside the issue of DSM-III diagnosis, a wide range of disordered eating habits was common. Among women aged 18–44 years, lifetime experience of recurrent binge eating was reported by 22.5 percent, depressed mood and self-depreciating thoughts after bingeing by 10.6 percent, use of at least three extreme weight control measures (such as use of cathartics, vomiting or fasting) by 8.2 percent, and fear of being unable to stop a binge once it had begun by 5.0 percent (Bushnell et al 1990).
Table 16.2: Lifetime prevalence of bulimia nervosa in women by age group

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Percentage</th>
<th>95% Confidence interval</th>
</tr>
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<tbody>
<tr>
<td>18–24</td>
<td>4.5</td>
<td>2.3–8.5</td>
</tr>
<tr>
<td>25–44</td>
<td>2.0</td>
<td>1.1–3.5</td>
</tr>
<tr>
<td>45–64</td>
<td>0.4</td>
<td>0.1–2.5</td>
</tr>
<tr>
<td>Total</td>
<td>1.9</td>
<td>1.2–3.0</td>
</tr>
</tbody>
</table>

Source: Bushnell et al 1990

Using the more narrowly defined syndrome of bulimia nervosa delineated later in a revision of *DSM-III (DSM-III-R)* (APA 1987), Welch and Hall (1990) found that 2.5 percent (95%CI 1.0–5.6) of a group of 243 nursing students met full criteria for current bulimia nervosa. This rate was higher than expected on the basis of the CPES data on women in the general population, and it has been suggested that this may be because young women in tertiary educational settings are at greater risk of developing these disorders (Welch and Hall 1990). Recent British studies using a narrow definition of bulimia nervosa have found between 1 percent and 2 percent of women in the general population meet criteria for this disorder (Cooper et al 1987; Fairburn and Hay 1993).

Two New Zealand studies using a scale known as the Eating Attitudes Test have also been conducted to establish the prevalence of disturbed attitudes to food and eating that are thought to be precursor to the development of eating disorder. In a sample of Auckland schoolgirls aged 13–17 years, 14 percent had the disturbed attitudes to food and eating that suggested a potential eating disorder. Among women attending their general practitioner in Christchurch, a study that used the same test found that 72 percent wanted to weigh less and 46 percent were dissatisfied with their body shape, although only 35 percent were in fact overweight for their height (Wells et al 1986). This is consistent with evidence from other Western countries which suggests that the experience of feeling ‘too fat’ is reported by most girls at some stage of their adolescence (Nylander 1971), an experience which increases with age and is more common among those with higher body weight (Dwyer et al 1967).

**INCIDENCE OF ANOREXIA NERVOSA AND BULIMIA NERVOSA**

The extent of change of the rate at which new cases of anorexia nervosa and bulimia nervosa are developing has been the subject of some debate. Most of the evidence from the United Kingdom and New Zealand suggests that the incidence of anorexia nervosa has remained stable at around 0.4–1.6 per 100000 per year (Hall and Hay 1991; Fombonne 1995), although there are some contradictory findings from case-register studies in Scandinavia (Moller-Madsen and Nystrup 1992; Pagsberg and Wang 1994).

Indirect evidence of a change in incidence of bulimia is described by Bushnell and colleagues (1990) who showed a strong cohort effect in lifetime prevalence: that is, having ever experienced the disorder was reported less often by older age groups studied. This may have been because during recent years the disorder has become more common among young women than it used to be. However there may be another explanation: that the older women either forgot or failed to disclose their experience when asked about behaviour about which they felt guilty and ashamed.
Among females aged 10–24 years in the general population of Bornholm, Denmark, between the years of 1970 and 1984, the rates of new cases of anorexia nervosa ranged between 8 and 16 per 100 000 per year (Pagsberg and Wang 1994). In contrast, during the five years 1985 to 1989, there were 57.1 new cases per 100 000 females 10–24 years of age, per year. The incidence of bulimia nervosa was also established in this study. Rates of bulimia nervosa ranged between 4 and 8 per 100 000 per year between 1970 and 1984, and increased to nearly 18 per 100 000 during the five years 1985 to 1989. This study provides some support for the observation that the incidence of eating disorders, both anorexia nervosa and bulimia nervosa, is rising – at least in Scandinavia. However, the case-register data may be influenced by greater awareness of the disorders by both health professionals and patients, and the rates of bulimia nervosa reported are not consistent with the findings from community samples of much higher rates of bulimic behaviour, suggesting that many cases may be missed by the assessment made to determine the presence of disorder for the register.

**RISK FACTORS**

Research into risk factors for eating disorders has focused on four broad classes of risk factor: developmental, familial, biological, and sociocultural. Developmental issues have included identity, body image, autonomy and separation-individuation. Familial issues have included family history of psychiatric disorder, degree of cohesion, handling of conflict, maintenance of rigid boundaries, and overprotection of children by their parents. Biological factors have included the onset of puberty, dieting and obesity. Sociocultural risk factors have included the value of perfectionism, the value of a thin-toned physique, the level and nature of media attention paid to the ‘ideal’ body shape for women, and the choice of certain professions (White 1992a, 1992b). This wide-ranging vista of potential risk factors is supported only in a patchy way by adequate research-based evidence.

It has been powerfully argued that the rise of societal perfectionism and narcissism has fuelled the rise in eating disorders. This argument asserts that the difficulty women have valuing themselves for what they are rather than how they look in a world organised for the convenience of men is intrinsically related to the value placed on slenderness:

> This form of tyranny has been attributed to a neurotic hatred of womanhood in male advertising moguls and their female colleagues who identify with the aggressor, in promoting a thin prepubertal male body as the ideal for females. It is of note that the current image of the ideal woman is contrived so as to remain out of reach of most at whom it is aimed.

* (Russell and Beumont 1995: 461)

However, the evidence for these assertions is far from conclusive.

Studies of women who have developed an eating disorder have described a range of psychological difficulties including the development of an identity (Lidz 1968), the development of body image and fear of maturation (Crisp and Kalucy 1974; Streigel-Moore et al 1986; Killen et al 1992), and the achievement of autonomy (Bruch 1978). It remains unclear whether these factors are part of the disorder, correlates of the disorder, or risk factors.

Many of the studies that purport to identify risk factors for eating disorders have failed to use appropriate methodology. In particular, several studies implicate as risk factors characteristics that do not necessarily antedate the onset of the disordered eating behaviour and a number of studies have described samples of bulimics from various sources, examining demographic or other characteristics without reference to the base rate of those characteristics in the general population, or in other common psychiatric disorders.
Although some elements of disordered eating are common among young women, a comparatively small proportion develop a full bulimic or anorexic syndrome. The strength of evidence for other factors that render some young women susceptible to developing an eating disorder is variable, but some interesting findings have emerged.

**YOUTH AND FEMALE GENDER**

The two most clearly identified risk factors for the development of an eating disorder are being female (see Table 16.1) and being young. The search for what factors lead some young women to develop an eating disorder while others do not has considered the role of puberty. Girls experiencing eating disorder symptoms have been found to be more developmentally advanced than those without the disorder (Killen et al 1992). Sexual maturity approximately doubled the risk of developing an eating disorder, (OR 1.8, 95%CI 1.2–2.8), and this effect was found to be independent of the effect of age or the interaction between age and sexual maturity. Female neurobiological responses to dieting may contribute to the sex differences in eating disorders. There is evidence of sex differences in the effects of food restriction on brain serotonin. Serotonin is known to play a role in the control of food intake (Silverstone and Goodall 1986). In women, but not in men, moderate weight loss has been shown to lead to a fall in plasma tryptophan, the essential amino acid from which the neurotransmitter serotonin is produced (Anderson et al 1990; Walsh et al 1995).

**CULTURE**

It has often been asserted that eating disorders are a Western culture-bound phenomenon. However, recent evidence suggests that although eating disorders may be more rare among non-Western cultures, they do occur, but do not exhibit the same characteristic features. Over half of a series of 70 Hong Kong Chinese patients with anorexia nervosa were similar to Western anorexics in most ways, but did not exhibit any fear of fatness throughout the course of their illness (Lee et al 1993). Poor food intake was blamed on gastric fullness, ‘no hunger’, or loss of appetite. Similar findings have also emerged from studies (albeit with small numbers of subjects) in Singapore (Ong and Tsoi 1982), India (Khandelwal and Saxena 1990), and West Malaysia (Goh et al 1993). A modified approach to the assessment of anorexia nervosa was used in a study of a large sample of over 36000 students in Japan. The criteria were defined as ‘complete unconcern regarding extreme thinness’ and a ‘distorted attitude and behaviour toward eating’ and found a point prevalence of 44 per 100000 high-school students for the anorexia nervosa syndrome. This is a considerably rarer disorder than anorexia nervosa in Western countries. Only a quarter of the cases identified at clinical assessment had a ‘thin ideal’ (Mizushima and Ishii 1983).

Lee and colleagues (1993) have argued that non-fat-phobic anorexia nervosa may not be a culture-bound syndrome, and the disorder may exist even in the absence of the current vogue for thinness that permeates Western cultures. One possibility that might explain both the differing features in other cultures, and the (probably) higher rates of disorder in the West, is the existence of a ‘core disorder’ that finds more fertile ground for development in the presence of the more widespread body shape and size idealisation, and associated dieting behaviour, that occur in the West.
Within Western culture, ethnic differences in attitudes to eating, weight and dieting seem to be reflected in rates of eating disorder. In the US, there is some evidence of greater fear of being overweight among Blacks than among Whites (Massara and Stunkard 1979), and less pressure to diet despite being overweight (Thomas and James 1988). Several studies have found ethnic differences in the rates of disordered eating. The prevalence of bulimia nervosa and related eating disorder has been found to be higher among White women than Black women (Nevo 1985; Gray et al 1987; Gross and Rosen 1988). The positive attitudes towards large body size among Pacific people (PHC 1994) may place them at risk of other diseases, but may function as a protective factor for eating disorders, at least until they begin to assimilate the views of the prevailing culture in New Zealand.

Among other migrants to a dominant Western culture, there seems to be a fairly rapid assimilation of the prevailing culture’s norms. Although there appear to be relatively low levels of eating disorders among Greek girls in Greece (Fichter et al 1988), among the children of Greek migrants to Australia, extreme weight loss behaviour was more common than for ‘Anglo-Australian’ girls (Mildred et al 1995). There is little direct evidence about the effect of this process on Pacific migrants to New Zealand (or on Māori), but anecdotal reports suggest a disproportionately low rate of referral from these cultural groups to specialist eating disorder treatment centres. Among referrals to the Auckland eating disorder service between 1993 and 1996, only 6.3 percent were Māori and 1.8 percent Pacific people (Dr H Clarkson, personal communication, 8 July 1996).

While eating disorders may occur in other cultures, the particular constellation of cultural beliefs and attitudes to body shape and dieting that prevail in Western cultures may have the effect of providing an environment within which eating disorders can be fostered and flourish without being identified from the outset as undesirable and abnormal. Interventions that militate against the idealisation of thinness and the ‘normalisation’ of dieting, while difficult to implement, may prove one of the most viable strategies for reducing the incidence of eating disorders.

**GENETICS AND THE ENVIRONMENT**

The relative contribution of genetic and environmental risk factors for several psychiatric disorders including bulimia nervosa has been explored (Kendler et al 1995). One thousand and thirty female twin pairs were assessed for a lifetime history of a range of psychiatric disorders. Multivariate statistical modelling of the relationship between disorder and genetic, familial/environmental, and individual specific environmental risk factors was carried out. Because the various risk factor domains cause different patterns of comorbidity, it was concluded that genetic and environmental risk factors for these disorders do not influence comorbidity in the same manner. The results of this study suggest the possibility of a genetically mediated neurobiological predisposition common to phobia, panic disorder and bulimia nervosa. Although many psychological theorists have stressed the essential role of the family in shaping emotional function and personality, little or no consistent evidence was found in this study that the liability to major depression, generalised anxiety disorder, phobia, alcoholism, or panic disorder was substantially influenced by environmental factors shared in families. The exception to this was bulimia nervosa. Although this analysis was limited by the low rates of disorder, and a resulting small number of affected twins, the results suggest that for bulimia nervosa, along with genetic factors described above, familial and environmental factors do play a significant aetiological role.
SEXUAL ABUSE

The impact of sexual and physical abuse on the subsequent development of eating disorders has been examined in a number of studies. Welch and Fairburn (1994) reported a series of three case-controlled comparisons in which 50 community-based subjects with bulimia nervosa were compared with 100 community-based comparison subjects without an eating disorder, 50 community-based comparison subjects with other psychiatric disorders, and 50 patients in a treatment clinic who had bulimia nervosa. While sexual abuse produced a three- to sixfold increase in the likelihood of subsequently developing bulimia nervosa, the increased risk of disorder does not appear to be specific to bulimia nervosa, and nor is it relevant to most cases of the disorder. Sexual abuse appears to be a risk factor for psychiatric disorder in general among young adult women.

Similar results have been found in other studies that have emphasised that childhood sexual abuse is never necessary nor sufficient for the late development of eating disorder, while an adverse family background may be a more important aspect of aetiology (Kinzl et al 1994).

Comparisons of rates of sexual abuse among eating-disordered patient samples have not found it to be significantly more common than in the general psychiatric population. Folsom and colleagues (1993) compared 102 in-patients admitted to an eating disorders programme with 49 in-patients consecutively admitted to a psychiatric unit. Although all of these patients reported high rates of sexual abuse, the hypothesis that sexual abuse or physical abuse has occurred more frequently in an out-patient eating disorder unit than in an in-patient general psychiatric population was not supported by these findings. In addition, eating disorder subjects did not experience more severe forms of sexual abuse than psychiatric subjects. Furthermore, there was no association between history of physical and sexual abuse and the severity of the eating disorder symptoms.

Two New Zealand studies have also examined the relationship between sexual abuse and eating disorders. A survey of 2250 women randomly selected from the electoral roll in Dunedin established that the experience of sexual abuse in childhood approximately doubled the risk of subsequently developing an eating disorder (OR 2.2, 95%CI 1.0–5.0) (Mullen et al 1993). If the sexual abuse during childhood involved intercourse, the likelihood of subsequently developing an eating disorder was almost four times that found in those who were not so abused (OR 3.7, 95%CI 1.2–11.6). The relationship between sexual abuse and subsequent disorder was not specific to eating disorders, however. Similar odds ratios were found for the definition of psychiatric ‘caseness’ based on the Present State Examination, suggesting a general increase in subsequent risk for all psychiatric disorder.

The CPES also explored the impact of intrafamilial sexual abuse in childhood on subsequent development of psychiatric disorders, including bulimia nervosa (Bushnell et al 1993). Among women aged 18–44 years in the general population, 14 percent (95%CI 8–21 percent) reported intrafamilial sexual abuse while they were growing up. For over half of these women, the experience involved intercourse or attempted intercourse, over half reported long-term effects on their emotions and nerves, and for one in five the experience involved physical violence. This study examined the relationship between sexual abuse and the development of substance use disorder (OR 1.2, 95%CI 0.5–2.5), depression/dysthymia (OR 2.0, 95%CI 0.5–2.5), and disordered eating (OR 1.5, 95%CI 0.9–3.8), concluding that although all three disorders showed increased risk ratios, only depression had an odds ratio significantly greater than 1.0, and the rates within each of these different types of disorder did not differ from one another. The effects of sexual abuse appeared to be widespread and not very specific, and showed as additional comorbidity rather than increased severity within a disorder. This finding conflicts with studies of eating-disordered patient samples. It may be that the high reported rates of abuse among eating-disordered patients (eg, Oppenheimer et al 1985; Hall et al 1989) are not related to the presence of bulimia nervosa per se, but rather to do with other factors (like comorbidity) that may interact with the likelihood of seeking treatment.
OTHER RISK FACTORS

In addition to the work described above, the CPES (Wells et al 1989) explored the relevance of several risk factors for the development of eating disorders. Some of these risk factors were determined at the initial interview, but others (e.g., parenting style of ‘affectionless control’, parental psychiatric disorder) were defined at a reinterview conducted two years later. Other factors examined were family background (parental socioeconomic status, family size); family breakdown (brought up by both natural parents, separated from both parents for more than a month, parental harmony); adolescent indicators (age of leaving school, school qualifications); marital status; years of education; parenthood; race; and country of origin (Bushnell et al 1990; Bushnell 1990).

Bulimia nervosa was not evenly distributed across parental socioeconomic status (SES), but was not associated with higher parental SES in a simple way as some clinical reports would suggest. No significant relationship was found between the presence of bulimia nervosa and marital status of subjects, years of education, parenthood, race or country of origin. Indicators of family breakdown and adolescent adjustment had modest predictive value for the development of the eating disorder. However, bulimia nervosa was associated with high rates of comorbid disorder (Bushnell et al 1994), and this predictive value seemed to be mediated largely through depression. Among people with bulimia nervosa, there was a trend towards higher rates of experience of parental ‘affectionless control’. This also was not specific to bulimia nervosa, as it was also found in substance use disorder and depression. There was a trend towards more depression having been experienced among the parents of those with bulimia nervosa than in the parents of non-disordered women (even more than among the parents of depressed subjects), although with the small sample sizes involved, and the consequent lack of power in this study, these differences across types of disorder do not reach statistical significance.

Overall, there was support for the concept that a disturbed family environment in which there was either parental psychiatric disorder, or a parenting style with low caring and nurturing but high levels of rules and control, with associated low school achievement, increases the risk of a child developing a range of psychiatric disorders, including eating disorders. The onset of puberty for young women increases the risk of developing an eating disorder.

The effect of level of exposure to media images of ideal body image on eating disorder symptoms was explored in a novel study of female college students (Stice et al 1994). Latent trait analysis of this cross-sectional study revealed a direct relation between level of media exposure and severity of eating disorder symptomatology. This study has a number of limitations: the cross-sectional design precludes strong causal inferences and may not reflect the temporal relationship between the variables. The measure of media exposure is quite approximate, and the study relies upon self-reported data from college students, which does not permit differentiation between different types of disordered eating. In spite of these reservations, the study provides evidence of a link between exposure to the thin ideal portrayed in the media and eating disorder.

PRIMARY PREVENTION

School-based and college-based preventive programmes have been designed to modify knowledge and thereby behaviour in relation to dieting and eating (Shisslak et al 1990), to target background factors such as self-esteem (Shisslak et al 1987), mastery, coping and depression (Phillips and Piran...
Eating Disorders

1992). Some of these programmes have been carefully designed to address potentially modifiable risk factors, targeting young adolescent females with knowledge about the harmful effects of unhealthy weight regulation, promotion of healthy forms of weight control, and developing skills for recognising and resisting the media and sociocultural influences linked to the pursuit of thinness (Killen et al 1993).

Although some of these programmes have shown a short-term change in attitudes to eating, body image, and knowledge about disordered eating behaviour (Shisslak et al 1990; Outwater 1991), other studies have found no discernible impact (Ben-Tovim 1992; Killen et al 1993). In spite of the energy, commitment and ingenuity shown in their development and implementation, there is no clear evidence of a long-term impact of school-based preventive programmes.

Another approach has been to target high-risk groups of young women such as models, gymnasts, ballet dancers and athletes which have a high prevalence of dieting and where there are widely held notions of an ideal physique (Moriarty and Moriarty 1986; Hergenroeder et al 1991). Phillips and Piran (1992) described a programme based in a ballet school aimed at improving self-esteem and discouraging a focus on weight and dieting. This produced a short-term change in eating attitudes in a positive direction. The impact on eating behaviour remains unclear. However encouraging these findings may be, the epidemiology of disordered eating inevitably means that such programmes can have only limited impact at a public health level: while many ballet dancers may be at risk of becoming restricters or binge eaters, most binge eaters and restricters are not ballet dancers.

SECONDARY PREVENTION

Treatment of bulimia nervosa has been shown to have beneficial effects in the short term (Mitchell et al 1993; Fairburn et al 1993; Agras et al 1994) with some evidence of longer-term effects (Fallon et al 1991; Fairburn et al 1995), although a proportion of patients, perhaps as high as one-third, continue to do poorly despite intensive treatment (Fallon et al 1991, 1994; Hartmann et al 1992).

The findings are less clear for anorexia nervosa. Long-term follow-up of patients in tertiary treatment facilities reveals high mortality rates, and rates of recovery that vary between one-quarter and one-half over periods of five to 10 years (Jarman et al 1991; Deter and Herzog 1994; Gillberg et al 1994; Eckert et al 1995). The extent to which the outcome of this often severe and chronic fluctuating disorder is influenced by treatment remains unclear.

The natural history of both anorexia nervosa and bulimia nervosa is not well established. The long-term follow-up studies that have been carried out have been based in specialist tertiary referral centres (Crisp 1988; Collings and King 1994), in which there is likely to be greater severity of illness, worse long-term outcome and more comorbid disorder. Those women with eating disorders outside of specialist treatment centres may have less severe disorders. Although the latter group may have a greater likelihood of response to treatment, they may also have a greater chance of spontaneous recovery. There are few data to suggest whether intervention produces a sustained improvement in the likelihood of recovery, over and above the likelihood of recovery that would occur naturally.
Primary prevention programmes require that risk factors are known, and that known risk factors are modifiable. Implementation of successful prevention strategies targeting eating disorders is not close to fruition. Beyond being female and adolescent, our knowledge of risk factors is fragmentary and incomplete. The strongest evidence concerning predisposing factors suggests a contribution of both genetics, biology and environment. Occurring at an increased rate in young women at the onset of sexual maturity, eating disorders are more likely in an adverse family environment in which sexual abuse has occurred, or a disturbed family environment in which there was either parental psychiatric disorder or a parenting style with low caring and nurturing but high levels of rules and control. These factors are not easily amenable to modification by primary prevention strategies.

Other potential (though more speculative) targets for a primary prevention programme might be the media and advertising industry, which have a significant role in creating the illusion of an unrealistic and unattainable body size and shape for women. However, data linking exposure to images of the desirability of thinness to harmful outcomes are necessary to inform and shape public opinion. When one considers the huge volume of evidence in relation to the harm resulting from the media promotion of alcohol and tobacco, and the decades of reluctant response within the media world to pressure from both the general public and public health agencies to change standards in relation to tobacco and alcohol advertising, one cannot be optimistic about the effects of putting resources into changing the images of women preferred by the media.

In spite of the paucity of data to suggest whether intervention produces a sustained improvement in the likelihood of recovery, over and above the likelihood of naturally occurring recovery, there is some evidence for the long-term effectiveness of treatment at the more severe end of the spectrum, at least for bulimia nervosa. This is more than can be said for the evidence of the efficacy of primary prevention strategies that have been conducted to date. It is not yet clear what aspects of which risk factors are amenable to change. Rather than targeting primary prevention, it may at this stage be more realistic for the expenditure of time, energy and financial resources to focus on secondary prevention: the early recognition and prompt and effective treatment of eating disorders once they have begun to develop. To do this effectively would require more widespread knowledge about the recognition of eating disorders, as well as the development of appropriate treatment facilities for these disorders once they have been recognised. While traditionally much of this role has fallen to the general practitioner, a concern with nutrition, fitness and health is an acceptable part of the role of many agencies such as gymnasiums, aerobics classes, ballet schools, sports clubs, health food shops and even modelling agencies. Carefully planned intervention with the staff of these agencies might enable some of them to not only give a more healthy message to young women using their services, but to encourage recognition and self-referral in the presence of an eating disorder.


