CAUSES OF EATING DISORDERS

Janet Polivy[1] and C. Peter Herman[2]

[1]Department of Psychology, University of Toronto, Erindale Campus, Mississauga, Ontario, Canada L5L 1C6; e-mail: polivy@psych.utoronto.ca

[2]Department of Psychology, University of Toronto, Toronto, Ontario, Canada M5S 1A1; e-mail: herman@psych.utoronto.ca

Key Words   anorexia nervosa, bulimia nervosa, contributory factors, identity, control

■ **Abstract**   Anorexia nervosa and bulimia nervosa have emerged as the predominant eating disorders. We review the recent research evidence pertaining to the development of these disorders, including sociocultural factors (e.g., media and peer influences), family factors (e.g., enmeshment and criticism), negative affect, low self-esteem, and body dissatisfaction. Also reviewed are cognitive and biological aspects of eating disorders. Some contributory factors appear to be necessary for the appearance of eating disorders, but none is sufficient. Eating disorders may represent a way of coping with problems of identity and personal control.

CONTENTS

INTRODUCTION

In the late 1960s, the previously obscure and extremely rare disorder anorexia nervosa (AN) became much more prevalent in Western societies. Young females from middle- and upper-class families were starving themselves, sometimes to death. The next decade saw the emergence of a new eating disorder, bulimia nervosa (BN), wherein young women alternated self-starvation with bingeing, usually followed by purging (i.e., active attempts to rid the body of calories). Clinicians and

the media focused intensively on these new disorders, which supplanted obesity as the primary eating disorder; indeed, obesity was removed from the Diagnostic and Statistical Manual (DSM) of Mental Disorders in the 1980s. Research on these new disorders grew at about the same rate as the disorders themselves. Some interesting historical research (Bemporad 1997) uncovered evidence of both AN and BN having existed since ancient times, but certainly not to the same extent—and possibly not in the same way—as at present. By the turn of the millennium, AN and BN were well-established—as was the research enterprise—but a clear understanding of the source of the disorders or how to prevent or treat them remained elusive.

In this chapter, we first describe the disorders; the bulk of the chapter is devoted to a review of research (and speculation) on the causes of eating disorders, emphasizing the discriminant validity of causal factors (i.e., why one person develops an eating disorder, whereas another seemingly similar person in a seemingly similar situation does not). One major area we do not review is treatment research; this decision is dictated mainly by space limitations, but also by the fact that studies on treatment are only loosely related to research on eating disorder's (ED) causes. Another arguable decision is to ignore binge eating disorder and eating disorder not otherwise specified, recently identified EDs; again, space limitations require strict focus on the major EDs.

DIAGNOSTIC CRITERIA AND CORE PATHOLOGICAL FEATURES

In this chapter we follow the convention of the research literature and consider AN and BN as separate disorders. How—or even whether—to separate the eating disorders, however, has been debated vigorously. The core symptoms (e.g., body dissatisfaction; preoccupation with food, weight, and shape; certain ego deficits) do not necessarily distinguish AN from BN patients, even if the diagnostic criteria for the two EDs differ. The spectrum hypothesis (VanderHam et al. 1997) considers the EDs as one syndrome with different manifestations.

According to the DSM-IV [American Psychiatric Association (APA) 1994], the diagnostic criteria for AN are maintaining a body weight at a level less than 85% of normal weight for age and height, an intense fear of fatness, disturbed experience of one's body weight or shape, and amenorrhea for at least three consecutive menstrual cycles. Although many AN patients engage in compulsive exercising, restrictor-type AN patients are distinguished by their resolute refusal to eat (much), whereas bulimic-type AN patients regularly engage in binge eating and purging. Some have questioned the wisdom of retaining amenorrhea as a diagnostic criterion (Cachelin & Maher 1998, Garfinkel et al. 1996); we could extend the argument to the 85% weight criterion.

The DSM-IV criteria for BN include recurrent episodes of both binge eating (i.e., eating a larger amount of food than most people would eat in a similar time

and circumstances, and a feeling of lack of control of one's eating during the episode) and compensatory behaviors (such as purging, exercising, or fasting) to prevent weight gain from the overeating. These behaviors must occur at least twice a week for a minimum of 3 months. In addition, self-evaluation is overly dependent on body weight and shape. Note that bingeing and purging are characteristics of one major type of AN, which leads to understandable confusion between AN and BN[1]; BN may differ from bulimic-type AN only in that BN patients are unable to suppress their weight below the 85% cut-off and thus fail to display amenorrhea. AN is an exclusionary criterion for BN, which otherwise might be confused with bulimic-type AN. A further subdivision of BN into purging-type (i.e., use of self-induced vomiting or laxative, diuretic, or enema abuse) and nonpurging type (i.e., fasting, exercising, or other nonpurging means of compensating for binge eating) would seem to differ only in the preferred tactic for eliminating calories and probably has little conceptual significance (APA 1994).

Although bingeing is a diagnostic feature of BN and bulimic-type AN, we have little guidance as to how to identify a binge (Herman & Polivy 1996). Exactly how much food is a larger-than-normal amount of food? How are we to assess the loss of control that allegedly characterizes binges (Johnson et al. 2000)? If an eating episode displayed only one aspect—a huge amount of food or loss of control but not both—would it still be a binge?

One correlative feature that distinguishes BN from AN is impulsivity. Sexual promiscuity, suicide attempts, drug abuse, and stealing or shoplifting are frequently noted in BN patients (e.g., Matsunaga et al. 2000; Wiederman & Pryor 1996). Indeed, impulsiveness may be what makes an aspiring anorexic into a bulimic; if an individual intent on restricting her intake cannot resist food under certain circumstances, she may capitulate to temptation, binge, and then feel obliged to compensate afterward. This pattern would seem to characterize both BN and bulimic-type AN patients, the only difference being the weight level around which they fluctuate.

Incidence and Prevalence

The incidence of AN and BN has increased markedly during the past 50 years, although there is some reason to believe that at least some of the increase is due to greater awareness and reporting of these disorders (Wakeling 1996). Precise estimates of incidence and prevalence vary wildly, perhaps because those who suffer from these disorders are often reluctant to reveal their condition. Prevalence estimates tend to range from about 3% to 10% of at-risk females (those between 15 and 29 years of age), with BN patients outnumbering AN patients by at least 2 to 1. Those with AN frequently deny any illness and are often seen for treatment only because of the concern of those close to them that their lives may be in

---

[1]Indeed, Gleaves et al. (2000) found that restrictor-type AN is more distinct from bulimic-type AN than bulimic-type AN is from BN. It has been argued that bulimic-type AN should simply be considered BN.

imminent danger. Those with BN, because their appearance is usually normal and their bingeing and purging occur in private, are usually more difficult to detect, although BN patients are more likely to present themselves for treatment, because the binge-purge cycle is often profoundly disturbing to them. (AN patients frequently appear to be indifferent to their disorder.) The prevalence of partial EDs is at least twice that of full-syndrome EDs. Longitudinal studies suggest a progression from less to more severe disturbances, with normal dieters occasionally becoming pathological dieters, who in turn occasionally progress to partial- or full-syndrome EDs (Shisslak et al. 1995). It must be remembered, though, that whereas normal dieting is a frequent precursor of EDs, most normal dieters do not progress to the point of pathology. One of the most significant questions facing us is why some dieters progress to EDs whereas most do not.

Prognosis

There is no easy treatment for EDs. About a third of patients continue to meet diagnostic criteria 5 years and longer after initial treatment (Fairburn et al. 2000, Keel et al. 1999). Estimates of mortality (including suicide) rates range from just over 5% to just over 8% (Herzog et al. 2000, Steinhausen et al. 2000). Still, more than 50% of patients show significant improvement more than 5 years after beginning treatment (Casper & Jabine 1996, Herpertzdahlmann et al. 1996, Steinhausen et al. 2000). Some research (e.g., Keel et al. 1999) has explored predictors of success in treatment, but there has been little investigation into natural recovery, self-cure, or remission of EDs without treatment. Perhaps only the most intractable cases find their way into treatment.

CAUSES OF EATING DISORDERS

The main purpose of this chapter is to survey the various attempts to explain why some people develop eating disorders. The literature displays an uneasy balance between studies exploring the role of particular putative causal factors and theories that attempt to combine such factors into a comprehensive whole. The main obstacles facing these attempts are, first and foremost, the virtual impossibility of conducting true experimental research in which a putative causal factor is manipulated, and secondly, the difficulty of combining all such factors into a model that is not unwieldy. A large proportion of studies examine AN and/or BN patients (and sometimes a healthy or psychiatrically impaired control group) with an eye to isolating correlates of the disorder, often in the vague hope that correlates can be persuasively argued into causes. The difficulty of finding suitable samples of ED patients to test has led to a proliferation of studies examining correlates of ED symptomatology as it exists in more-or-less normal populations. Any sample of high school or college females (and even males) will manifest a distribution of self-reported symptoms (albeit far short of true pathology), which may be leveraged

by extrapolation and the confusion of correlation and causation (especially in cross-sectional data) into examinations of the causes of EDs. The difficulty of doing proper research, along with the relative infancy of this field of research, should make us tolerant of these shortcomings. Still, the explanatory achievements to date are modest. Some theorists, such as Bruch (1973), have provided elegant theories; however, empirical data to confirm this and other theories are incomplete and inconclusive at this point.

Literally thousands of studies, plus numerous books and chapters, have attempted to specify exactly what causes EDs. The consensual approach to integrating the various factors that contribute to EDs is the "biopsychosocial" model. This model has the advantage of taking into account all sorts of factors—ranging from the broadly cultural to the narrowly biological, with stops along the way for familial, social, cognitive, learning, personality, and other factors—that have been alleged to make a difference (Leung et al. 1996). The model lacks specificity, however; moreover, each version of the biopsychosocial model differs from the next. Space limitations preclude an exhaustive review here, but we attempt to provide an overview of the most salient findings and issues. We begin at the broadest level with culture, proceed to familial and social factors, and then examine individual factors such as personality, cognition, and physiology.

Sociocultural Contributors to Eating Disorders

Eating disorders do not occur uniformly in all cultures at all times. An obsession with slimness—a core feature of EDs—is concentrated in cultures in which food is abundant. In cultures of scarcity, the ideal body shape is much more likely to be rotund, suggesting that ideals tend toward what is difficult to achieve. In this sense, then, a culture of caloric abundance may be considered a cause of EDs. It is important to note from the outset, however, that this cause is not specific; growing up in a culture of abundance, while perhaps increasing the chances of your developing an ED, does not make it likely that you will develop an ED; after all, most people in even the most affluent of cultures do *not* develop EDs. A culture of abundance should be regarded as at most a background cause. Such a culture may value slimness, but whether a particular individual takes this valuation to a pathological extreme depends on additional factors. For instance, there is variation in the extent to which people internalize our culture's valuation of slimness, and the extent of such internalization predicts body dissatisfaction, drive for thinness, and certain bulimic characteristics (Stormer & Thompson 1996, Stice 2001). What factors, though, determine the extent to which the value of thinness is internalized? We must refer to more individual factors (see below).

Initially, it was believed (see e.g., Garfinkel & Garner 1982) that the idealization of slimness, and the consequent tendency toward EDs, was concentrated in the upper-SES strata of the culture of abundance, where after all, abundance is even greater. As our culture becomes increasingly homogenized, with media images of a thin ideal physique now permeating every corner of society, EDs have

become correspondingly more democratic (Gard & Freeman 1996, Striegel-Moore 1997).

Not surprisingly, the media are often blamed for the (increasing) incidence of EDs, on the grounds that media images of idealized (slim) physiques motivate or even force people to attempt to achieve slimness themselves. The media are accused of distorting reality, in that the models and celebrities portrayed in the media are either naturally thin (i.e., at the tail of the normal distribution of body weight) and thus unrepresentative of normality, or unnaturally thin (i.e., the products of exceptional exertions to achieve and maintain a slim physique). As with the culture of abundance, idealized media images are at best a background cause of EDs. Exposure to the media is so widespread that if such exposure were *the* cause of EDs, then it would be difficult to explain why anyone would *not* be eating-disordered. Furthermore, as Tiggemann & Pickering (1996, p. 202) noted upon discovering that among girls, body dissatisfaction and drive for thinness were associated with increased exposure to certain types of TV shows, "although it is tempting to conclude that watching a large dose of thin idealized images on television leads to dissatisfaction with one's body, a correlation cannot determine causality. An alternative scenario, for example, might be that those most dissatisfied with their bodies or wishing to be thinner, seek out or are more interested in particular types of television."

The idealization of slimness and derogation of fatness in cultures of abundance is more intense for females than for males (Striegel-Moore 1993, 1997). This sex-linked valuation of thinness is usually invoked to account for the fact that EDs are more than 10 times more prevalent in females than in males (Striegel-Moore 1997). As a result of the societal disparagement of overweight and glorification of underweight, many—perhaps most—young women express dissatisfaction with their weight and shape. This dissatisfaction often has emotional overtones of self-disgust. Body dissatisfaction, in fact, may be regarded as an essential precursor (and continuing accompaniment) of EDs. The more intense this dissatisfaction, the more likely that one will undertake attempts to lose weight. When combined with other pathogens (see below), these attempts may well eventuate in AN (if the individual has particularly strong restraints on eating) or BN (if she does not).

Like the media, peer influence is often cited as a contributor to EDs (e.g., Levine et al. 1994, Shisslak et al. 1998, Stice 1998, Wertheim et al. 1997). Adolescent girls learn certain attitudes (i.e., the importance of slimness) and behaviors (i.e., dieting, purging) from their peers (Levine et al. 1994), both by example and encouragement and by way of teasing for failure to adhere to peer norms. Adolescent female friendship cliques tend to be homogenous with respect to body-image concerns (Paxton et al. 1999), suggesting direct peer influence; however, the possibility remains that cliques do not influence their members so much as "recruit" them on the basis of shared concerns (Ennett & Bauman 1994). It is difficult to weigh the relative importance of peer influence, as opposed, say, to the influence of the media or the family, which teach the same lessons; some evidence suggests that peers and family are more potent influences than the media (Stice 1998), whereas other

studies find the reverse (Wertheim et al. 1997). Moreover, peer influence, like these other influences, is so broad and pervasive that it ought to cause more pathology than actually occurs. Paxton et al.'s (1999) analysis reminds us that not all peers are equally concerned about attaining a slim physique, so blanket condemnation of peer influence or pressure is unwarranted.

Media and peer pressure no doubt impinge more powerfully on females than on males, but we should not be too complacent about explaining the huge disproportion of females among ED patients solely in terms of these influences. At the very least, we must consider the possibility that it is not simply that our culture exhorts females (more than males) to be thin; it may be the case that females are more attentive than are males to such exhortations, for various reasons that are examined below.

Among American women, blacks were thought to be "protected" from EDs owing to the reduced pressure on them to be thin. Black men prefer heavier women than do white men (see e.g., Greenberg & Laporte 1996), and black women (e.g., Powell & Kahn 1995) and children (Thompson et al. 1997) have larger ideal physiques. Thus, when black women develop an ED, it is more likely to be binge eating disorder, an ED that does not prominently feature a drive toward thinness (Striegel-Moore & Smolak 1996). Still, recent case reports (Striegel-Moore 1997; Striegel-Moore & Smolak 1996) suggest that the diffusion of the thin ideal has reached the black subculture as well. The widespread adoption of the dominant American cultural ideals (in American ethnic subcultures and indeed around the world) has meant that "ethnicity" no longer protects individuals from AN and BN (e.g., Chamorro & Flores-Ortiz 2000, Mumford & Choudry 2000, Polivy et al. 2001). Evidence that high-income black women are just as dissatisfied as are high-income white women suggests that whatever black-white differences may exist in population samples may be more a function of SES than of race (Caldwell et al. 1997).

The influence of sociocultural factors in the context of EDs can be summarized succinctly as the idealization of thinness, which is sometimes regarded as a principal cause of EDs on its own. More plausibly, it may channel women's dissatisfactions and distress toward a focus on body shape and size, providing an outlet for individual pathology; thinness is thus relentlessly pursued by those who see no better way to solve their problems. As we narrow our focus toward individual pathology, we now move to family influences.

Familial Influences on Eating Disorders

In what ways might families contribute to EDs? The most obvious way is by encouraging EDs. Families (and friends) often praise AN patients' slenderness, and envy the self-control and discipline required to achieve it (Branch & Eurman 1980); this reinforcement frequently persists even when the anorexic becomes severely emaciated. Of course, this reinforcement does not cause the disorder so much as help to perpetuate it. Certainly, people with AN do not require family

approval in order to starve themselves. In fact, those with AN as often as not use their families' increasing concerns about their inordinate slimness as a manipulative tool (Branch & Eurman 1980, Minuchin et al. 1978).

Family dynamics have been implicated not only in the perpetuation of EDs but also in their development (e.g., Minuchin et al. 1978). Case reports and studies of family interaction show eating-disordered families to be enmeshed, intrusive, hostile, and negating of the patient's emotional needs (Minuchin et al. 1978) or overly concerned with parenting (Shoebridge & Gowers 2000); some, such as Minuchin, have argued that the entire family unit must be treated if therapy is to be effective. Within the past few years, several studies have found that attachment processes are abnormal in eating-disordered populations; insecure attachment is common in this group (Ward et al. 2000a,b). Ward and colleagues (Ward et al. 2000b, p. 279), however, conclude that "many of these (family dysfunction) characteristics are regarded as secondary to the presence of an ill family member, rather than causative."

ED patients generally describe a critical family environment, featuring coercive parental control (Haworth-Hoeppner 2000). Adolescents who perceive family communication, parental caring, and parental expectations as low and those who report sexual or physical abuse are at increased risk for developing EDs (Haudek et al. 1999, Neumark-Sztainer et al. 2000). BN patients also report greater parental intrusiveness, specifically maternal invasion of privacy, jealousy, and competition, as well as paternal seductiveness (Rorty et al. 2000). In contrast, perceived parental encouragement of autonomy is associated with less dieting behavior (Strong & Huon 1998), possibly serving a protective function against EDs.

Mothers of girls with EDs may well have an influence on their daughters' pathology. They think that their daughters should lose more weight and describe them as less attractive than do comparison mothers or the girls themselves (Hill & Franklin 1998, Pike & Rodin 1991). Mothers of ED patients are more dissatisfied with the general functioning of the family system and are themselves more eating-disordered than are mothers of girls who do not have EDs (Hill & Franklin 1998, Pike & Rodin 1991). Direct maternal comments appear to be more powerful influences than is simple modeling of weight and shape concerns (Ogden & Steward 2000, Smolak et al. 1999), although even modeling does appear to affect elementary schoolchildren's weight and shape-related attitudes and behaviors (Smolak et al. 1999). Mothers' critical comments prospectively predicted ED outcome for their daughters (Vanfurth et al. 1996).

Mothers who themselves have an ED tend to have a negative influence on their children's attitudes and behaviors, feeding them irregularly, using food for nonnutritive purposes, and expressing concern about their daughters' weight as early as the age of 2. By 5 years of age, these children exhibit greater negative affect than do the offspring of mothers without EDs and are at serious risk for the later development of an ED (Agras et al. 1999). In fact, maternal EDs produce childhood feeding problems in offspring (Whelan & Cooper 2000), and 50% of children of mothers with EDs have psychiatric disorders (Hodes et al. 1997).

Most studies of family functioning are, predictably, correlational, making it difficult to determine whether family dysfunction contributes to EDs, EDs contribute to family dysfunction, or some common factor contributes to both. Moreover, the role of the family is often ascertained by retrospective questioning, further undermining our certainty about what caused what. Finally, case studies of dysfunctional/ eating-disordered families, in the absence of control families, preclude any certainty about whether these family problems are unique to families with a member who suffers from an ED.[2]

If we were to conclude that negative family influences were in fact responsible for the development of EDs, we would still need to ask exactly how a dysfunctional family induces EDs. Below, we consider the possibility that problems of identity and/or control are central to EDs, with the individual attempting to resolve these problems by investing emotionally and behaviorally in the pursuit of slimness. The family, of course, may contribute directly to problems of identity or control and may also suggest the solution, by emphasizing slimness as a panacea. Steiger et al. (1996) conclude that families (including so-called normal families as well) may transmit eating concerns, but such transmission may not be sufficient for the emergence of an ED, which requires "some additional vulnerability factor" (p. 156), either biological or experiential. This brings us to the consideration of individual factors that conduce to the development of EDs.

Individual Risk Factors

There are many factors specific to the individual that have been proposed as contributors to the development of EDs. Some of these factors (e.g., personality traits, self-esteem deficits) are seen as resident in the individual, whereas others involve personal experiences and seem to fall somewhere between the environment and the individual. The latter accordingly is examined first.

EXPERIENCES CONTRIBUTING TO EATING DISORDER DEVELOPMENT   Interpersonal experiences that have been most frequently linked to the development of EDs include abuse, trauma, and teasing. Self-reports of having been teased about one's appearance or body shape are associated with increased ED symptomatology

---

[2]A dramatically different approach to assessing familial influences on EDs is behavior genetic analysis, which includes common (familial) environment as a potential explanation of twin concordance. The Virginia group found a substantial familial influence on BN (Kendler et al. 1995) but later reversed itself, deciding that "the tendency to focus on common or familial environment as playing a primary causal role in the development of disordered eating is overemphasized" (Bulik et al. 1998, p. 1216). The often implausibly huge heritability coefficients for EDs (see below) leaves the skeptical reviewer disinclined to draw conclusions about family influence on the basis of genetics-vs.-environment models. Moreover, as Spelt & Meyer (1995) point out, the absence of common family environment effects in twin studies may simply mean that children react differently to family environment influences, not that such influences do not exist.

(Lunner et al. 2000). Similarly, ED patients report more premorbid life stresses and difficulties than do controls (e.g., Raffi et al. 2000, Schmidt et al. 1997, Welch et al. 1997). The joint occurrence (and possible mutual influence) of stressful life events and affective deficiencies such as low self-esteem, depressed mood, anhedonia, generalized anxiety, and irritability may be particularly pathogenic for BN (Raffi et al. 2000).

The influence of childhood abuse is somewhat more complex. There does appear to be a connection between childhood sexual abuse (CSA) and bulimic symptomatology (Everill & Waller 1995), although CSA is also associated with depression and other psychological disturbances. Not surprisingly, all of these problems are more likely to be associated with CSA in conjunction with other physical and emotional abuse and when the family does not provide support (deGroot & Rodin 1999). Some, however, maintain that BN patients do not have an elevated incidence of CSA, although the severity of the abuse may be greater (Groth-Marnat & Michel 2000). Casper & Lyubomirsky (1997) argue that sexual abuse causes BN only indirectly, by conducing to individual psychopathology. Kent & Waller (2000) maintain that childhood emotional abuse (CEA)—possibly through its profound influence on self-esteem and anxiety—is the only type of childhood trauma that predicts eating pathology in adults.

How might abuse lead to EDs? It has been argued abuse induces intolerable emotions and undermines identity. EDs serve as desperate attempts to regulate overwhelming negative affect and to construct a coherent sense of self when internal structures are lacking (Rorty & Yager 1996). Similarly, EDs have been seen as coping mechanisms favored by women who do not have more constructive ways of dealing with personal crises (Troop 1998).

How do EDs serve to deal with continuing emotional or identity problems? By refocusing one's attention onto weight, shape, and eating, one enters a domain in which one can gain some emotional control. The AN patient achieves at least partial emotional gratification by avoiding food and achieving slimness (albeit never enough). The BN patient gains emotional relief by bingeing (and then by purging). For both, obsessive focus on weight loss and its associated tactics provide a narrow, apparently viable way to channel identity concerns (and to avoid dealing with broader issues). More recent theorists concur that an extreme need to control both eating and other aspects of behavior is a central feature of EDs (Fairburn et al. 1999). Gaining a sense of control and pride in one's ability to control one's eating combats the feeling of being taken over by thoughts of food or of lacking control of one's thoughts, eating, and weight (Serpell et al. 1999). An experimental induction of loss of perceived control led ED patients to report feeling fatter and more pessimistic (Waller & Hodgson 1996).

AFFECTIVE INFLUENCES   Although a temperamental characteristic such as negative emotionality is unlikely to lead to EDs in and of itself, stress and negative mood are commonly reported antecedents for EDs (e.g., Ball & Lee 2000, Leon et al. 1997). For example, BN patients have elevated self-directed hostility scores, even controlling for other factors such as mood and family hostility level

(Friedman et al. 1997), and women with EDs score higher than controls in guilt, covert hostility (BN) (Allen et al. 1998), and suppressed anger (AN) (Geller et al. 2000). Furthermore, negative affect has been shown to mediate the relation between dieting and binge eating, although dieting and negative affect remain independent risk factors for binge eating (Stice et al. 2000a). It may be, though, that negative affectivity increases vulnerability for overall psychopathology rather than EDs per se (Leon et al. 1997). Longitudinal studies often find that although initial depressed mood and self-perceptions predict later ED symptomatology, none of these predictors remain significant if initial disordered eating is ruled out. Thus, affective factors may not be etiologically important for EDs, although they may be associated with subclinical eating problems (Wichstrom 2000).

A functional relation between negative affect and ED symptoms has been proposed (e.g., Steinberg et al. 1989). According to Johnson & Larson (1982), people with BN attempt to elevate their mood by eating; purging allows them to avoid gaining weight. Unfortunately, the bulimic eventually realizes that the binges are out of control, and begins to feel guilty and dread eating. Eating no longer provides relief, but rather induces negative emotions such as guilt. Purging may then relieve the guilt, as well as discharging anger. It is thus possible that purging eventually replaces bingeing as a means of tension reduction (Johnson & Larson 1982). In fact, BN patients report reduced anxiety and depression following a binge/purge episode (Sanftner & Crowther 1998, Steinberg et al. 1989). After bingeing in the laboratory, bulimics reported reduced anxiety, tension, and guilt, although depression was unchanged (Kaye et al. 1986). Bulimic behaviors also reduce anger, particularly when the individual has a strong tendency to avoid expressing that emotion (Milligan & Waller 2000).

The co-occurrence of ED and mood disorders (particularly depression) has been frequently noted and may reflect primary mood disturbance in EDs, mood disorders secondary to EDs, or common third variables (biological or psychosocial) leading to both, such as genetic or familial transmission. The lack of a well-organized body- and self-image is unique to EDs, however (Steiger et al. 1992). Some evidence supports the primacy of the ED; ED symptoms and dietary restraint predicted subsequent depression in initially nondepressed individuals (Stice et al. 2000b). Some studies find that ED onset follows that of mood disorder (Godart et al. 2000, Gruber & Dilsaver 1996), whereas still others suggest that depression and anxiety are more state-dependent features that resolve when ED symptoms remit (Lehoux et al. 2000). Actually inducing negative affect increases body dissatisfaction and body-size perception in BN patients (Carter et al. 1996; Kulbartz-Klatt et al. 1999), suggesting that whichever is primary, negative affect can contribute to ED symptoms.

SELF-ESTEEM   Low self-esteem may conduce to a variety of disorders, including EDs (e.g., Fairburn et al. 1997, Leary et al. 1995, Striegel-Moore 1997). Self-esteem reflects how others react to the individual; thus, (perceived) rejection may cause lower self-esteem and maladaptive behaviors, including EDs. Furthermore, dieting, which is highly prone to disruptions that result in overeating, often

produces a downward spiraling of self-esteem that contributes more specifically to EDs (as opposed to, say, depression) (Heatherton & Polivy 1992). Shape- and weight-based self-esteem is reduced in ED patients (Geller et al. 1998), and prospective research confirms that girls with low self-esteem are more likely to develop disordered eating in the next few years (Button et al. 1996).

Empirically, self-esteem has been found to moderate perfectionism and feeling overweight in predicting bulimic symptoms (Bardone et al. 2000, Vohs et al. 1999). Women high in perfectionism, who consider themselves overweight exhibit bulimic symptoms only if they have low self-esteem (i.e., if they doubt they can attain their high body standards); women with high self-esteem and the same diathesis-stress conditions are less likely to exhibit bulimic symptoms. Similarly, low self-esteem and negative affect predicted ED symptomatology 4 years later (Leon et al. 1997). Moreover, a program aimed at improving self-esteem in 11- to 14-year-olds resulted in lowered incidence of weight loss and ED symptomatology 1 year later in participants considered at risk when the program began (O'Dea & Abraham 2000). Finally, lower self-esteem in ED patients predicts worse outcome (Hesse-Biber et al. 1999, VanderHam et al. 1998). Alternatively, women who recover from BN report an increase in their self-esteem (Troop et al. 2000).

BODY DISSATISFACTION   Negative affect and negative feelings about the self are channeled in ED more specifically into negative feelings about the body or body dissatisfaction (BD).[3] BD is sometimes operationalized as the gap between one's actual and ideal weight/shape; however, this is inappropriate, because one may see one's body as far from ideal and yet still be reasonably satisfied with it. More direct assessment of BD involves asking people specifically how (dis)satisfied they are with their bodies or parts thereof.

Virtually all conceptualizations of EDs—including the DSM criteria—make reference to BD. Most models of EDs involving multiple factors (e.g., Stice 2001) assign BD a prominent causal role. BD (sometimes referred to as weight concern) is often associated with dieting behavior; indeed, BN and AN are sometimes referred to as the dieting disorders. Dieting has been posited to precipitate, if not cause, bingeing (Polivy & Herman 1985) and EDs in general (Polivy & Herman 1987; Stice 2000a, 2001), and it is BD that presumably causes dieting. Weight concern and dieting help to predict the emergence of an ED (e.g., Joiner et al. 1997, Steiger et al. 1996, Stice et al. 1998), and bulimic women seek out the

---

[3]The broader construct of "negative body image" comprises both body dissatisfaction and body misperception—typically, overestimation of one's actual body size. Although both types of body image problems can in principle drive weight loss attempts, and although they may well overlap empirically, it is possible to experience one and not the other (Stormer & Thompson 1996). For a time, misperception was considered a critical aspect of EDs and research on it flourished, but its importance has faded in recent years. ED-control differences in misperception are much weaker than are comparable differences in dissatisfaction (Cash & Deagle 1997).

very appearance feedback that is likely to aggravate these concerns (Joiner 1999). Moreover, such interest in negative appearance feedback predicts the development of later symptoms, via the mediating effects of increased body dissatisfaction. Conversely, satisfaction with one's weight acts as a "protective" factor in adolescent girls who are otherwise at high risk for developing EDs (Chandy et al. 1995).

Most of the alleged causes of EDs that we have reviewed operate through BD. Thus, media influence is thought to precipitate EDs by making women feel dissatisfied with their appearance. Family and peer pressure, teasing, and more individual psychological influences such as general anxiety converge on the "final common pathway" of BD (Paxton et al. 1999).

In our view, while BD is probably a necessary factor in the emergence of EDs, it is not sufficient. After all, it is possible to be dissatisfied with one's body and yet not do anything about it. Why is it that of two dissatisfied people, one throws herself into (usually futile) attempts to achieve a satisfactory body, whereas the other remains dissatisfied but does not diet/starve, binge, or purge? The determining factor, we suggest, is whether or not the individual seizes upon weight and shape as the answer to the problems of identity and control. Some young women become invested in achieving a "perfect" body as an existential project (i.e., as a way of giving their lives meaning, coherence, and emotional fulfillment that are otherwise lacking). Some become invested in achieving complete control over their eating, weight, and shape, believing that control in these domains is possible even though such control is not possible elsewhere in their lives. For many with EDs, these two goals overlap. In the final analysis, BD may contribute to EDs primarily by conferring purpose: The narrow ambitions of the ED patient—in particular, the exclusive focus on weight—may make her life simpler, more certain, and more efficacious. "She finds a maladaptive solution to her suffering, confusion, and sense of inadequacy by identifying herself with her weight" (Vitousek & Hollon 1990, p. 197).

COGNITIVE FACTORS   EDs feature several cognitive aberrations, including obsessive thoughts, inaccurate judgments, and rigid thinking patterns. The prominence of these has suggested that cognitive pathology may contribute to EDs. Schematicity for weight, and using one's weight as a basis for self-evaluation, are central features of both AN and BN and contribute to the persistence of EDs (Vitousek & Hollon 1990). An obsession with becoming thin may be seen as driving AN (and is present in BN as well). Judging one's own body to be larger than it really is may justify a relentless pursuit of thinness. Examination of cognitions in EDs has been stimulated by two developments: (*a*) the ascendancy of cognitive-behavior therapy for EDs, which is premised on the notion that normalizing cognitions may prove effective against EDs and (*b*) the importation of well-developed experimental paradigms for studying cognition.

Obsessive thoughts   Individuals with EDs tend to spend an inordinate amount of time obsessing about food/eating, weight/shape, and related matters (Gleaves

et al. 2000). One survey (Sunday et al. 1995) found that 74% of ED patients spent more than 3 hours/day on obsessional thoughts; 42% spent more than 8 hours/day! Sixty-two percent had fewer than 3 hours/day entirely free of such obsessive thoughts, and 37% had no free hours at all. Most (72%) patients tried to suppress these obsessive thoughts, but "50% were not successful and felt that they had little or no control over the preoccupations" (Sunday et al. 1995, p. 241). About 20% of the patients—especially restricting anorexics—found the obsessive thoughts comforting and did not wish to be rid of them; more than half the patients considered the obsessions to be ego-syntonic.

Perfectionism   Related to obsession is perfectionism, the belief that one must (strive to) be perfect. Perfectionism can easily be applied to eating, weight, and shape. Perfectionism is not a defining characteristic of eating disorders but it has long been thought to be involved in AN and, to a lesser extent, BN (Garner et al. 1983, 1984). Hewitt et al. (1995) argue that perfectionism can contribute to eating disorders by making normal shortcomings more traumatic or by making a normal body a sign of imperfection. The eating disorder inventory (EDI) (Garner et al. 1983) includes a perfectionism subscale.

AN patients display elevated scores on paper-and-pencil measures of perfectionism (Bastiani et al. 1995). The fact that some indices of perfectionism remain high even after weight restoration suggests that perfectionism may be a precursor of AN; this view accords with Strober's (1991) theory in which self-doubting perfectionism is one of a small number of characteristics that predispose one to AN. Bruch (1973) and Casper (1983) also accord perfectionism a causal role in AN. Recent debates contrasting self-imposed perfectionism and other-imposed perfectionism add further complexities to the analysis of EDs (Hewitt et al. 1995); whom are ANs trying to please, themselves or others?

Dissociation   One of the more profound psychological tactics used to escape unpleasant realities is to adopt the dynamic defense of dissociation, in which traumatic elements are split off from focal consciousness, which turns instead to something more tractable. In BN "immersion" in the binge may protect the individual from emotional distress (Heatherton & Baumeister 1991, Lacey 1986). Evidence for dissociative tendencies in EDs—as measured using the self-report Dissociative Experiences Scale (Bernstein & Putnam 1986)—is not very impressive, however. In their review Everill & Waller (1995) claim that "high levels of dissociative tendencies have been reported in eating-disordered women" (p. 4), but these same authors found that bulimic women display only a weak elevation of Dissociative Experiences Scale scores (Everill et al. 1995). Valdiserri & Kihlstrom conclude that "within the population of normal college-age women . . . there appears to be no particular relationship between abnormal eating and dissociative experiences" (1995, p. 149).

Cognitive style   ED patients display evidence of abnormal cognitive style or information processing. For instance, BNs are much quicker than are restricting ANs on the Matching Familiar Figures Test, reflecting their greater impulsiveness (Kaye

et al. 1995). Such aberrations might contribute to their disorder; Bruch (1973) argued that ANs engaged in unwarranted all-or-none thinking, which might lead them to regard themselves as failures after even minor infractions.

Cognitive bias   ED patients display aberrations in information processing and memory, especially for material related to weight, shape, and food. Although there are many controversies pertaining to technical issues, the consensus from studies assessing cognitive bias such as those using the modified Stroop (color-naming) (e.g., Green et al. 1999) and dot/visual probe (e.g., Rieger et al. 1998) paradigms is clear: BNs tend to show bias for weight/shape words (compared with control words), whereas ANs are more likely to show bias for food words. What is less clear is the value of this discovery. We already knew that ED patients have an emotional concern with weight and shape; this is a defining feature of the disorders. That normal dieters and possibly hungry people in general (Mogg et al. 1998) also show attentional bias to shape and food stimuli renders the findings in ED patients even less important.

Studies of memory bias make the same point, with ED patients showing differential memory for disorder-related material (e.g., Hermans et al. 1998, Sebastian et al. 1996). Even if it were established conclusively that ED patients are schematic for weight, shape, and food information, "it is, however, still unclear what role (if any) these schemata play in the etiology of anorexia nervosa" (Hermans et al. 1998, p. 199) or in BN. It seems just as likely that a preoccupation with food is a result as it is a cause of weight loss (see Herman & Polivy 1993).

BIOLOGICAL INFLUENCES   Because EDs so prominently involve appetite— apparent lack of appetite in the case of AN and apparently uncontrollable appetite in the case of BN—there is an understandable temptation to look for biological causes of these disorders. There are all sorts of biological abnormalities associated with EDs, some of which we review. Researchers in this area have for the most part shown commendable reluctance to promote biological correlates of EDs into candidate causes, probably because of repeated warnings that AN and BN both have the potential to disrupt appetitive and broader neuroendocrine systems, so that biological anomalies may be just as likely to be effects as causes. Indeed, many of the anomalies are well-known effects of starvation or stress, undermining their causal candidacies. The preferred approach to identifying biological (or any other class of potential) causes is to conduct prospective research in which a normal (pre-symptomatic) sample is assessed, permitting the identification of differential predictors of eventual ED, and avoiding the contaminating influence of the ED on the predictor. As Leon et al. (1997, p. 407) note, however, "the assessment of many types of biological risk factors is often too costly and invasive to be done in prospective studies with large nonclinical samples." (The same can be said for many nonbiological risk factors as well.)

Genetics   Twin and family studies provide evidence for the genetic transmission of EDs, although such evidence is not conclusive. For one thing, behavior genetics currently emphasizes environmental factors as much as genetic factors. (The

double irony of modern behavior genetics is that not only does it not necessarily focus on genetics, but it also rarely focuses on behavior. In the case of EDs, the criterion is usually either clinical diagnosis or questionnaire responses, both of which are only remotely related to actual behavior.) Family studies (see Spelt & Meyer 1995 for a review) indicate that EDs aggregate in families, but such studies cannot easily disentangle genetic from environmental transmission. Very high heritability coefficients—reflecting the relative importance of strictly genetic factors—have been claimed for both AN and BN. Klump et al.'s (2000) reading of the literature leads them to conclude that 50–83% of the variance in AN and BN is genetic. These estimates are based on twin studies, which may yield artifactually high heritability estimates, owing to contrast and assimilation effects and the skewing effect of nonadditive genetic variance (Plomin et al. 1990). More troubling, in our view, is the common finding that not only the disorder itself but also the associated attitudes (concerns, fears, and preoccupations) are highly heritable (Klump et al. 2000). We are prepared to concede a role for genetics in the regulation of physiological parameters that might in principle contribute to an ED; however, statistics notwithstanding, it is difficult to understand how genes influence these attitudes, which in turn influence the development of the disorder. We are confronted with analyses that purport to show *that* genes have a major influence on EDs, but that do not indicate *how*. A persuasive case for genetic influence awaits a clearer indication of the pathways from genes to (pathological) behavior, affect, and cognition (Allison 1997). As Hewitt (1997, p. 355) concludes, "although there is consistent evidence of genetic factors influencing vulnerability to eating disorders, the details are far from clear."

Neuroendocrine factors   Just as the former ED, obesity, was often explained as "glandular" in origin, so current EDs are often seen as explicable in terms of neuroendocrine dysfunction, which might or might not itself be genetic in origin. Such dysfunction might be primary (i.e., a hormonal aberration triggers the disorder) or mediational (i.e., stress or some other environmental factor disrupts hormonal functioning, which in turn affects eating). Because appetite is commonly regarded as responsive to hormonal controls, a neuroendocrine explanation for EDs is attractive. The major problem with such an explanation is that EDs are not simply disorders of appetite. Almost everyone sees AN as principally attributable to the relentless pursuit of thinness (RPT) that dictates restrictive food intake, which leads directly to AN. What causes RPT is debatable, but it is difficult to see how appetite suppression could precede RPT (unless you wanted to construct a convoluted scenario in which RPT develops as an ex post facto attitudinal justification for not eating). In the most prevalent view of BN, RPT and restrictive intake precedes bingeing, and bingeing is simply a breakdown of restraint (e.g., Polivy & Herman 1985, Stice et al. 1998). An alternative view of BN regards bingeing as primary and restrictive intake as secondary and compensatory (e.g., Bulik et al. 1997, Mussell et al. 1997); this minority view might possibly be compatible with a primary neuroendocrine disorder, but it does not easily accommodate the centrality of RPT in BN.

One notable feature of neuroendocrine accounts of EDs is that AN and BN are seen as virtual opposites, whereas most other theories regard them as more alike than different. The neuroendocrine approach emphasizes starving (AN) versus bingeing (BN). What factors might go awry to produce the extremes of normal appetite?

One possibility would be primary dysfunction of the hypothalamus, the "seat" of appetite. The evidence that AN is due to a disturbance of the hypothalamus, either structural or functional, is weak. "Although structural lesions involving the hypothalamus can lead to cachectic states, . . . a syndrome that fulfills strict criteria for AN is excessively rare. . . . there is no good evidence for a primary hypothalamic disorder in AN in general, although this possibility is not excluded" (Study Group on Anorexia Nervosa [SGAN] 1995, p. 239). The SGAN indicates (p. 237) that "a combination of elevated CRH (corticotropin-releasing hormone) and elevated AVP (arginine vasopressin) may have a key role in sustaining the reduced food intake that is characteristic of the disorder," but note the careful wording: the hormonal abnormality is characterized as "sustaining" the reduced intake, not causing it.

Although tests of hormonal functioning and evidence of hormonal aberrations in AN are both prevalent, the balance of opinion is that these aberrations are not what cause the disorder (Leung et al. 1996). "In fact, refeeding alone, leading to consistent weight gain and balanced nutrition, reverses the endocrine changes observed in AN" (SGAN 1995, p. 238).

One popular neurochemical candidate cause for EDs is serotonin (Brewerton 1995). Might ANs suffer from increased 5-Hydroxytryptamine (HT) activity, which would be consistent with their generally obsessive-compulsive approach to life, even after recovery from AN (SGAN 1995)? Both AN and obsessive-compulsive disorder (OCD) are responsive to fluoxetine, leading some to speculate that AN might simply be a variant of OCD (Pryor et al. 1995). By contrast, BN has been interpreted in terms of a 5-HT deficit (and a possible connection to both impulsivity and seasonal affective disorder). Recovered BNs show persistent abnormalities related to serotonin function, suggesting that such abnormalities may underlie the development of BN (Kaye et al. 1998). Serotonin imbalance has more surface appeal than most other biological hypotheses of ED, but the case is not yet strong. Recovered ANs do not show persistent 5-HT anomalies (O'Dwyer et al. 1996), leading Ward et al. (2000c) to conclude that "an abnormality of serotonin regulation is unlikely to be a major vulnerability factor in AN" (p. 283). Moreover, the excess/AN vs. deficit/BN explanation would seem to have difficulty accounting for alternating restriction and bingeing, which occurs to some extent in BN and more dramatically in bulimic-type AN.

Other biologically based factors   One interesting theme in much of the research is that biological factors can act to perpetuate a disorder that might not initially have been biological in origin. For instance, very-low-weight AN patients show olfactory impairment; such impairment is in all probability a consequence of starvation, but may act to diminish the appeal of food (Fedoroff et al. 1995). Similarly,

food may not appeal to ANs because it loses its "incentive-value"; this decline in incentive value may be a consequence of ironic aversive taste conditioning in food-deprived individuals (Pinel et al. 2000). Gastric distress and bloating, commonly reported in EDs, may also make eating aversive (e.g., Krahn et al. 1996).

Addiction models of EDs have been proposed (Wilson 1991). In the case of BN, the emphasis is on craving. Just as an addict craves a drug, BNs are postulated to experience intense food cravings, which may be due to exaggerated cephalic phase responses to food cues and/or distress, which has been associated with overeating in the past (e.g., Wilson 1991, Woods & Brief 1988). ANs may be "addicted" to the body's endogenous opioids, which are released in self-starvation accompanied by excessive exercise (Davis & Claridge 1998). Wilson dismisses the addiction model because of the lack of evidence for an addictive personality; the model does not address the core clinical characteristics of EDs (such as RPT) or the identified underlying psychopathology (such as low self-esteem, interpersonal distrust, and feelings of ineffectiveness), and it fails to account for psychobiological connections between dieting and EDs. Like Wilson, we are reluctant to apply the addiction metaphor to EDs.

Lack of internal awareness   Lack of interoceptive awareness (LIA) refers to an inability to accurately identify internal states or feelings, including physiological states (such as hunger and satiety) and emotional states (Garner et al. 1983). (Note that the problem is not an inability to detect internal states, but rather an inability to identify them.) It has long been argued (since at least Bruch 1969) that defective interoceptive awareness may produce confusion, so that the individual misinterprets her emotions as hunger, with the result that emotional arousal may trigger eating or even bingeing. By the same token, it seems possible that genuine hunger may not be perceived even when it is there (AN?), just as it may be perceived when it is not there (BN?).

There is little question that ED patients suffer from LIA (as reflected in its incorporation into the EDI). Questions remain, though, as to whether LIA is a cause or a consequence of EDs, and whether it should be regarded as a biological, perceptual, or cognitive problem. Bruch (1973) regarded LIA as a consequence of faulty learning beginning in infancy, when caretakers do not provide food merely to assuage hunger, but in response to all expressions of distress. Bruch further theorized that such faulty learning undermines trust in one's body and increases the need to control both the body and the self, which are seen as intertwined.

CONCLUSIONS

Reviewing the literature on EDs leaves us with many questions about how these disorders develop. To some extent, this may be unavoidable; research on clinical syndromes is of necessity mostly nonexperimental, and nonexperimental data preclude causal inferences. Unfortunately, many researchers lose sight of this

limitation in their efforts to explain these syndromes. Moreover, the interaction of etiological factors in a complex behavioral syndrome such as EDs is difficult if not impossible to capture. There are so many possible influences that their particular combination in any given individual becomes almost unique, and thus impractical to generalize to others. Finally, much of the literature consists of atheoretical attempts to measure and correlate particular researchers' favorite variables, rather than attempting to test etiological hypotheses about EDs. This is not to say that there are not theoreticians doing systematic studies testing conceptual views of the disorder, but such research is often overshadowed by the myriad studies in this area that do not rely on theoretical underpinnings. The noise-to-signal ratio in the literature is thus higher than one would like.

Eating disorders did not emerge as a serious problem until the late 1970s, so perhaps it is not surprising that a mere 25 years later we still seem to be so far from understanding their etiology. In fact, how much do we really know about the causes of other, longer-studied disorders such as depression? As with the EDs, we know that there is a biological/genetic contribution in at least some, though not all, depressives, and that life stresses along with psychological factors such as low self-esteem also make a contribution. We are still incapable, though, of predicting who will develop depression, when, and why other at-risk individuals will be spared. The literature suggests that EDs result similarly from the convergence of several facilitating factors, but the causal mechanisms are not yet identified. No single agent seems to be sufficient, but we may perhaps distinguish among stronger and weaker contributory factors.

What factors appear to be most necessary for the development of EDs? It is difficult to imagine developing an ED without the presence of body dissatisfaction, although the majority of individuals who are dissatisfied with their bodies will never go on to develop an ED. Similarly, negative emotion (such as depression or anxiety) and markedly low self-esteem are prominent features of EDs, and seem to be virtually invariant precursors of their development. Other elements that appear to be strongly implicated in the development of EDs include environmental stressors (usually the triggering factor) and cognitive distortions such as obsessive thoughts (e.g., that one is too fat). Finally, personality features such as a need for control (over oneself or one's body) and inadequate identity formation have been plausibly suggested as being necessary for the development of an ED.

What about the other risk factors that have received so much research attention? Sociocultural pressure to be thin, family influences such as criticism or enmeshment, dieting, biological contributors, genetic predisposition, personality variables such as perfectionism, dissociation, maturity fears, and interpersonal distrust all appear to contribute to the development of EDs, and the more of them impinging on an individual, the greater the risk. Some or all of these may contribute to the features delineated in the preceding paragraph, or they may contribute in their own right. Although it may turn out that one or more of these factors will be shown to be a prime cause of EDs, for the moment their connection seems less central, although clearly this is a judgment call.

Establishing which factors are more tightly linked to EDs does not bring us closer to understanding the mechanism underlying EDs. On the one hand, we are left with the question of what produces these causal factors (e.g., body dissatisfaction, identity deficits) in the first place; on the other, establishing the connection does not explain the process of going from the cause (body dissatisfaction, identity deficits) to the effects (EDs). Arguably, effective treatment does not require a full understanding of mechanisms. (We can treat headaches with aspirin without understanding either where the headache came from or how aspirin works.) Still, as scientists, our first obligation must be understanding. Our review indicates that we are a long way from understanding EDs. Constraints on research in this field will not make it easy to achieve empirical or conceptual progress. As with other psychological problems, we may have to be satisfied with recognizing contributory risk factors and devising therapies to help alleviate the discomfort, without conclusive proof of exactly what causes the disorder in any individual.

Visit the Annual Reviews home page at www.AnnualReviews.org

LITERATURE CITED

Agras S, Hammer L, McNicholas F. 1999. A prospective study of the influence of eating–disordered mothers on their children. *Int. J. Eat. Disord.* 25:253–62

Allen FCL, Scannell ED, Turner HR. 1998. Guilt and hostility as coexisting characteristics of bulimia nervosa. *Aust. Psychol.* 33:143–47

Allison D. 1997. Issues in mapping genes for eating disorders. *Psychopharmacol. Bull.* 33:359–68

Am. Psychiatr. Assoc. 1994. *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC: Am. Psychiatr. Assoc. 4th ed.

Ball K, Lee C. 2000. Relationships between psychological stress, coping and disordered eating: a review. *Psychol. Health* 14:1007–1035

Bardone AM, Vohs KD, Abramson LY, Heatherton TF, Joiner TE. 2000. The confluence of perfectionism, body dissatisfaction, and low self-esteem predicts bulimic symptoms: clinical implications. *Behav. Ther.* 31:265–80

Bastiani AM, Rao R, Weltzin T, Kaye WH. 1995. Perfectionism in anorexia nervosa. *Int. J. Eat. Disord.* 17:147–52

Bemporad JR. 1997. Cultural and historical aspects of eating disorders. *Theor. Med.* 18:401–20

Bernstein EM, Putnam FW. 1986. Development, reliability, and validity of a dissociation scale. *J. Nerv. Ment. Dis.* 174:727–34

Branch CHH, Eurman LJ. 1980. Social attitudes toward patients with anorexia nervosa. *Am. J. Psychiatry* 137:631–32

Brewerton TD. 1995. Review: toward a unified theory of serotonin dysregulation in eating and related disorders. *Psychoneuroendocrinology* 20:561–90

Bruch H. 1969. Hunger and instinct. *J. Nerv. Ment. Dis.* 149:91–114

Bruch H. 1973. *Eating Disorders: Obesity, Anorexia Nervosa and the Person Within*. New York: Basic Books

Bulik CM, Sullivan PF, Carter FA, Joyce PR. 1997. Initial manifestations of disordered eating behavior: dieting versus bingeing. *Int. J. Eat. Disord.* 22:195–201

Bulik CM, Sullivan PF, Kendler KS. 1998. Heritability of binge-eating and broadly defined

bulimia nervosa. *Biol. Psychiatry* 44:1210–18

Button EJ, Sonugabarke EJS, Davies J, Thompson M. 1996. A prospective study of self-esteem in the prediction of eating problems in adolescent schoolgirls: questionnaire findings. *Br. J. Clin. Psychol.* 35:193–203

Cachelin FM, Maher BA. 1998. Is amenorrhea a critical criterion for anorexia nervosa? *J. Psychosom. Res.* 44:435–40

Caldwell MB, Brownell KD, Wilfley DE. 1997. Relationship of weight, body dissatisfaction, and self-esteem in African American and white female dieters. *Int. J. Eat. Disord.* 22:127–30

Carter FA, Bulik CM, Lawson RH, Sullivan PF, Wilson JS. 1996. Effect of mood and food cues on body image in women with bulimia and controls. *Int. J. Eat. Disord.* 20:65–76

Cash TF, Deagle EA. 1997. The nature and extent of body-image disturbances in anorexia nervosa and bulimia nervosa: a meta-analysis. *Int. J. Eat. Disord.* 22:107–25

Casper RC. 1983. Some provisional ideas concerning the psychologic structure in anorexia nervosa and bulimia. In *Anorexia Nervosa: Recent Developments in Research*, ed. PL Darby, PE Garfinkel, DM Garner, DV Coscina, pp. 387–92. New York: Liss

Casper RC, Jabine LN. 1996. An eight-year follow-up: outcome from adolescent compared to adult onset anorexia nervosa. *J. Youth Adolesc.* 25:499–517

Casper RC, Lyubomirsky S. 1997. Individual psychopathology relative to reports of unwanted sexual experiences as predictor of a bulimic eating pattern. *Int. J. Eat. Disord.* 21:229–36

Chamorro R, Flores-Ortiz Y. 2000. Acculturation and disordered eating patterns among Mexican American women. *Int. J. Eat. Disord.* 28:125–29

Chandy JM, Harris L, Blum RW, Resnick MD. 1995. Female adolescents of alcohol misusers: disordered eating features. *Int. J. Eat. Disord.* 17:283–89

Davis C, Claridge G. 1998. The eating disorders as addiction: a psychobiological perspective. *Addict. Behav.* 23:463–75

deGroot J, Rodin G. 1999. The relationship between eating disorders and childhood trauma. *Psychiatr. Ann.* 29:225–29

Ennett ST, Bauman KE. 1994. The contribution of influence and selection to adolescent peer group homogeneity: the case of adolescent cigarette smoking. *J. Pers. Soc. Psychol.* 67:653–63

Everill JT, Waller G. 1995. Reported sexual abuse and eating psychopathology: a review of the evidence for a causal link. *Int. J. Eat. Disord.* 18:1–11

Everill JT, Waller G, Macdonald W. 1995. Dissociation in bulimic and non-eating-disordered women. *Int. J. Eat. Disord.* 17:127–34

Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. 2000. The natural course of bulimia nervosa and binge eating disorder in young women. *Arch. Gen. Psychiatry* 57:659–65

Fairburn CG, Shafran R, Cooper Z. 1999. Invited essay: a cognitive behavioural theory of anorexia nervosa. *Behav. Res. Ther.* 37:1–13

Fairburn CG, Welch SL, Doll HA, Davies BA, O'Connor ME. 1997. Risk factors for bulimia nervosa—a community-based case-control study. *Arch. Gen. Psychiatry* 54:509–17

Fedoroff IC, Stoner SA, Andersen AE, Doty RL, Rolls BJ. 1995. Olfactory dysfunction in anorexia nervosa. *Int. J. Eat. Disord.* 18:71–77

Friedman MA, Wilfley DE, Welch RR, Kunce JT. 1997. Self-directed hostility and family functioning in normal-weight bulimics and overweight binge eaters. *Addict. Behav.* 22:367–75

Gard MCE, Freeman CP. 1996. The dismantling of a myth: a review of eating disorders and socioeconomic status. *Int. J. Eat. Disord.* 20:1–12

Garfinkel PE, Garner DM. 1982. *Anorexia Nervosa: A Multidimensional Perspective*. New York: Brunner/Mazel

Garfinkel PE, Lin E, Goering P, Spegg C,

Goldbloom D, et al. 1996. Should amenorrhoea be necessary for the diagnosis of anorexia nervosa? Evidence from a Canadian community sample. *Br. J. Psychiatry* 168:500–6

Garner DM, Olmsted MP, Polivy J. 1983. Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *Int. J. Eat. Disord.* 2:15–34

Garner DM, Olmsted MP, Polivy J, Garfinkel PE. 1984. Comparison between weight preoccupied women and anorexia nervosa. *Psychosom. Med.* 46:255–66

Geller J, Cockell SJ, Goldner EM. 2000. Inhibited expression of negative emotions and interpersonal orientation in anorexia nervosa. *Int. J. Eat. Disord.* 28:8–19

Geller J, Johnston C, Madsen K, Goldner EM, Remick RA, Birmingham CL. 1998. Shape- and weight-based self-esteem and the eating disorders. *Int. J. Eat. Disord.* 24:285–98

Gleaves DH, Lowe MR, Snow AC, Green BA, Murphy-Eberenz KP. 2000. Continuity and discontinuity models of bulimia nervosa: a taxometric investigation. *J. Abnorm. Psychol.* 109:56–68

Godart NT, Flament MF, Lecrubier Y, Jeammet P. 2000. Anxiety disorders in anorexia nervosa and bulimia nervosa: co-morbidity and chronology of appearance. *Eur. Psychiatry* 15:38–45

Green M, Corr P, DeSilva L. 1999. Impaired color naming of body shape-related words in anorexia nervosa: affective valence or associative priming? *Cogn. Ther. Res.* 23:413–22

Greenberg DR, Laporte DJ. 1996. Racial differences in body type preferences of men for women. *Int. J. Eat. Disord.* 19:275–78

Groth-Marnat G, Michel N. 2000. Dissociation, comorbidity of dissociative disorders, and childhood abuse in a community sample of women with current and past bulimia. *Soc. Behav. Pers.* 28:279–92

Gruber NP, Dilsaver SC. 1996. Bulimia and anorexia nervosa in winter depression: lifetime rates in a clinical sample. *J. Psychiatry Neurosci.* 21:9–12

Haudek C, Rorty M, Henker B. 1999. The role of ethnicity and parental bonding in the eating and weight concerns of Asian-American and Caucasian college women. *Int. J. Eat. Disord.* 25:425–33

Haworth-Hoeppner S. 2000. The critical shapes of body image: the role of culture and family in the production of eating disorders. *J. Marriage Fam.* 62:212–27

Heatherton TF, Baumeister RF. 1991. Binge-eating as escape from self-awareness. *Psychol. Bull.* 110:86–108

Heatherton TF, Polivy J. 1992. Chronic dieting and eating disorders: a spiral model. In *The Etiology of Bulimia: The Individual and Familial Context*, ed. J Crowther, SE Hobfall, MAP Stephens, DL Tennenbaum, pp. 133–55. Washington, DC: Hemisphere

Herman CP, Polivy J. 1993. Mental control of eating: excitatory and inhibitory food thoughts. In *Handbook of Mental Control*, ed. DM Wegner, JW Pennebaker, pp. 491–505. Englewood Cliffs, NJ: Prentice-Hall

Herman CP, Polivy J. 1996. What does abnormal eating tell us about normal eating? In *Food Choice*, *Acceptance*, *and Consumption*, ed. H Meiselman, H MacFie, pp. 207–38. London: Blackie Acad. Prof.

Hermans DG, Pieters G, Eelen P. 1998. Implicit and explicit memory for shape, body weight, and food-related words in patients with anorexia nervosa and nondieting controls. *J. Abnorm. Psychol.* 107:193–202

Herpertzdahlmann B, Wewetzer C, Henninghausen K, Remschmidt H. 1996. Outcome, psychosocial functioning, and prognostic factors in adolescent anorexia nervosa as determined by prospective follow-up assessment. *J. Youth Adolesc.* 25:455–71

Herzog DB, Greenwood DN, Dorer DJ, Flores AT, Ekeblad ER, et al. 2000. Mortality in eating disorders: a descriptive study. *Int. J. Eat. Disord.* 28:20–26

Hesse-Biber S, Marino M, Watts-Roy D. 1999. A longitudinal study of eating disorders among college women—factors that influence recovery. *Gender Soc.* 13:385–408

Hewitt JK. 1997. Behavior genetics and eating

disorders. *Psychopharmacol. Bull.* 33:355–58

Hewitt PL, Flett GL, Ediger E. 1995. Perfectionism traits and perfectionistic self-presentation in eating disorder attitudes, characteristics, and symptoms. *Int. J. Eat. Disord.* 18:317–26

Hill AJ, Franklin JA. 1998. Mothers, daughters and dieting: investigating the transmission of weight control. *Br. J. Clin. Psychol.* 37:3–13

Hodes M, Timimi S, Robinson P. 1997. Children of mothers with eating disorders: a preliminary study. *Eur. Eat. Disord. Rev.* 5:11–24

Johnson C, Larson R. 1982. Bulimia: an analysis of moods and behavior. *Psychosom. Med.* 44:341–51

Johnson WG, Boutelle KN, Torgrud L, Davig JP, Turner S. 2000. What is a binge? The influence of amount, duration, and loss of control criteria on judgments of binge eating. *Int. J. Eat. Disord.* 27:471–79

Joiner TE. 1999. Self-verification and bulimic symptoms: Do bulimic women play a role in perpetuating their own dissatisfaction and symptoms? *Int. J. Eat. Disord.* 26:145–51

Joiner TE, Heatherton TF, Rudd MD, Schmidt NB. 1997. Perfectionism, perceived weight status, and bulimic symptoms: two studies testing a diathesis-stress model. *J. Abnorm. Psychol.* 106:145–53

Kaye WH, Bastiani AM, Moss H. 1995. Cognitive style of patients with anorexia nervosa and bulimia nervosa. *Int. J. Eat. Disord.* 18:287–90

Kaye WH, Greeno CG, Moss H, Fernstrom J, Fernstrom M. 1998. Alterations in serotonin activity and psychiatric symptoms after recovery from bulimia nervosa. *Arch. Gen. Psychiatry* 55:927–35

Kaye WH, Gwirtzman HE, George DT, Weiss SR, Jimerson DC. 1986. Relationship of mood alterations to bingeing behavior in bulimia. *Br. J. Psychiatry* 149:479–85

Keel PK, Mitchell JE, Miller KB, Davis TL, Crow SJ. 1999. Long-term outcome of bulimia nervosa. *Arch. Gen. Psychiatry* 56:63–69

Kendler KS, Walters EE, Neale MC, Kessler RC, Heath AC, Eaaves LJ. 1995. The structure of the genetic and environmental risk factors for six major psychiatric disorders in women. *Arch. Gen. Psychiatry* 52:374–83

Kent A, Waller G. 2000. Childhood emotional abuse and eating psychopathology. *Clin. Psychol. Rev.* 20:887–903

Klump K, McGue M, Iacono WG. 2000. Age differences in genetic and environmental influences on eating attitudes and behaviors in preadolescent and adolescent female twins. *J. Abnorm. Psychol.* 109:239–51

Krahn D, Kurth C, Nairn K, Redmond L, Drewnowski A, Gomberg E. 1996. Dieting severity and gastrointestinal symptoms in college women. *J. Am. Coll. Health* 45:67–71

Kulbartz-Klatt YJ, Florin I, Pook M. 1999. Bulimia nervosa: mood changes do have an impact on body width estimation. *Br. J. Clin. Psychol.* 38:279–87

Lacey JH. 1986. Pathogenesis. In *Current Approaches: Bulimia Nervosa*, ed. LJ Downey, JC Malkin, pp. 17–26. Birmingham, Engl.: Dupar Med.

Leary MR, Schreindorfer LS, Haupt AL. 1995. The role of low self-esteem in emotional and behavioral problems: Why is low self-esteem dysfunctional? *J. Soc. Clin. Psychol.* 14:297–314

Lehoux PM, Steiger H, Jabalpurlawa S. 2000. State/trait distinctions in bulimic syndromes. *Int. J. Eat. Disord.* 27:36–42

Leon GR, Keel PK, Klump KL, Fulkerson JA. 1997. The future of risk factor research in understanding the etiology of eating disorders. *Psychopharmacol. Bull.* 33:405–11

Leung F, Geller J, Katzman M. 1996. Issues and concerns associated with different risk models for eating disorders. *Int. J. Eat. Disord.* 19:249–56

Levine MP, Smolak L, Moodey AF, Shuman MD, Hessen LD. 1994. Normative developmental challenges and dieting and eating disturbances in middle school girls. *Int. J. Eat. Disord.* 15:11–20

Lunner K, Werthem EH, Thompson JK, Paxton SJ, McDonald F, Halvaarson KS. 2000.

A cross-cultural examination of weight-related teasing, body image, and eating disturbance in Swedish and Australian samples. *Int. J. Eat. Disord.* 28:430–35

Matsunaga H, Kiriike N, Iwasaki Y, Miyata A, Matsui T. 2000. Multi-impulsivity among bulimic patients in Japan. *Int. J. Eat. Disord.* 27:348–52

Milligan RJ, Waller G. 2000. Anger and bulimic psychopathology among nonclinical women. *Int. J. Eat. Disord.* 28:446–50

Minuchin S, Rosman BL, Baker L. 1978. *Psychosomatic Families: Anorexia Nervosa in Context*. Cambrige, MA: Harvard Univ. Press

Mogg K, Bradley BP, Hyare H, Lee S. 1998. Selective attention to food-related stimuli in hunger: Are attentional biases specific to emotional and psychopathological states, or are they also found in normal drives states? *Behav. Res. Ther.* 36:227–37

Mumford DB, Choudry IY. 2000. Body dissatisfaction and eating attitudes in slimming and fitness gyms in London and Lahore: a cross-cultural study. *Eur. Eat. Disord. Rev.* 8:217–24

Mussell MP, Mitchell JE, Fenna CJ, Crosby RD, Miller JP, Hoberman HM. 1997. A comparison of onset of binge eating versus dieting in the development of bulimia nervosa. *Int. J. Eat. Disord.* 21:353–60

Neumark-Sztainer D, Story M, Hannan PJ, Beuhring T, Resnick MD. 2000. Disordered eating among adolescents: associations with sexual/physical abuse and other familial/psychosocial factors. *Int. J. Eat. Disord.* 28:249–58

O'Dea JA, Abraham S. 2000. Improving the body image, eating attitudes, and behaviors of young male and female adolescents: a new educational approach that focuses on self-esteem. *Int. J. Eat. Disord.* 28:43–57

O'Dwyer AM, Lucey JV, Russell GF. 1996. Serotonin activity in anorexia nervosa after long-term weight restoration: response to D-fenfluramine challenge. *Psychol. Med.* 26:353–59

Ogden J, Steward J. 2000. The role of the mother-daughter relationship in explaining weight concern. *Int. J. Eat. Disord.* 28:78–83

Paxton SJ, Schutz HK, Wertheim ES, Muir SL. 1999. Friendship clique and peer influences on body image concerns, dietary restraint, extreme weight-loss behaviors, and binge eating in adolescent girls. *J. Abnorm. Psychol.* 108:255–66

Pike KM, Rodin J. 1991. Mothers, daughters, and disordered eating. *J. Abnorm. Psychol.* 100:198–204

Pinel JPJ, Assanand S, Lehman DR. 2000. Hunger, eating, and ill health. *Am. Psychol.* 55:1105–16

Plomin R, Chipuer HM, Loehlin JC. 1990. Behavioral genetics and personality. In *Handbook of Personality Theory and Research*, ed. LA Pervin, pp. 225–43. New York: Guilford

Polivy J, Herman CP. 1985. Dieting and binging: a causal analysis. *Am. Psychol.* 40:193–201

Polivy J, Herman CP. 1987. The diagnosis and treatment of normal eating. *J. Consult. Clin. Psychol.* 55:635–44

Polivy J, Herman CP, Mills J, Wheeler HB. 2001. Eating disorders in adolescence. In *The Blackwell Handbook of Adolescence*, ed. G Adams, M Berzonsky. Oxford: Blackwell. In press

Powell AD, Kahn AS. 1995. Racial differences in women's desires to be thin. *Int. J. Eat. Disord.* 17:191–95

Pryor TL, Martin RL, Roach N. 1995. Obsessive-compulsive disorder, trichotollimania, and anorexia nervosa: a case report. *Int. J. Eat. Disord.* 18:375–79

Raffi AR, Rondini M, Grandi S, Fava GA. 2000. Life events and prodromal symptoms in bulimia nervosa. *Psychol. Med.* 30:727–31

Rieger E, Schotte DE, Touyz SW, Beumont PJV, Griffiths R, Russell J. 1998. Attentional biases in eating disorders: a visual probe detection procedure. *Int. J. Eat. Disord.* 23:199–205

Rorty M, Yager J. 1996. Histories of childhood trauma and complex posttraumatic

sequelae in women with eating disorders. *Psychiatr. Clin. N. Am.* 19:773–87

Rorty M, Yager J, Rossotto E, Buckwalter G. 2000. Parental intrusiveness in adolescence recalled by women with a history of bulimia nervosa and comparison women. *Int. J. Eat. Disord.* 28:202–8

Sanftner JL, Crowther JH. 1998. Variability in self-esteem, moods, shame and guilt in women who binge. *Int. J. Eat. Disord.* 23: 391–97

Schmidt U, Tiller J, Blanchard M, Andrews B, Treasure J. 1997. Is there a specific trauma precipitating anorexia nervosa? *Psychol. Med.* 27:523–30

Sebastian SB, Williamson DA, Blouin DC. 1996. Memory bias for fatness stimuli in the eating disorder. *Cogn. Ther. Res.* 20:275–86

Serpell L, Treasure J, Teasdale J, Sullivan V. 1999. Anorexia nervosa: friend or foe? *Int. J. Eat. Disord.* 25:177–86

Shisslak CM, Crago M, Estes LS. 1995. The spectrum of eating disturbances. *Int. J. Eat. Disord.* 18:209–19

Shisslak CM, Crago M, McKnight KM, Estes LS, Gray N, Parnaby OG. 1998. Potential risk factors associated with weight control behaviors in elementary and middle school girls. *J. Psychosom. Res.* 44:301–13

Shoebridge P, Gowers SG. 2000. Parental high concern and adolescent-onset anorexia nervosa—a case-control study to investigate direction of causality. *Br. J. Psychiatry* 176:132–37

Smolak L, Levine MP, Schermer F. 1999. Parental input and weight concerns among elementary school children. *Int. J. Eat. Disord.* 25:263–71

Spelt J, Meyer JM. 1995. Genetics and eating disorders. In *Behavior Genetic Approaches in Behavioral Medicine*, ed. JR Turner, LR Cardon, JK Hewitt, pp. 167–85. New York: Plenum

Steiger H, Leung FYK, Puentes-Neuman G, Gottheil N. 1992. Psychological profiles of adolescent girls with varying degrees of eating and mood disturbances. *Int. J. Eat. Disord.* 11:121–31

Steiger H, Stotland S, Trottier J, Ghadirian AM. 1996. Familial eating concerns and psychopathological traits: causal implications of transgenerational effects. *Int. J. Eat. Disord.* 19:147–57

Steinberg S, Tobin D, Johnson C. 1989. The role of bulimic behaviors in affect regulation: different functions for different patient subgroups? *Int. J. Eat. Disord.* 9:51–55

Steinhausen HC, Seidel R, Metzke CW. 2000. Evaluation of treatment and intermediate and long-term outcome of adolescent eating disorders. *Psychol. Med.* 30:1089–98

Stice E. 1998. Modeling of eating pathology and social reinforcement of the thin-ideal predict onset of bulimic symptoms. *Behav. Res. Ther.* 36:931–44

Stice E. 2001. A prospective test of the dual-pathway model of bulimic pathology: mediating effects of dieting and negative affect. *J. Abnorm. Psychol.* 110:1–12

Stice E, Akutagawa D, Gaggar A, Agras WS. 2000a. Negative affect moderates the relation between dieting and binge eating. *Int. J. Eat. Disord.* 27:218–29

Stice E, Hayward C, Cameron RP, Killen JD, Taylor CB. 2000b. Body-image and eating disturbances predict onset of depression among female adolescents: a longitudinal study. *J. Abnorm. Psychol.* 109:438–44

Stice E, Shaw H, Nemeroff C. 1998. Dual pathway model of bulimia nervosa: longitudinal support for dietary restraint and affect-regulation mechanisms. *J. Soc. Clin. Psychol.* 17:129–49

Stormer SM, Thompson JK. 1996. Explanations of body image disturbance: a test of maturational status, negative verbal commentary, social comparison, and sociocultural hypotheses. *Int. J. Eat. Disord.* 19:193–202

Striegel-Moore R. 1993. Etiology of binge eating: a developmental perspective. In *Binge Eating: Nature*, *Assessment and Treatment*, ed. CG Fairburn, GT Wilson, pp. 144–72. New York: Guilford

Striegel-Moore R. 1997. Risk factors for eating disorders. *Ann. NY Acad. Sci.* 817:98–109

Striegel-Moore R, Smolak L. 1996. The role of race in the development of eating disorders. In *The Developmental Psychopathology of Eating Disorders: Implications for Research, Prevention, and Treatment*, ed. MP Levine, LL Smolak, R Striegel-Moore, pp. 259–84. Hillsdale, NJ: Erlbaum

Strober M. 1991. Disorders of the self in anorexia nervosa: an organismic-developmental paradigm. In *Psychodynamic Treatment of Anorexia Nervosa and Bulimia*, ed. CL Johnson, pp. 354–73. New York: Guilford

Strong KG, Huon GF. 1998. An evaluation of a structural model for studies of the initiation of dieting among adolescent girls. *J. Psychosom. Res.* 44:315–26

Study Group Anorexia Nervosa. 1995. Anorexia nervosa: directions for future research. *Int. J. Eat. Disord.* 17:235–41

Sunday SR, Halmi KA, Einhorn A. 1995. The Yale-Brown-Cornell eating disorder scale: a new scale to assess eating disorder symptomatology. *Int. J. Eat. Disord.* 18:237–45

Thompson SH, Corwin SJ, Sargent RG. 1997. Ideal body size beliefs and weight concerns of fourth-grade children. *Int. J. Eat. Disord.* 21:279–84

Tiggemann M, Pickering AS. 1996. Role of television in adolescent women's body dissatisfaction and drive for thinness. *Int. J. Eat. Disord.* 20:199–203

Troop NA. 1998. Eating disorders as coping strategies: a critique. *Eur. Eat. Disord. Rev.* 6:229–37

Troop NA, Schmidt VA, Turnbull SJ, Treasure JL. 2000. Self-esteem and responsibility for change in recovery from bulimia nervosa. *Eur. Eat. Disord. Rev.* 8:384–93

Valdiserri S, Kihlstrom J. 1995. Abnormal eating and dissociative experiences: a further study of college women. *Int. J. Eat. Disord.* 18:145–50

VanderHam T, Meulman JJ, VanStrien DC, vanEngeland H. 1997. Empirically based subgrouping of eating disorders in adolescents: a longitudinal perspective. *Br. J. Psychiatry* 170:363–68

VanderHam T, VanStrien DC, vanEngeland H. 1998. Personality characteristics predict outcome of eating disorders in adolescents: a 4-year prospective study. *Eur. Child Adolesc. Psychiatry* 7:79–84

Vanfurth EF, Vanstrien DC, Martina LML, Vanson MJM, Hendrickx JJP, vanEngeland H. 1996. Expressed emotion and the prediction of outcome in adolescent eating disorders. *Int. J. Eat. Disord.* 20:19–31

Vitousek KB, Hollon SD. 1990. The investigation of schematic content and processing in eating disorders. *Cogn. Ther. Res.* 14:191–214

Vohs KD, Bardone AM, Joiner TE, Abramson LY, Heatherton TF. 1999. Perfectionism, perceived weight status, and self-esteem interact to predict bulimic symptoms: a model of bulimic symptom development. *J. Abnorm. Psychol.* 108:695–700

Wakeling A. 1996. Epidemiology of anorexia nervosa. *Psychiatry Res.* 62:3–9

Waller G, Hodgson S. 1996. Body image distortion in anorexia and bulimia nervosa—the role of perceived and actual control. *J. Nerv. Ment. Dis.* 184:213–19

Ward A, Ramsay R, Treasure JL. 2000a. Attachment research in eating disorders. *Br. J. Med. Psychol.* 73:35–51

Ward A, Ramsay R, Turnbull S, Benedettini M, Treasure J. 2000b. Attachment patterns in eating disorders: past in the present. *Int. J. Eat. Disord.* 28:370–76

Ward A, Tiller J, Treasure J, Russell G. 2000c. Eating disorders: psyche or soma? *Int. J. Eat. Disord.* 27:279–87

Welch SL, Doll HA, Fairburn CG. 1997. Life events and the onset of bulimia nervosa: a controlled study. *Psychol. Med.* 27:515–22

Wertheim EH, Paxton SJ, Schutz HK, Muir SL. 1997. Why do adolescent girls watch their weight? An interview study examining sociocultural pressures to be thin. *J. Psychosom. Res.* 42:345–55

Whelan E, Cooper PJ. 2000. The association

between childhood feeding problems and maternal eating disorder: a community study. *Psychol. Med.* 30:69–77

Wichstrom L. 2000. Psychological and behavioral factors unpredictive of disordered eating: a prospective study of the general adolescent population in Norway. *Int. J. Eat. Disord.* 28:33–42

Wiederman MW, Pryor T. 1996. Multi-impulsivity among women with bulimia nervosa. *Int. J. Eat. Disord.* 20:359–65

Wilson GT. 1991. The addiction model of eating disorders: a critical analysis. *Adv. Behav. Res. Ther.* 13:27–72

Woods SC, Brief DJ. 1988. Physiological factors. In *Assessment of Addictive Behaviors*, ed. DM Donovan, GA Marlatt, pp. 296–322. New York: Guilford