

Eating Disorders

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An array of cognitive, behavioral, emotional, and motivational disturbances characterizes individuals with eating disorders (EDs). The diagnostic criteria for each disorder vary with regard to the cognitive and behavioral features of the disorder, which include abnormal eating behaviors, maladaptive weight control efforts, and distorted perceptions or self-evaluations involving weight or shape. We begin by discussing the central features of the major forms of eating pathology.

DESCRIPTION OF THE DISORDER AND CLINICAL PICTURE

Anorexia Nervosa

The diagnostic criteria specified by the *DSM-IV* (American Psychiatric Association, 1994) for anorexia nervosa (AN) include (1) body weight less than 85 percent expected for height and age; (2) unreasonable fear of gaining weight or becoming overweight despite extreme thinness; (3) disturbances in perception of weight and shape, undue influence of weight and shape on self-evaluation, or denial of the gravity of severe underweight; and (4) amenorrhea in postmenarcheal females. The *DSM-IV* makes a further diagnostic distinction between restricting and binge-eating/purging subtypes of the disorder based on the absence or presence of binge-eating or purging behaviors. Some research suggests that the binge-purge type of AN, compared to the restricting type, is associated with higher levels of psychopathology, such as elevated risk of suicidality, stealing, drug abuse, emotional distress (e.g., Garner, 1993), self-injury (Favaro & Santonastaso, 1996), and family and personal histories of obesity (Garner, Garfinkel, & O'Shaughnessy, 1985). However, other findings call this subtyping scheme into question (Strober, Freeman, & Morrell, 1997). A recent prospective study with a lengthy follow-up period did not find significant differences between the current diagnostic subtypes on measures of impulsivity (kleptomania, substance abuse, suicide

attempts, or borderline personality diagnosis), course, or outcome (Eddy et al., 2002), an issue to which we return in addressing subtyping based on personality characteristics.

The clinical features of AN include a relentless pursuit of thinness and overvaluation of body shape. This typically results in a state of semistarvation via severe dietary restriction and/or high levels of physical activity, accompanied by mood disturbances, preoccupation with food, and ritualistic and stereotyped eating (Fairburn & Harrison, 2003). Physical symptoms of the disorder, aside from emaciation, may include lanugo (a light growth of hair covering the body), skin discoloration, and cardiac irregularities (Gupta, Gupta, & Haberman, 1987; Kreipe & Harris, 1992; Pomeroy, 2004; Schulze et al., 1999). Theoretical and empirical work also implicate interpersonal dysfunction as characteristic of the disorder, particularly with regard to family functioning (Leung, Schwartzman, & Steiger, 1996; Minuchin, Rosman, & Baker, 1978; Slade, 1982), a point to which we also return.

The pursuit of thinness that characterizes AN poses challenges with regard to treatment. Because patients often perceive the ED as a personal accomplishment rather than a psychiatric disorder in need of treatment, individuals with AN are typically very resistant to treatment, which invariably involves weight restoration.

Bulimia Nervosa

The diagnostic criteria specified by the *DSM-IV* (American Psychiatric Association, 1994) for bulimia nervosa (BN) include (1) recurrent episodes (at least two days per week for previous three months) of uncontrollable consumption of large amounts of food, (2) recurrent use (at least twice weekly for previous three months) of compensatory behavior to prevent consequent weight gain (e.g., vomiting, laxative abuse, diuretic abuse, fasting, or excessive exercise), and (3) undue influence of weight and shape on self-evaluation. If these symptoms occur exclusively during a period of time in which the individual satisfies diagnostic criteria for AN, this latter

diagnosis is given precedence. Individuals with BN (and binge-eating disorder) typically consume between 1,000 and 2,000 kilocalories per binge episode, usually consisting of foods with high fat and sugar content (Walsh, 1993; Yanovski et al., 1992). These individuals often experience marked feelings of guilt and shame regarding their secretive eating behaviors (Wilson, Becker, & Heffernan, 2003) and are thus typically less resistant to treatment than their anorexic counterparts (Fairburn & Harrison, 2003).

The clinical presentation of BN, as in AN, is characterized by disturbances in eating behaviors and an overvaluation of thinness. However, the commonly observed physical characteristics associated with BN differ from those associated with AN. Bulimic individuals are often of normal weight (or are overweight, rather than underweight), and a host of physiological symptoms are common but not necessary for the diagnosis of the disorder, including dorsal scarring on the hand, swelling of the parotid (salivary) glands in the face, cardiac irregularities, and varying degrees of enamel damage (Kreipe & Harris, 1992; Metzger, Levine, McArdel, Wolfe, & Jimerson, 1999; Pomeroy, 2004; Schulze et al., 1999).

Eating Disorder Not Otherwise Specified and Binge-Eating Disorder

In addition to the two widely recognized EDs noted previously, *DSM-IV* also allows for the diagnosis of EDs not otherwise specified, or EDNOS (American Psychiatric Association, 1994). Like other NOS categories in *DSM-IV*, EDNOS is an artifact of a categorical diagnostic system that needs a classification for patients with clinically meaningful or subthreshold symptomatology who fall through the cracks of established diagnoses. This is particularly problematic because the majority of patients seeking treatment for EDs receive an EDNOS diagnosis (Fairburn & Harrison, 2003; Herzog, Hopkins, & Burns, 1993), with possible implications for insurance coverage and the quality of treatment they may receive given the relatively sparse research on subthreshold or atypical presentations (Franko, Wonderlich, Little, & Herzog, 2004).

This EDNOS diagnosis is used for a variety of subthreshold conditions (Fairburn & Harrison, 2003). For example, an individual who meets all the criteria for BN except for the requisite frequency of compensatory behaviors (e.g., only once a week) would have to be diagnosed using *DSM-IV* with EDNOS rather than BN. The EDNOS category also includes partial syndrome EDs. For instance, an individual who engages in weekly compensatory behaviors in the absence of uncontrollable binge eating would probably receive a diag-

nosis of EDNOS. The EDNOS category also includes other atypical EDs, such as rumination or pica exhibited during adulthood.

Researchers are paying increasing attention to one condition currently included under EDNOS and under consideration for "elevation" to its own diagnosis in *DSM-V*, namely binge-eating disorder (BED; American Psychiatric Association, 2000). The proposed diagnostic criteria for BED include (1) repeated episodes (at least twice weekly for previous six months) of uncontrollable binge eating characterized by certain features (e.g., rapid eating, eating until uncomfortably full, eating large amounts of food when not physically hungry, eating alone because of embarrassment, and feeling guilty or depressed after overeating); (2) marked distress regarding binge eating; and (3) the absence of compensatory behaviors. If these symptoms occur exclusively during a period of time in which the individual satisfies diagnostic criteria for AN, this latter diagnosis is given precedence. If the symptoms of BED occur exclusively during a period of time in which the individual satisfies diagnostic criteria for BN, this latter diagnosis is given precedence.

BED is associated with overweight and obesity, with interview-based prevalence estimates ranging from just more than 2 percent in the general population (Stunkard et al., 1996) to nearly 19 percent (Brody, Walsh, & Devlin, 1994) in treatment-seeking obese populations. Compared to non-bingeing obese persons, those with BED evidence increased weight and shape concerns and higher levels of psychopathology, particularly affective disorders (Eldredge & Agras, 1996; Marcus et al., 1996; Mitchell & Mussell, 1995; Telch & Stice, 1998; Wilfley, Schwartz, Spurrell, & Fairburn, 2000). Although obesity is characterized by a disturbance in eating, wherein caloric intake exceeds the caloric expenditure (Rosenbaum, Leibel, & Hirsch, 1997), it is not considered a psychiatric disorder. This is in large part due to the variation in eating behaviors and psychological characteristics among the obese. The two EDs most closely associated with obesity are the aforementioned BED and night-eating syndrome, the latter having yet to appear in an edition of the *DSM* (Stunkard, 2002).

As suggested previously, numerous questions remain with respect to the classification of EDs, notably the validity of diagnostic subtypes, the problem of subthreshold diagnoses, and the utility of a widely diagnosed by heterogeneous NOS diagnosis. In addition, unlike the criteria for other disorders, the current diagnostic system does not include significant functional impairment among the requisite criteria for ED diagnoses.

PERSONALITY DEVELOPMENT AND PSYCHOPATHOLOGY

Characterizing the links between personality and disordered eating presents a number of challenges. Personality could predispose individuals to EDs, EDs could affect personality (e.g., patients with AN could become more rigid or obsessional while starving), or personality and eating pathology could mutually influence each other (see Lilienfeld, Wonderlich, Riso, Crosby, & Mitchell, 2004). Although personality and ED variables appear to influence each other, the evidence seems clear that personality variables represent diatheses for EDs (e.g., they often predate eating pathology, persist after treatment of ED symptoms, and aggregate in families of ED probands; see Gillberg, Rastam, & Gillberg, 1995; Nilsson, Gillberg, Gillberg, & Rastam, 1999; Rastam, Gillberg, & Gillberg, 1995; Sunday, Reeman, Eckert, & Halmi, 1996).

Personality researchers have approached PDs using several different models. In this section we examine four ways researchers have studied personality in EDs: (1) assessment of salient personality dimensions originally identified through clinical observation; (2) application of omnibus trait models, largely derived from personality psychology; (3) assessment of *DSM-IV* Axis II pathology (personality disorders; PDs); and (4) identification of personality subtypes in ED patients.

Clinically Observed Personality Dimensions

Three clinically observed traits have received substantial empirical attention, two originally identified in AN (perfectionism and obsessionalism) and one in BN (impulsivity). Perfectionism has been consistently identified as a salient trait in AN patients (e.g., Bastiani, Rao, Weltzin, & Kaye, 1995; Halmi et al., 2000; Strober, 1980), although it is common in BN patients as well (Vitousek & Manke, 1994). Indeed, perfectionism appears to be a significant risk factor for the development of both disorders (Fairburn, Cooper, Doll, & Welch, 1999; Fairburn, Welch, Doll, Davies, & O'Connor, 1997) and persists after weight restoration and/or recovery from EDs (Bastiani et al., 1995; Srinivasagam et al., 1995). Research has also found elevated levels of perfectionism in parents of individuals with AN, especially mothers (Woodside et al., 2002), suggesting that perfectionism is a diathesis for EDs or a phenotypic marker of a genetic vulnerability to EDs.

Closely related to the concept of perfectionism is obsessionalism. As many as 30 percent of individuals with AN have marked obsessional features upon first presentation (Thornton & Russell, 1997), and obsessive-compulsive personality traits in childhood are highly predictive of subsequent ED devel-

opment (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003). Obsessionalism also appears to persist in patients with AN after treatment, although obsessive-compulsive symptoms generally decrease as preoccupation with food and rituals surrounding food intake subside (Strober, 1980). Further, studies have consistently reported significant comorbidity between EDs and obsessive compulsive disorder (OCD; Fahy, 1991; Hsu, Kaye, & Weltzin, 1993; Rubenstein, Altemus, Pigott, Hess, & Murphy, 1995). Estimates of co-occurrence range from 25 percent to 79 percent for AN (Halmi et al., 1991, 2003; Hudson, Pope, Jonas, & Yurgelun-Todd, 1983; Rothenberg, 1986) and 25 percent to 36 percent for BN (Braun, Sunday, & Halmi, 1994; Hudson, Pope, Jonas, Yurgelun-Todd, & Frankenburg, 1987). Although the data are not yet conclusive as to the causal sequence linking obsessionalism and EDs, a number of studies strongly suggest that obsessionalism predates and is a significant risk factor for eating pathology (Anderluh et al., 2003; Smart, Beumont, & George, 1976; Thornton & Russell, 1997).

As early as 1980, researchers found impulsivity to be significantly more descriptive of bulimic anorexics than restricting anorexics (Casper, Eckert, Halmi, Goldberg, & Davis, 1980; Garfinkel, Modlofsky, & Garner, 1980). Subsequent research has consistently found heightened impulsivity in bulimic subjects compared to both restricting anorexics (e.g., Casper, Hedeker, & McClough, 1992; Vervaet, van Heeringen, & Audenaert, 2004) and normal controls (e.g., Casper et al., 1992; Diaz-Marsa, Carrasco, & Saiz, 2000). In a study of the long-term prognosis of patients with AN and BN, Sohlberg and colleagues (Sohlberg, Norring, Holmgren, & Rosmark, 1989) found impulsivity to be the strongest predictor of negative outcome. Like perfectionism, impulsivity is a multidimensional construct (Barratt, 1993). For example, one study found that lack of planning was not associated with bulimic symptomatology, but the tendency to act rashly during episodes of negative affect was (Fischer, Smith, & Anderson, 2003).

A widely studied distinction is between "multi-impulsive" versus "uni-impulsive" bulimia (Lacey & Evans, 1986). Multi-impulsive individuals with BN display several impulsive behaviors (e.g., stealing, substance abuse) in addition to binge eating, whereas uni-impulsive patients have binge eating as their only symptom or behavior that could be described as impulsive. Empirically, multi-impulsive bulimic individuals tend to have significantly more pathology than uni-impulsive patients, with greater rates of borderline PD (BPD) and mood disorders (Fichter, Quadflieg, & Rief, 1994). Data on impulsivity appear to be particularly important in light of research

linking impulsivity to early dropout rates from psychotherapy (Agras, Crow, et al., 2000).

Omnibus Personality Trait Models

Several researchers have examined the personality correlates of EDs using omnibus personality measures. A handful of studies have examined individuals with EDs using Eysenck's (1990) three-factor model of personality (neuroticism, extraversion, and psychoticism) or the Five-Factor Model (FFM; McCrae & Costa, 1999; neuroticism, extraversion, agreeableness, conscientiousness, and openness to experience). Studies using the Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975) have found neuroticism to correlate with AN (e.g., Geller, Cockell, & Goldner, 2000; Walters & Kendler, 1995). The limited number of studies applying the FFM to EDs has also consistently found increased levels of neuroticism in ED samples (Ghaderi & Scott, 2000; Heaven, Mulligan, Merrilees, Woods, & Fairouz, 2001; Podar, Hannus, & Allik, 1999; Tylka & Subich, 1999). Studies comparing subgroups of ED patients (e.g., AN with and without binge-purging) are rare but tend to find purging anorexics to be both higher in neuroticism and higher in extraversion (Ben-Tovim, Marilov, & Crisp, 1979; Gomez & Dally, 1980).

Several studies have used Cloninger's psychobiological trait model, which identifies four temperamental dimensions (novelty seeking, harm avoidance, reward dependence, and persistence) and three character dimensions (self-directedness, cooperativeness, and self-transcendence). Consistent with the clinical portrait of patients with (restricting) anorexia, AN individuals tend to be characterized by high harm avoidance and low novelty seeking (Brewerton, Hand, & Bishop, 1993; Cloninger, Przybeck, Svrakic, & Wetzel, 1994; Fassino et al., 2002; Klump et al., 2000). Researchers have obtained conflicting results on reward dependence, with most finding AN patients to be low on this dimension but some finding them to be high (e.g., Bulik, Sullivan, Weltzin, & Kaye, 1995). Other studies find AN individuals to be low on self-directedness and high on persistence (Diaz-Marsa et al., 2000; Fassino et al., 2002).

Much like their anorexic counterparts, individuals with BN tend to be high in harm avoidance (e.g., Fassino et al., 2002; Waller et al., 1993), reflecting the tendency of most ED patients to be prone to negative affect states. BN patients tend, however, to be higher on novelty seeking (Bulik, Sullivan, Joyce, & Carter, 1995; Bulik, Sullivan, Weltzin, et al., 1995; Fassino et al., 2002) and RD (Brewerton et al., 1993) and lower on self-directedness than BN patients (Diaz-Marsa et al., 2000; Klump et al., 2000; Vervaeke, Audenaert,

& van Heeringen, 2003). Fassino et al. (2002) found that patients with both anorexic and bulimic features tend to have personality profiles falling midway between those of "pure" cases.

In general, studies using omnibus trait measures have tended to produce a similar portrait to that painted by studies of clinically observed traits: AN patients tend to be high in negative affectivity or neuroticism (anxious, fearful, and harm-avoidant) and obsessional (persistent), whereas BN patients do not seem to fit any single profile. Although BN patients tend, like those with AN, to be high in negative affectivity, some studies have shown them to resemble AN patients in other respects, whereas other studies have found them to be more extraverted, impulsive, novelty-seeking, and reward dependent.

Axis II Comorbidity

Studies assessing the comorbidity between EDs and PDs have yielded highly disparate estimates, ranging from 21 percent to 97 percent depending on samples and measures (Skodol et al., 1993; Vitousek & Manke, 1994). Cluster A (odd-eccentric) diagnoses are infrequent in ED samples (Sunday et al., 2001). Cluster B (dramatic-erratic) diagnoses are the PDs most frequent observed in BN patients (e.g., Rosenvinge, Martinussen, & Ostensen, 2000), whereas Cluster C (anxious-fearful) disorders are the PDs most frequently observed in AN patients (Gartner, Marcus, Halmi, & Loranger, 1989; Herpertz-Dahlmann et al., 2001; Herzog, Keller, Sacks, Yeh, & Lavori, 1992; Nilsson et al., 1999).

Patients with AN are more likely than other ED patients to have obsessive-compulsive and avoidant features (Gartner et al., 1989; Herzog et al., 1992; Rastam, 1992; Skodol et al., 1993). Studies comparing restricting and binge-purging anorexics tend to find elevated OCPD only in patients with restricting AN, however; patients with both anorexic and bulimic symptoms tend to display more pervasive personality pathology (see Herzog et al., 1992; Wonderlich, Swift, Slotnik, & Goodman, 1990). Borderline PD (BPD) is common in both binge-purging AN and BN (Braun et al., 1994; Skodol et al., 1993; Vitousek & Manke, 1994). Little research has addressed the incidence of personality pathology in patients with ED not otherwise specified (EDNOS), which is probably the most prevalent ED diagnosis (Andersen, Bowers, & Watson, 2001; Grilo, Devlin, Cachelin, & Yanovski, 1997). However, a large longitudinal study of women with BPD found that 33 percent had a lifetime history of ED-NOS (Marino & Zinarini, 2001).

Personality Subtypes in Eating Disorders

Research on Axis II comorbidity in EDs thus tends to echo both the consistencies and inconsistencies in the literature using other personality constructs. Patients with AN, particularly restricting AN, tend to be avoidant (i.e., higher in negative affectivity and harm avoidance, and lower in extraversion) and obsessional (higher on rigidity, constraint, and compulsivity). Patients with BN, and AN patients with binge-purging symptoms, are more likely to have borderline features (including negative affectivity, impulsivity, extraversion), although in some samples they are distinguished from other ED patients by their relative freedom from personality pathology.

These findings raise two questions. First, what accounts for the inconsistency in findings for BN and for the tendency of binge-purging AN patients sometimes to resemble AN patients and sometimes to resemble BN patients? Second, given that many patients cross over from AN to BN or vice versa at some point in their lives (Eddy et al., 2002), how can patients with different EDs show such different personality profiles?

One promising explanation lies in the hypothesis that patients with similar ED diagnoses may be heterogeneous vis-à-vis personality styles but that this heterogeneity is systematic (i.e., patterned) rather than random. To put it another way, patients may be vulnerable to EDs by virtue of personality styles that only imperfectly map onto *DSM-IV* ED diagnoses.

Several research groups have in fact attempted to cluster ED patients empirically based on personality patterns, and a convergence among different methods and measures has begun to emerge (Goldner, Srikanth, Schroeder, Livesley, & Birmingham, 1999; Pryor & Wiederman, 1996; Rybicki, Lepkowsky, & Arndt, 1989; Strassberg, Ross, & Todt, 1995; Strober, 1981; Vitousek & Manke, 1994; Westen & Harnden-Fischer, 2001). For example, using a dimensional self-report personality pathology measure, Goldner et al. (1999) identified three personality subtypes within an ED sample, including a "rigid" group, a borderlinelike group, and a third group with differences from a normal comparison group.

Using a Q-sort measure of personality pathology, Westen and Harnden-Fischer (2001) similarly identified three personality subtypes that have now replicated across instruments and samples (Thompson-Brenner & Westen, 2004), which they labeled high-functioning/perfectionistic, constricted/overcontrolled, and emotionally dysregulated/undercontrolled. Constricted/overcontrolled patients were most likely to have AN, with or without bulimic symptoms, whereas emotionally dysregulated/undercontrolled patients were likely to have BN, with or without AN symptoms (classified in *DSM-*

IV as AN, binge-purging subtype). High-functioning/perfectionistic patients were not limited to any single diagnosis but were most common among BN patients without AN symptoms. Across samples, the three subtypes differ in frequency of various Axis II symptoms in ways that make sense of the consistencies and inconsistencies in the literature. Constricted patients, who are likely to have a diagnosis of restricting AN, tend to receive diagnoses of avoidant PD and OCPD. Dysregulated patients, who are likely to have either BN or binge-purging AN, are most likely to have a diagnosis of BPD. High-functioning/perfectionistic patients, who are most likely to have BN, are least likely to receive a PD diagnosis. These findings make sense of the consistent finding of constricted personality traits in restricting anorexics, borderline and impulsive traits in a subset of patients with BN and binge-purging AN, and negative affectivity without a PD diagnosis in a subset of patients with both BN and restricting AN.

EPIDEMIOLOGY

Findings from large community-recruited studies that used diagnostic interviews suggest that the lifetime prevalence of AN is between 0.5 percent and 1.0 percent for females and between 0.0 percent and 0.3 percent for males, the lifetime prevalence for BN is between 1.5 percent and 4.0 percent for females and between 0.1 percent and 0.5 percent for males, and the lifetime prevalence for BED is between 1.5 percent and 3.0 percent for females and is 0.5 percent and 2.0 percent for males (Garfinkel et al., 1995; Johnson, Cohen, Kasen, & Brook, 2002; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Newman et al., 1996). Including subthreshold cases, however, makes clear the prevalence of eating pathology in the population. Community-recruited samples indicate that for females, the rates of subthreshold or partial syndrome AN range between 1.1 percent and 3.0 percent, that the rates of subthreshold or partial syndrome BN range between 2.0 percent and 5.4 percent, and that the rate of subthreshold BED is 1.6 percent (Garfinkel et al., 1995; Lewinsohn, Striegel-Moore, & Seeley, 2000; Spitzer et al., 1992; Stice, Presnell, & Bearman, 2004). Although less attention has focused on subthreshold EDs among males, the available evidence suggests that the rates appear to be consistently lower for males than females (e.g., Garfinkel et al., 1995).

Research suggests that the incidence of new cases of AN and BN ranges between 1.3 percent and 2.8 percent during adolescence for females and that the risk for onset of these disorders peaks between 16 and 17 years of age (Lewinsohn et al., 2000; Stice, Killen, Hayward, & Taylor, 1998; Stice,

Presnell, et al., 2004). These incidence estimates suggest a relatively narrow developmental window for the emergence of the majority of cases of AN and BN, a factor that distinguishes EDs from many psychiatric disorders.

ETIOLOGY

There are numerous theories regarding the etiologic processes that promote the development of AN, implicating a wide variety of risk factors for AN, including norepinephrine abnormalities, serotonergic abnormalities, childhood sexual abuse, low self-esteem, perfectionism, need for control, disturbed family dynamics, internalization of the thin-ideal, dietary restraint, and mood disturbances (Fairburn & Harrison, 2003; Kaye, Klump, Frank, & Strober, 2000; Wilson et al., 2003). However, there have been very few prospective investigations of factors that predict subsequent onset of anorexic pathology or increases in anorexic symptoms and no prospective tests of multivariate etiologic models. The paucity of prospective studies is of concern, as these tests are essential to determining whether a putative risk factor is a precursor, concomitant, or consequence of eating pathology.

The only prospective study that tested predictors of subsequent onset of threshold or subthreshold AN found that girls with the lowest relative weight and those with very low scores on a dietary restraint scale at baseline were at increased risk for future onset of anorexic pathology over a five-year period (Stice, Presnell, et al., 2004). In contrast to hypotheses, early puberty, perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, depressive symptoms, and deficits in parental and peer support did not predict onset of anorexic pathology; however, these null findings should be interpreted with care because of the low base rate of this outcome. Unfortunately, the four additional studies we were able to locate collapsed across anorexic and bulimic pathology (e.g., McKnight Investigators, 2003; Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990; Santonastaso, Friederici, & Favaro, 1999). Thus, surprisingly little is known about the risk factors for anorexic pathology or how they work together to promote this pernicious eating disturbance.

In contrast to the dearth of prospective research in anorexic pathology, greater progress has been made regarding our understanding of the risk factors for bulimic pathology. According to the sociocultural model, an internalization of the socially sanctioned thin-ideal for females interacts with direct pressures for female thinness (e.g., weight-related teasing) to promote body dissatisfaction, which in turn is thought to increase the risk for the initiation of dieting and for negative affect and consequent bulimic pathology (Cattarin &

Thompson, 1994; Garner, Olmstead, & Polivy, 1983; Polivy & Herman, 1985; Stice et al., 1998). Body dissatisfaction may lead to dietary restraint, a behavioral manifestation of the desire to conform to the thin-ideal, which paradoxically increases the likelihood of onset of binge eating. Dieting also entails a shift from a reliance on physiological cues to cognitive control overeating behaviors, the disruption of which may lead to overeating. Body dissatisfaction is also theorized to contribute to negative affect, which increases the risk of binge eating to provide comfort and distraction from these negative emotional states.

Consistent with this general etiologic model, thin-ideal internalization, perceived pressure to be thin, body dissatisfaction, dietary restraint, and negative affect have been consistently found to increase the risk for future onset of bulimic symptoms and bulimic pathology (Field, Camargo, Taylor, Berkey, & Colditz, 1999; Killen et al., 1994, 1996; Stice et al., 1998; Stice, Presnell, et al., 2004). Support for this model derives from experimental evidence that a reduction in thin-ideal internalization, body dissatisfaction, and negative affect produce decreases in bulimic symptoms; however, these studies have failed to provide support for the role of dietary restraint as a risk factor for future onset of bulimic pathology (for a review, see Stice, 2002). Other risk factors, such as deficits in social support, substance abuse, and elevated body mass, have received limited empirical support, but these effects have not been consistently replicated (Stice, 2002). Interestingly, several hypothesized risk factors for bulimic pathology have not received support in prospective studies, including early menarche and temperamental impulsivity (Stice, 2002).

There has been comparatively little theoretical work regarding the etiologic processes that promote BED, but extant models display conceptual overlap with etiologic theories of bulimic pathology (Vogeltanz-Holm et al., 2000). Prospective studies have provided evidence that initial elevations in body mass, body dissatisfaction, dietary restraint, negative affect and emotional eating increase the risk for future onset of binge eating (Stice et al., 1998; Stice, Presnell, & Spangler, 2002; Vogeltanz-Holm et al., 2000).

Genetic factors likely contribute to the development of EDs, though the findings in this area are inconsistent. Twin studies have produced conflicting results, with heritability estimates ranging from 0.0 percent to 70 percent for AN and from 0.0 percent to 83 percent for BN (Bulik, Sullivan, & Kendler, 1998; Fairburn, Cowen, & Harrison, 1999; Kaye et al., 2000). Concordance rates are also inconsistent, as one study found that the rate for monozygotic twins was greater than for dizygotic twins (Treasure & Holland, 1989), but another observed findings in the opposite direction (Walters &

Kendler, 1995). Similarly, studies that have tried to identify specific receptor genes that are associated with EDs have produced inconsistent results that have not replicated (e.g., Hinney et al., 1998, 1999). The large range in parameter estimates suggests fundamental problems with sampling error resulting from small samples, the reliability of diagnostic procedures, or statistical models used to estimate genetic effects.

There are several important considerations regarding the interpretation of the etiologic findings. First, we were unable to locate any prospective study testing whether any biological variable, including structural or functional abnormalities in the brain or neurotransmitter abnormalities, predicted onset of any ED. This dearth of prospective research renders a determination of the causal relation between biological factors and eating pathology difficult. Second, there have been very few studies that predicted onset of anorexic pathology, bulimic pathology, or BED or compared and differentiated the risk factors for these three classes of EDs. In the absence of this type of research, it is not possible to distinguish among the etiologic processes that give rise to EDs. Third, given the focus on adolescence, very little is known about risk factors for adult onset eating pathology. Fourth, as with other disorders, research into etiology may need to focus on subcomponents or endophenotypes of the disorders (e.g., negative affect in BN) rather than on complete syndromes, given that syndromes are likely to have more complex and multifaceted etiologies than specific features or deficits. Finally, as described in the following section, another avenue for exploring etiology focuses on personality vulnerabilities or subtypes that may map only imperfectly onto particular disturbances in eating behavior.

COURSE, COMPLICATIONS, AND PROGNOSIS

Anorexia nervosa appears to have a highly variable course. Some individuals recover after a single episode, others show a chronic course marked by fluctuating patterns of weight restoration and relapse, and still others may go on to develop other EDs (Wilson et al., 2003). Severe medical complications also result from the disorder, including permanent organ damage, cerebral atrophy, and osteoporosis, which necessitates close medical monitoring during periods of low body weight. Anorexia nervosa is also associated with the highest rates of suicidal ideation and mortality of any psychiatric condition (Herzog et al., 2000; Newman et al., 1996).

Findings from community-recruited samples suggest that BN typically shows a chronic course that is characterized by periods of recovery and relapse, although subthreshold bu-

limic pathology shows less chronicity (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Stice, Burton, & Shaw, 2004). Bulimia nervosa typically results in marked subjective distress and functional impairment (Lewinsohn et al., 2000). Serious medical complications may arise in the most severe cases, some requiring hospitalization (e.g., esophageal tears) or even causing death (e.g., electrolyte imbalances that result in cardiac arrest). Community recruited samples indicate that BN is also associated with an increased risk for suicide attempt and elevated rates of comorbid affective disorders, anxiety disorders, and substance abuse (Johnson, Cohen, Kotler, Kasen, & Brook, 2002; Lewinsohn et al., 1993; Newman et al., 1996). Threshold and subthreshold BN increase the risk for future onset of depression, suicide attempts, anxiety disorders, substance abuse, obesity, and health problems (Johnson, Cohen, Kotler, et al., 2002; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Striegel-Moore, Seeley, & Lewinsohn, 2003). The treatment prognosis for BN is fair, as lasting symptom remission typically occurs for only 30 percent to 40 percent of patients who are provided with the treatment of choice (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Fairburn et al., 1995).

Community-recruited natural history studies suggest that BED shows a high autoremission rate over time, with fewer than 20 percent of cases meeting diagnostic criteria over long follow-up periods (Fairburn et al., 2000; Wilson et al., 2003), although the majority appear to meet criteria for EDNOS. Another noteworthy finding is that a large portion of individuals with binge ED show onset of obesity (Fairburn et al., 2000). Binge-eating disorder is associated with elevated major depression and slight elevations in Axis I and Axis II psychiatric disorders, as well as obesity and the consequent elevated morbidity and mortality associated with this health problem (Striegel-Moore, Wilfley, Pike, Dohm, & Fairburn, 2000; Telch & Stice, 1998). The prognosis for the treatment of BED may be hopeful, with psychotherapeutic interventions showing abstinence rates of approximately 60 percent at one-year follow-up (Wilson et al., 2003) and evidence that low-calorie behavioral weight loss interventions can be effective (Goodrick, Poston, Kimball, Reeves, & Foreyt, 1998; Reeves et al., 2001).

ASSESSMENT AND DIAGNOSIS

There are a variety of self-report questionnaires and structured diagnostic interviews for the assessment of eating pathology (Anderson & Paulosky, 2004). The following is a brief overview of the evidence for the reliability and validity of the most widely researched assessment strategies. With the

exception of the Eating Disorders Diagnostic Scale (Stice, Telch, & Rizvi, 2000), however, it is important to note that self-report eating disorder measures generally provide dimensional assessments and cannot be used to make *DSM-IV* categorical diagnoses.

The Eating Attitudes Test (EAT; Garner & Garfinkel, 1979) is a 40-item questionnaire designed to assess the symptoms of AN, but various adaptations of this scale exist, including a 26-item short form and forms for children and adolescents (Anderson & Paulosky, 2004). There is considerable evidence that the EAT possesses internal consistency, test-retest reliability, and discriminant validity with adolescents and adults.

The Eating Disorder Inventory (EDI; Garner et al., 1983) is a multi-item scale that assesses the symptoms and features of AN and BN. The current version (EDI-2; Garner, 1991) includes 11 scales, such as the bulimia, body dissatisfaction, drive for thinness, perfectionism, and impulse regulation subscales. Research suggests that the EDI possesses adequate internal consistency and test-retest reliability, and good discriminant and predictive validity with adolescents and adults.

The Bulimia Test-Revised (BULIT-R; Thelen, Farmer, Wonderlich, & Smith, 1991) and its predecessor the BULIT were designed to assess the symptoms of BN. Research has found that the BULIT-R possesses good internal consistency, test-retest reliability, discriminant validity, and predictive validity with adolescents and adults (Anderson & Paulosky, 2004; Thelen et al., 1991).

The Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 1994) is a questionnaire version of the Eating Disorder Examination interview (EDE; Fairburn & Cooper, 1993), a validated measure of eating pathology. This scale assesses the diagnostic symptoms of AN and BN, and contains subscales assessing features that are commonly associated with these EDs, such as dietary restraint and eating concern. Research has found that this scale possesses good internal consistency and test-retest reliability with adolescents and adults (Black & Wilson, 1996; Fairburn & Beglin, 1994; Luce & Crowther, 1999; Stice, 2001).

The Questionnaire for Eating Disorder Diagnoses (QEDD; Mintz, O'Halloran, Mulholland, & Schneider, 1997) is a questionnaire assessing the diagnostic criteria for AN, BN, and BED. Research has found that this scale showed good test-retest reliability and criterion validity (Mintz et al., 1997).

The Eating Disorder Diagnostic Scale (Stice et al., 2000) is a brief self-report scale assessing the *DSM-IV* diagnostic criteria for AN, BN, and BED. Research has found that the continuous symptom composite shows good internal consistency, test-retest reliability, and predictive validity, and that the ED diagnoses made with this scale also possess good test-retest

reliability and show good criterion validity with interview-based diagnoses (mean $k = .81$; Stice, Fisher, & Martinez, 2004; Stice et al., 2000).

There are structured psychiatric interviews for arriving at *DSM-IV* (American Psychiatric Association, 1994) diagnoses of AN, BN, and BED (e.g., the Eating Disorder Examination [EDE]; Fairburn & Cooper, 1993; Spitzer, Williams, Gibbon, & First, 1990; Structured Clinical Interview for *DSM* [SCID]).

The Eating Disorder Examination (EDE; Fairburn & Cooper, 1993) is a semistructured psychiatric interview assessing the diagnostic criteria for AN and BN. This interview also contains subscales assessing features that are commonly associated with these EDs, such as dietary restraint and eating concern. Research has found that the continuous scales from this interview possess good internal consistency, test-retest reliability, and discriminant validity, and that the diagnoses show good interrater reliability and test-retest reliability (Fairburn & Cooper, 1993; Rizvi, Peterson, Crow, & Agras, 2000; Stice, Burton, et al., 2004; Williamson, Anderson, Jackman, & Jackson, 1995).

The SCID (Spitzer et al., 1990) is a standardized interview that assesses current and lifetime psychiatric status for major Axis I psychiatric disorders using criteria in accordance with the *DSM*. The reliability and validity of the SCID I have been well documented, with interrater reliability agreement (k) ranging from .70 to 1.00 (Segal, Hersen, & Van Hasselt, 1994; Williams et al., 1992). However, we were unable to locate information concerning the test-retest reliability for specific ED diagnosis with the SCID.

IMPACT ON ENVIRONMENT

Research on PDs in general would suggest that individuals with the kinds of pathology to which ED patients are vulnerable would likely suffer substantial social and occupational problems. A large body of research suggests that social functioning is worse across all categories of PD than in non-PD comparison groups (Casey & Tyrer, 1986), and that PD traits predict poor social functioning above and beyond variance accounted for by Axis I symptoms such as depression and anxiety (Oltmanns, Melley, & Turkheimer, 2002; Seivewright, Tyrer, & Johnson, 2004). Indeed, interpersonal dysfunction, or personality traits or dynamics that render individuals vulnerable to such dysfunction, comprises many PD criteria in *DSM-IV*. For example, the *DSM-IV* criteria for BPD characterize these patients' interpersonal functioning as unstable and intense, with alternating patterns of idealization and devaluation, inappropriate rage, and desperate fears of rejection

and fears of aloneness. Work histories of patients with BPD are also frequently impaired, with many losing jobs for interpersonal reasons (Zittel & Westen, in press).

Interpersonal pathology also characterizes the other most frequent PDs seen in ED patients, avoidant PD and OCPD. Avoidant PD, which by definition disrupts social functioning, involves a pervasive pattern of social inhibition and fears of becoming close to others. The work and interpersonal functioning of patients with OCPD is characterized by the sacrifice of friendships and leisure activity to excessive devotion to productivity and difficulty delegating tasks to others unless they submit to the patient's often critical standards. The *ICD-10* adds to this list excessive pedantry and adherence to social conventions (World Health Organization, 1993). Recent research finds, in fact, that OCPD patients can be socially aversive because of their self-righteousness, often couched in moral terms (Shedler & Westen, 2004).

The available research on comorbid personality pathology in patients with EDs suggests that personality pathology has a strong impact on the patient's environment—particularly through social and work functioning—above and beyond the considerable problems that EDs alone can cause. Cluster B personality problems in ED patients are associated with suicide attempts, self-destructiveness, and substance abuse, as in other patients with Cluster B (particularly borderline) pathology (Johnson, Tobin, & Enright, 1989; Milos, Spindler, Hepp, & Schnyder, 2004). These problems in turn are associated with significant disruptions in social and occupational functioning. For example, studies comparing patients with BN and BPD to those with BN without BPD have found that the borderline patients showed significantly more disturbed psychosocial adaptation and family environment (Johnson et al., 1989). As a result, these patients may appear more often for treatment than ED patients without significant personality pathology.

Similarly, several studies have found that the subtypes of patients with both dysregulated/undercontrolled and rigid/overcontrolled personality disturbances show substantially lower global functioning scores than patients without these profiles (Thompson-Brenner & Westen, 2004; Westen & Harnden-Fischer, 2001). ED patients matching these personality-disturbed profiles also show higher rates of psychiatric hospitalization (Thompson-Brenner & Westen, 2004; Westen & Harnden-Fischer, 2001). In another study, individuals characterized by a dietary-depressive subtype identified in other research and associated with considerable personality pathology showed more symptom persistence and social impairment over a five-year follow-up period (Stice & Fairburn, 2003).

TREATMENT IMPLICATIONS

Clinicians have treated EDs in the community for more than three decades (see Bruch, 1973; Fairburn, 1997; Minuchin et al., 1978), employing a range of therapies, mostly cognitive behavioral (CBT), psychodynamic, family systems (particularly for adolescents with the disorder), biological, and eclectic or integrative. With the exception of some promising early studies using family therapy to treat adolescents with AN (Dare, Eisler, Russell, & Szmukler, 1990; Eisler et al., 1997; Le Grange, Eisler, Dare, & Russell, 1992), a recent study employing CBT after hospitalization (Pike, Walsh, Vitousek, Wilson, & Bauer, 2003), and some limited data on medication response (Kaye et al., 2001), data from controlled trials are limited for AN. In part this reflects the refractoriness of AN to randomized controlled trials (RCTs) methods, given that many patients require hospitalization, even in the midst of outpatient treatments that are ultimately successful. Even less data are available regarding treatment of the substantial numbers of patients in clinical practice who receive a diagnosis of EDNOS, and research on the treatment of BED is just beginning, although promising (see National Institute for Clinical Excellence guidelines, 2004).

In contrast, a substantial empirical literature exists using RCTs to assess treatments for BN, particularly CBT, with some studies also testing alternative behavioral treatments and interpersonal psychotherapy (IPT). A recent meta-analysis of these trials, analyzing not only effect size but additional variables bearing on outcome and generalizability, indicates that CBT (18 to 19 sessions on average), IPT, and various behavior therapies produce substantial reductions in BN symptomatology (Thompson-Brenner, Glass, & Westen, 2003). Additional treatments with promise for treating EDs include guided self-help (Bailer et al., 2004) and dialectical behavior therapy (Safer, Telch, & Agras, 2001). Meta-analytic data suggest that individual CBT produces substantially better outcomes than group CBT and slightly better outcomes than other therapies tested on some indexes. Even the most successful treatments, however, produce only a 50 percent recovery rate among treatment completers, and approximately 40 percent among those who begin treatment, including those who do not complete it (intent to treat analyses). Furthermore, approximately 40 percent of patients seeking inclusion in RCTs are ruled out of the average study for meeting one or more of a long list of exclusion criteria (Thompson-Brenner et al., 2003).

Before examining data on the relation between personality and treatment, we briefly describe three treatments for BN, which have implications for the treatment of all EDs given their theoretical postulates and intervention strategies that are

readily adapted to other EDs: CBT, IPT, and psychodynamic psychotherapy. We focus on CBT and IPT because they have been most often tested in clinical trials. We include psychodynamic therapy because it is frequently practiced or integrated with CBT techniques in the community (Arnow, 1999; Thompson-Brenner & Westen, in press b).

Cognitive Behavioral Therapy

CBT for BN was first developed and tested by Christopher Fairburn and colleagues in the 1980s, and the 19-session manual produced for a treatment-comparison study in 1993 remains the standard for CBT in the field (Fairburn, Marcus, & Wilson, 1993). CBT has recently been compared with nutrition management in AN patients following weight restoration (Pike et al., 2003), and Fairburn's latest CBT manual is designed to treat patients with any ED diagnosis (Fairburn, Cooper, & Shafran, 2003). CBT is based on the premise that patients with EDs are overly concerned with their shape and weight that they too closely link their self-esteem to shape and weight concerns. The CBT model proposes that overconcern with shape and weight leads to particular behaviors (such as rigid dietary rules, overall calorie restriction, and driven exercise) that leave patients vulnerable to subjective overeating and objective binge eating. Overeating and binge eating, in the context of shape and weight concerns, in turn lead to purging behavior, increased restriction, and other forms of calorie and weight elimination. In addition, the low self-esteem produced by overeating, binge eating, and purging also may produce additional motivation to raise self-esteem through weight loss.

This vicious cycle is hypothesized to lie at the core of ED behavior, although the CBT model has recently begun to expand to emphasize other behaviors that contribute to the maintenance of the disorder, such as avoidance of looking at one's appearance and weight (Shafran, Fairburn, Robinson, & Lask, 2004). This avoidance in turn contributes to the maintenance of the association between shape and weight and anxiety and prevents more accurate appraisal of appearance and weight. Similarly, patients may engage in body checking, producing selective information that reinforces the belief that their body or a body part is too large. CBT researchers view these core cognitive and behavioral processes as central to the development and maintenance of pathological eating behaviors in patients with and without personality pathology (Wilson & Fairburn, 1998).

Cognitive behavioral treatment for EDs (Fairburn, Marcus, et al., 1993; Pike et al., 2003) consists of multiple interventions arranged in a phased sequence. The interventions include psychoeducation regarding the model and the symptoms; self-

monitoring of eating and symptom behavior; prescription of regular eating, including three meals and two snacks at regular intervals; reintroduction to feared foods and loosening of dietary rules; and cognitive and behavioral interventions for shape and weight concerns (as well as mood and interpersonal problems and other issues that require problem solving). The early phase of treatment is highly focused on behavior change and self-monitoring; later phases of treatment focus on addressing obstacles to behavior change and vulnerabilities to relapse. Fairburn and colleagues have recently proposed a flexible transdiagnostic version of CBT that may include modules even more directly targeting interpersonal and mood regulation issues as well as certain aspects of personality (notably perfectionism and self-esteem) using CBT interventions such as cognitive restructuring (Shafran et al., 2004).

Interpersonal Psychotherapy

Interpersonal psychotherapy was originally developed for use with major depressive disorder and was chosen as a short-term treatment comparison for later trials of CBT (Fairburn, Jones, Peveler, Hope, & O'Connor, 1993). Since then, however, it has demonstrated efficacy for reducing the symptoms of BN in two trials (Agras, Walsh, et al., 2000; Fairburn et al., 1991; Fairburn, Jones, et al., 1993). IPT focuses on four general domains of interpersonal functioning believed to contribute to the development or maintenance of depression, BN, and other psychiatric disorders, called interpersonal deficits, grief, conflict, and role transitions (Fairburn, 1997; Wilfley, Douchis, & Welch, 2000).

IPT includes an initial extended assessment period, which links the onset and development of ED symptoms to important interpersonal events. Following the assessment, the interpersonal problem is formulated, and strategies to solve it are proposed, with the goal of resolving the problem or problems in roughly 20 sessions. Reflecting its origins as a credible control condition for CBT that does not overlap substantially with CBT in intervention strategies (Westen, Novotny, & Thompson-Brenner, 2004), IPT therapists do not focus on eating symptoms themselves, focusing instead on current interpersonal problems in the patient's life.

Psychodynamic Psychotherapy

Psychodynamic treatments for EDs assume that eating symptoms typically arise in the broader context of personality patterns, such as difficulty identifying and regulating emotions; conflicts about, or difficulties in, the self-regulation of impulses (either undercontrol or overcontrol); problematic ways

of viewing the self and others and interacting with significant others, particularly in meaningful relationships such as attachment relationships ("object relations"; Westen, 1991); and deficits in the consolidation of a sense of self or identity (Waller, Dickson, & Ohanian, 2002). Psychodynamic approaches do not assume that all patients with the same ED diagnosis have the same underlying problems, and although contemporary dynamic theorists acknowledge the substantial roles of culture and biology in symptom generation and maintenance, they tend to view eating symptoms and body image distortions as symptomatic expressions of broader personality patterns that may manifest in other realms of the patient's life as well, such as impulse and emotion regulation in situations not involving food (e.g., the literature on multi-impulsive bulimics; Waller et al., 2002; Waller, Ohanian, Meyer, & Osman, 2000), relationships, or sexuality (e.g., Eddy, Novotny, & Westen, in press).

Psychodynamic practice with ED patients is nowhere presented in a manual, and probably its most definitive statement remains Bruch's (1973, 1978) conceptualization of the nature and treatment of ED pathology, although several others have contributed as well (e.g., Tobin & Johnson, 1991). In a recent naturalistic study of treatments for BN in the community (Thompson-Brenner & Westen, in press a), although most clinicians self-reporting a primary psychodynamic orientation reported using a range of strategies, including relatively directive strategies targeting specific symptoms typically associated with CBT, the following interventions were among those with high loadings on an identifiable psychodynamic factor: encouraging exploration of feelings the patient found uncomfortable or unacceptable; focusing on the similarities between the patient's relationships and perceptions of relationships repeated over time, settings, or people; focusing on the patient's conflicting feelings or desires; helping the patient come to terms with her relationships with and feelings about significant others from the past; addressing the patient's avoidance of important subjects and shifts in mood; identifying maladaptive interpersonal patterns and the thoughts, feelings, and motives underlying them; focusing on ways the patient deals with anger or aggression; encouraging the patient to experience and express feelings in the session; using the therapeutic relationship to offer the patient a different model for relationships than she had previously experienced; linking the patient's current feelings or perceptions to experiences from the past; encouraging the discussion of the patient's wishes, fantasies, dreams, and so forth; exploring issues of sexuality; focusing on the relationship between the therapist and patient; encouraging the patient to assert herself or get her needs met in relationships; and helping the patient regulate intense emotions. Many of these interven-

tions were among those identified by Hilsenroth and colleagues (Hilsenroth, Ackerman, Blagys, Baity, & Mooney, 2003) as distinguishing psychodynamic from CBT treatments for other disorders, notably depression.

Personality, Treatment Interventions, and Outcome

Data from a number of studies indicate that personality pathology has a negative influence on ED treatment outcome. Debate exists over inconsistencies in these findings, however, such as differences in the personality variables associated with negative outcome and failures to replicate. Some of these inconsistencies likely reflect limitations of sample size for detecting moderators in most outcome studies. A number of common exclusion criteria—such as suicidality, upper weight limits, and other comorbidity—may also inadvertently rule out patients with the most personality disturbance from inclusion in major trials and hence prevent definitive conclusions on personality pathology as a moderator of outcome (Thompson-Brenner & Westen, in press a). Application of four common RCT exclusion criteria to a naturalistic BN sample suggested that 40 percent of all patients—including 66 percent of patients with BPD—would likely have been excluded, and as reported in the following paragraph, these patients in fact fared considerably worse and required different intervention strategies (Thompson-Brenner & Westen, in press a). Given that the average treatment group in an RCT has 30 patients (Thompson-Brenner & Westen, 2003), a third or less of the sample is likely to show the same form of significant personality pathology, and as much as 60 percent may be ruled out by other exclusion criteria, at present little can be said with certainty about the relation between personality pathology and outcome.

The majority of studies investigating associations between assorted personality variables (e.g., BPD, Cluster B PDs, any PD) and assorted outcome variables have, in fact, found significant negative association (or strong trend) in RCTs for BN (e.g., Davis, Olmsted, & Rockert, 1992; Fahy & Russell, 1993; Fairburn, Jones, et al., 1993; Garner et al., 1990; Johnson, Tobin, & Dennis, 1990; Rossiter, Agras, Telch, & Schneider, 1993; Steiger & Stotland, 1996; Wonderlich, Fullerton, Swift, & Klein, 1994), although such findings are not universal (e.g., Bossert, Schmolz, Wiegand, Junker, & Krieg, 1992; Bulik, Sullivan, Joyce, Carter, & McIntosh, 1998; Grilo et al., 2003). Multiple studies suggest that personality variables such as perfectionism, obsessive compulsive personality disorder, and asceticism predict poor outcome in AN (e.g., Bizeul, Sadowsky, & Rigaud, 2001; Fassino et al., 2001; Rastam, Gillberg, & Wentz, 2003; Sutandar-Pinnock,

Blake Woodside, Carter, Olmsted, & Kaplan, 2003; for a review, see Steinhausen, 2002).

To what extent each of the treatments described previously is likely to address personality or to show moderation of outcome by personality variables is speculative. The effort to broaden CBT for BN to include interventions targeted at particular aspects of personality (i.e., perfectionism and self-esteem) and problems often associated with personality disorders (i.e., interpersonal problems and mood dysregulation) may increase its applicability to the range of problems typically presented by patients with personality disturbance, although this is not likely to be accomplished in the brief manualized version of the treatment. Indeed, in a recent naturalistic study of treatments for BN in the community (Thompson-Brenner & Westen, in press a, in press b), the average length of treatment for CBT was 69 sessions, and treatment length was associated with degree of personality pathology. Because of the flexible nature of IPT treatment planning, it could conceivably be adapted to the needs of patients with personality pathology, particularly those with dysregulated pathology, which has substantial interpersonal components. To what extent it can address personality problems such as obsessiveness, perfectionism, or rigidity, which are more internalizing, or the noninterpersonal problems of dysregulated patients (such as deficits in emotion regulation), is unclear. In either case, there is no evidence suggesting that enduring personality pathology is likely to change in 20 weeks (see Westen et al., 2004), suggesting that adaptations of IPT to patients with substantial personality pathology would likely need to be significantly extended. Psychodynamic therapy is unique in its focus on personality as a diathesis for EDs; unfortunately, however, it has not been subjected to empirical test (except as an "intent to fail" condition in one study, in which therapists were forbidden to talk with the patient about eating or weight concerns; see Westen et al., 2004).

A naturalistic study of treatment in the community may shed some preliminary light on personality as a moderator of outcome and of the kinds of interventions that may prove useful to test in future studies of treatment of ED patients with significant personality pathology (Thompson-Brenner & Westen, in press a, in press b). A national sample of 145 experienced MD and PhD clinicians provided data concerning their most recently terminated (successful or unsuccessful) treatment of a patient with bulimic symptomatology (including mixed ED diagnoses). The sample was roughly evenly split by theoretical orientation, with most clinicians reporting a primary psychodynamic or CBT orientation (about 40 percent each), although the majority described themselves as drawing heavily from other approaches (which was ap-

parent in the interventions they reported using). Personality pathology was the norm rather than the exception and did not vary by clinicians' theoretical orientation: Clinicians diagnosed almost 60 percent of patients with a personality disorder, with approximately one in four meeting criteria for BPD and one in four for dependent PD. Clinicians classified 41.7 percent as high-functioning/perfectionistic, 30.9 percent as constricted, and 27.3 percent as dysregulated. Pretreatment global functioning and posttreatment outcome were both significantly related to personality style, with dysregulated patients faring most poorly, followed by constricted patients, followed by high-functioning patients. In multiple regression analyses, both dysregulation and constriction significantly predicted poor outcome, above and beyond variance accounted for by severity of eating symptoms and comorbid Axis I disorders.

CONCLUSIONS

Researchers have made substantial inroads into the understanding of EDs over the last 25 years, providing increasing knowledge about their nature, prevalence, course, and treatment. Controversies remain, however, about the classification and etiology of EDs and about the complex relations between symptoms of disordered eating and personality.

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