

EDITORIAL

Can dieting cause an eating disorder?¹

Although claims that the eating disorders have reached epidemic proportions in the West are clearly exaggerated (Fairburn & Beglin, 1990), anorexia and bulimia nervosa are among the commonest psychiatric disorders to affect young women in Western societies (Whitaker *et al.* 1990; Kendler *et al.* 1991). Because of the high morbidity and mortality associated with these disorders (for reviews see Hsu, 1990, 1991), there is no cause for complacency, and continued research into their pathogenesis, treatment and outcome is needed.

Unfortunately, not much ground has been gained in our understanding of the aetiology of the eating disorders since my last review of the topic (Hsu, 1983). In his authoritative review of anorexia and bulimia nervosa, Steinhausen (1994) again emphasized ‘the multifactorial origin coupled with multiple determinants and risk factors ... and their interaction within a developmental framework’ (p. 428). Is there a way to synthesize these multiple determinants and risk factors and their interaction in order to avoid what has become a climate of intellectual agnosticism regarding the aetiology of the eating disorders?

THE ROLE OF DIETING BEHAVIOUR

Elsewhere, I have stated that dieting may be a major risk factor for the pathogenesis of an eating disorder, and that the prevalence of the eating disorders occur in direct proportion to the prevalence of dieting behaviour in a given community (Hsu, 1990). However, Steinhausen (1994) stated, in the aforementioned review, that dieting may be a benign practice in the great majority of young women, without progression to clinical status. Can dieting cause an eating disorder?

Longitudinal studies are one of the best ways to answer this question. To the best of my knowledge, there are five longitudinal studies that examined the evolution of eating disturbances. King (1989), using the Eating Attitudes Test (EAT-26, Garner & Garfinkel, 1979, 1980), surveyed 720 London general practice patients aged between 16 to 35 years (534 female, 186 male). He identified 76 high-scorers (71 female), of whom 69 (64 females) were interviewed as well as a randomly chosen sample of 40 low-scorers. Among the female high-scorers, he found six bulimia nervosa and 15 partial syndrome, 13 obsessional and 23 normal dieters. None of the low-scorers had any eating disorder symptomatology. At the 18 months postal follow-up of 57 female high-scorers, 12 had worsened in their symptomatology, seven had improved, while the remainder were unchanged. In particular, at least nine normal or obsessional dieters (23%) at follow-up had developed bulimic symptoms while only four (10%) were no longer dieting. Clearly, dieting behaviour is often a precursor of an eating disorder. Unfortunately, King did not follow-up any of the low EAT scorers or identify the factors that turned dieting into an eating disorder.

Using the EAT-26 (Garner *et al.* 1982) and the General Health Questionnaire (GHQ-28), (Garfinkel & Garner, 1982), Patton and colleagues (1990) screened 1011 15-year-old London schoolgirls and selected three groups for interview: all girls scoring above the EAT cut-off ($N = 83$), a randomly selected group scoring above the GHQ cut-off but below the EAT cut-off ($N = 74$), and a randomly selected group scoring below the cut-off on both questionnaires ($N = 73$). Among the EAT + group, 20 (24%) were cases (two bulimia nervosa (BN), 18 partial syndrome, presumably all

¹ Address for correspondence: Professor L. K. George Hsu, Department of Psychiatry, Tufts University School of Medicine, 750 Washington Street, NEMC 1007, Boston, MA 02111, USA.

bulimic), 49 (59%) were dieters and 14 (17%) normal eaters. In the EAT-group, two (1%) had BN, both were GHQ+, 30 (20%) were dieters and 115 (79%) normal eaters. No anorexia nervosa (AN) was identified in the survey, although at least two AN were subsequently found (through medical records and independent informants) among the girls who did not participate in the study, but they were inexplicably excluded from the final analysis. The point prevalence of dieting was estimated to be 31%. At 1-year follow-up, 176 of the original 230 girls were re-interviewed. Three of the four original BN remained cases, but one had become a dieter. It was unclear how many of the original 17 partial syndromes were re-interviewed, but at least two had developed BN. Of particular interest is the finding that among the 61 original dieters, 13 (21%) had become cases (two BN and 11 partial syndrome), 23 (38%) remained dieters and 25 (41%) were non-dieters. In contrast, three (3%) of the original 98 non-dieters had developed a partial syndrome, 13 (13%) had become dieters and 82 remained as non-dieters. The authors stated that the relative risk of dieters becoming cases was eight times that of non-dieters. Among the many risk factors studied, the investigators found several (higher weight, familial obesity/dieting, social stress) to correlate with dieting behaviour, but total change in GHQ was the only factor that distinguished between dieters who became cases and those who did not. Unfortunately, the authors did not define what constitutes 'dieter' or 'subclinical case', and they did not elaborate on what 'total change in GHQ' actually means, but it would appear that general psychopathology mediates the effect of dieting behaviour to result in the onset of an eating disorder.

A Swedish study yielded similar findings although the author drew a different and, in my view, unjustified conclusion. Schleimer (1983) followed-up 130 young women who had reported significant eating disturbance during an earlier survey conducted by Nylander (1971). Among these 130 girls, 107 had, in Nylander's survey, endorsed three of 10 'anorectic symptoms' in addition to weight loss. The 10 symptoms were: anxiety, depression, anxiety associated with meals, increased interest in food and food preparation, chilliness, constipation, amenorrhoea of at least 3-months' duration, fatigue, deteriorating school performance and loss of interest. In addition, four girls had reported a weight loss of at least 10 kg and 19 had consulted a child psychiatrist for anorexic symptoms. In retrospect, Schleimer diagnosed 10 as having anorexia nervosa and two as 'anorectic behaviour with bulimic disorder' among these 130 'dieting' girls.

At 10-year follow-up, Schleimer was able to interview 97 (74.9%) girls, while 12 (9.2%) completed a questionnaire. Results at 10-year follow-up of the 97 interviewed can be summarized as follows. Sixty-seven women (61.5%, all between 24 and 38 years of age) reported persistent dieting behaviour, two were vomiters, and one abused laxatives. Sixteen (10%) had lost at least 6 kg, while five (5.1%) had at least 3 months of amenorrhoea. On actual weighing, 13 (13.4%) were at least 15% below normal weight. Twenty-seven had elevated anorexic scores (of at least 10) on the Anorectic Behavior Scale (Fries, 1975) (mean for normal = 3.2, s.d. not stated). In summary, Schleimer stated that at the time of the follow-up interview, 47 (48.5%) had no eating disturbance, 26 (26.8%) had a dieting neurosis (i.e. a persistent dieting problem with 'fixation' on diet and weight), 21 (21.6%) had anorexic-like behaviour but not diagnosable as AN, and three (3.1%) had diagnosable AN. She concluded: 'Thus, no direct causal relationship between dieting and AN could be demonstrated. If dieting was caused by mental insufficiency, this may lead to the development of anorectic behavior and/or AN. However, it appears that dieting in a physically or mentally healthy teenager can hardly have such effects.' (page 1 of Abstract).

Aside from the obvious problems of defining 'mental insufficiency' and deciding whether any persistent dieting behaviour can be regarded as 'mentally or physically healthy', Schleimer's conclusion is not justified for the following reason: during the entire 10-year follow-up period, four of the women (case Nos. 10, 31, 32, 106) who initially did not have diagnosable AN had developed AN; three of them were still AN at the time of follow-up, while one (case No. 106) had recovered. The 'lifetime' prevalence of AN among those initial non-AN dieters is, therefore, 3.4% (4/118). The Mayo Clinic study (Lucas *et al.* 1991) found that in Rochester, Minnesota, on 1 January 1985, 99 females with a lifetime history of AN were alive among a female population of 32353. The age adjusted prevalence rate for females is, therefore, 269.9 per 100000 persons, i.e. 0.27%. Assuming

that the prevalence of AN is similar in Minnesota as in Southern Sweden, the role of dieting behaviour in increasing the risk of the development of AN may be considerable.

Marchi & Cohen (1990) followed the development of eating problems in a large random sample of children originally living in two upstate New York counties (male = 333, female = 326). Interviews were conducted in 1975, 1983 and 1985, when the children were 6-, 14- and 16-years-old respectively. In late childhood and adolescence (presumably during the 1985 assessment), the children and their mothers were interviewed with the Diagnostic Interview Schedule for Children (DISC) (Costello *et al.* 1984; Cohen *et al.* 1987), allowing diagnosis of AN and BN to be made. However, details of how eating disturbances changed longitudinally among individuals who were dieting were not reported. The authors simply stated that extreme symptoms of AN in adolescence were foreshadowed by picky eating in early and later childhood and extreme symptoms in the 12- to 20-year group were associated with elevated anorexic symptom levels 2 years earlier. In addition, problem meals in early childhood, weight reduction concerns and bulimic symptoms were all predictive of extreme symptoms of BN 2 years later in adolescence. Despite the lack of well-defined criteria of what constitutes anorexic or bulimic symptoms, the findings suggest that rigorous dieting behaviour predicted both the development of AN and BN within a 2-year period.

A final longitudinal study is described by Rathner & Messner (1993). They used the EAT and the ANIS (Anorexia Nervosa Inventory for Self-rating) questionnaire to screen two German-speaking state secondary (11–14 year-olds) and two German-speaking state high schools (15–19 year-olds) in a rural Italian town in Southern Tyrol. A total of 517 girls participated in the first-stage screening, representing 75.6% of the total enrolled female students in these four schools. The interview stage was, however, carried out 1 year later. All the at-risk girls ($N = 50$) and a random sample of sub-risk ($N = 25$, 46% of sub-risk group) and no-risk ($N = 32$, 8% of no-risk group) girls were interviewed. At-risk was defined as a score of > 30 on the EAT-40, or > 65 on the ANIS, and sub-risk was defined as scoring 20–29 on the EAT-40, or 46–64 on the ANIS. In addition to the interview, a search was made of all the in-patient case registers for the previous 3 years at the local hospitals. In all, three cases of AN (two by interview, one by case register), three subclinical AN, and two subclinical BN (all by interview) were identified. All cases and subclinical cases (except the one AN identified by case register) belonged to the at-risk group at screening, none belonged to the sub-risk or no-risk group. The AN case identified by case register belonged to the sub-risk group, but she was not among those chosen for interview. The authors, therefore, concluded that abnormal eating attitudes and behaviour (including dieting) preceded the onset of subclinical or full-blown cases of eating disorders. However, their conclusion was undermined somewhat by the fact that the subjects were not interviewed at the screening stage (i.e. it is not certain that the at-risk or sub-risk subjects were not actually cases at the time of the screening). Furthermore, the criteria for a case or a sub-clinical case were not provided.

In summary, the longitudinal studies point unanimously to the role of dieting behaviour in the pathogenesis of an eating disorder. Obviously, not all dieters proceed to develop an eating disorder; and, therefore, other risk factors must be also involved if a dieter is to develop an eating disorder.

OTHER RISK FACTORS

Concordance rate for AN among monozygotic twins is much higher than among dizygotic twins (Holland *et al.* 1984, 1988; for earlier studies see Garfinkel & Garner, 1982; Hsu, 1990). However, Walters & Kendler (1995) found no monozygotic twin pairs and two dizygotic twin pairs were concordant (dizygotic proband wise concordance = 25%) for AN in a population based sample of 2163 female twins. This unexpected finding may be related to the relatively low response rate and questionable validity of the interview instrument for detecting AN in an epidemiological survey. For BN, concordance rate among monozygotic twins is higher than among dizygotic twins (Kendler *et al.* 1991; for earlier studies see Hsu, 1990). Many studies have found that the eating disorders run in families (e.g. Winokur *et al.* 1980; Hudson *et al.* 1983; Gershon *et al.* 1984; Kasset *et al.* 1989;

Logue *et al.* 1989; Strober *et al.* 1990). Family members of eating disorder patients also have a significantly higher rate of mood disorders (Winokur *et al.* 1980; Hudson *et al.* 1983; Gershon *et al.* 1984; Rivinus *et al.* 1984), although some studies found this association to occur only for depressed eating disorder patients (Biederman *et al.* 1985; Strober *et al.* 1990). In the aforementioned population-based twin study, Kendler and his associates (Kendler *et al.* 1991; Walters & Kendler, 1995) found shared familial aetiological factors between AN, BN and major depression, although they could not determine if the familial component is environmental or genetic. Family history of alcoholism or substance use is reported by a few studies (Rivinus *et al.* 1984; Hudson *et al.* 1987; Kasset *et al.* 1989).

Setting aside whether genetic or environmental factors are at play, we can conclude at least that a family history of an eating disorder, a mood disorder and perhaps alcohol or substance use, increases the risk of developing an eating disorder.

Solid data to support the role of other risk factors are lacking. Among the many risk factors suggested, personality traits such as over-control of emotionality and obsessiveness (Strober, 1980; Rothenberg, 1986; Casper, 1990), and aberrant family interaction (for review see Humphrey, 1992) are often mentioned. Furthermore, because the eating disorders are much more common among women than men, and their onset occur most commonly during adolescence and early adulthood, developmental factors particularly those affecting the female (Striegel-Moore, 1995) are likely to be involved in the pathogenesis of an eating disorder. Finally, neurobiological dysfunction triggered by dieting may also intensify the eating disturbance (Kaye, 1995).

It is unclear why these risk factors culminate in anorexia nervosa in some individuals and in others, bulimia nervosa. I speculate, without support of research data, that a propensity towards obesity and poor impulse control may be involved in the development of bulimia nervosa.

A COHERENT SYNTHESIS

Dieting causes the onset of an eating disorder if there is a family history of an eating disorder, mood disorder or alcohol/substance use, and if it is intensified by certain developmental issues. Other factors such as personality characteristics, family interaction pathology and biological vulnerability may also contribute to this pathogenetic pathway. Research utilizing a prospective longitudinal design or perhaps a case-control design should be undertaken to test these variables.

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