

Disturbed families, or families disturbed?†

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Parents frequently perceive themselves as responsible for their child's eating disorder. Clinicians who have observed a family's overinvolvement with an ill, underweight child may readily subscribe to this view. Historical observations certainly support the perspective that the family has a key role in the development and treatment of the eating disorders. In the 19th century, Gull, Lasègue and others recommended the removal of the anorexic patient from the family environment. As with autism and schizophrenia, the notion of the psychopathogenic mother received temporary credence in the mid-20th century. Later, Minuchin *et al* (1978) and the Philadelphia group proposed their structural model of family organisation in anorexia nervosa. Characteristic enmeshment, overprotectiveness, rigidity and lack of conflict resolution were identified as features of the 'psychosomatic family'.

The family of a child with an eating disorder may or may not demonstrate dysfunction after the child has recovered. Of siblings raised in the same family, one may develop an eating disorder while another does not. The presence of recognised aetiological factors may not add up to the development of an eating disorder in all exposed individuals. Is it possible to establish the direction of causality, when complex intrafamilial dynamics are at play with biological, psychological and other social factors? Or are families responding to – rather than generating – the symptoms of anorexia?

Anorexia and bulimia nervosa differ from other psychiatric disorders in terms of the skewed age and gender distribution. Given the point in the life cycle at which these conditions tend to arise, it makes intuitive sense to consider the role of the family. This can be seen to operate within a wider, multifactorial model of causation.

There is a growing evidence base for genetic and biological causal mechanisms, as well as psychosocial factors, including, of course, maturational issues and the role of adverse life events. In a recent meta-analysis, Stice (2002) highlighted the limited predictive power of individual risk and maintenance factors. Independent causal relationships are generally difficult to demonstrate, although the study did find good evidence for the role of body dissatisfaction (for example) as a risk factor for dieting, negative affect and eating disorders, as well as a maintaining factor for bulimic conditions. Multivariate aetiological and maintenance models merit further exploration to elucidate the true risk factors, potentiating or protective factors and the function of mediating variables.

Some aetiological factors are shared with other psychiatric conditions, rather than being uniquely predictive of eating disorders. Fairburn *et al* (1997) proposed that bulimia nervosa is the result of exposure to general risk factors for psychiatric disorder in addition to risk factors for dieting. Further, particular factors may carry variable weight depending upon the age of the patient and indeed the type of eating disorder. The adolescent patient living at home with the family differs from the older patient living independently. Casper & Troiani (2001) described the different perceptions of family functional characteristics across diagnostic groups. In restrictive anorexia nervosa, as in controls, functional disturbance was not identified, whereas overt distress, negative affect and unresolved conflict were more likely to be recognised by people with purging anorexia nervosa and their families. Again, Webster & Palmer (2000) found that in terms of adverse childhood experiences, women with bulimia nervosa tended to report rates similar to those reported by women with a diagnosis of major depression. Women with anorexia nervosa resembled a non-morbid

comparison group. Caution should be exercised in assuming that research findings for one diagnostic group can easily be extrapolated to another.

Nevertheless, eating disorders do aggregate in families. Rates of anorexia and bulimia nervosa are higher in the first-degree relatives of index patients (Strober *et al*, 2000). Genetic factors are suggested to account for up to half of the variance in eating disorders; however, more work is needed and this figure is probably overestimated. Most would agree that environmental and genetic factors interplay. Of interest is the extent to which non-shared environmental factors, such as disparate parental treatment or peer group characteristics, influence the differential expression of eating disorders in siblings (Klump *et al*, 2002).

A contribution by families to the causation of eating disorders has face validity and is supported by clinical observation and by research, yet it is not easily explained in terms of mechanisms of intergenerational transmission. General parental factors have been explored, as well as the influence of maternal eating disorders upon offspring. Steiger *et al* (1996) demonstrated that parental psychopathological traits, such as affective instability and narcissism, and parental eating attitudes contribute to the aetiology of eating disorders. However, he suggests that intergenerational transmission of such familial traits is insufficient alone to account for the development of eating disorder in offspring, and other vulnerability factors are required.

Maternal concern about infant weight, as well as efforts to slim babies down, is recognised to occur in bulimia nervosa (Lacey & Smith, 1987). Stein *et al* (1994) suggested a relationship between eating disorder in mothers and disturbances in parenting, mother–infant interaction and infant development at age 1 year. Certain effects appeared to be specifically related to the maternal eating disorder: childhood disturbance either manifesting in the domain of eating and weight/shape concern, or arising as a direct consequence of maternal eating behaviour. However, general and non-specific effects were also noted. This cross-sectional study did not provide evidence of causality, nor did it identify the mechanism involved.

A prospective study of childhood eating disturbance (Agras *et al*, 1999; Stice *et al*, 1999) studied infants from birth to age 5 years, identifying early development of

†See pp. 210–215, this issue.

abnormal eating patterns. The daughters of a subgroup of women with eating disorders showed higher avidity for feeding than did controls. The authors propose that this could represent a risk factor for later eating disorder in the context of maternal concern for their daughters' weight and the use of food for non-nutritive purposes such as a behavioural reward. The study by Cooper *et al* (2004, this issue) examines family environmental factors that may potentially link childhood feeding problems and maternal eating disorder. In a path analysis, two environmental variables, 'mealtime disorganisation' and 'maternal strong control and disharmony', were shown to mediate the association between child and maternal disturbance.

Family dysfunction is only one paradigm of the aetiology of eating disorders; it is not essential for the development of these conditions but is a significant factor in a proportion of sufferers. Families, their dynamics and pathologies, may also be of importance to the recovery process. There is robust evidence for the efficacy of family therapy in eating disorders of early onset and short duration (Russell *et al*, 1987). Family therapy need not be restricted to the task of addressing family dysfunction, should it exist; it is a forum in which the affected individual and the family may prepare for change: the return to the family system of a newly autonomous person. The importance of engaging the family, together with the patient, in treatment cannot be overstated. In most individual clinical circumstances, to alienate the family is to undermine the possibility of therapeutic gain.

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(First received 15 July 2003, final revision 17 October 2003, accepted 27 October 2003)

A supportive, educational and non-critical approach, together with family therapy where indicated, is more likely to reap benefits.

DECLARATION OF INTEREST

None.

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