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Eating disorders and personality: A methodological and empirical review

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Abstract

Methodological approaches utilized to evaluate models of the relationship between personality and eating disorders, as well as empirical support for each model, are reviewed. Limited prospective research suggests that negative emotionality, perfectionism, drive for thinness, poor interoceptive awareness, ineffectiveness, and obsessive-compulsive personality traits are likely predisposing factors. Limited family study research suggests that obsessive-compulsive personality disorder (OCPD) and anorexia nervosa share a common familial liability. Potential pathoplastic personality factors include Cluster B personality disorders and OCPD, which predict a poorer course and/or outcome, and histrionic personality traits and self-directedness, which predict a more favorable course and/or outcome. Future research should focus upon sophisticated prospective and family study research in order to best evaluate competing models of the eating disorder–personality relationship.
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Behavioral science is best advanced when empirical data analysis is preceded and guided by the careful development of explanatory psychological construct systems (Haynes, 1992; Junker & Pilkonis, 1993). In psychopathology research, well explicated causal models may attempt to explain a number of parameters of the behavior in question, including occurrence, duration, magnitude, latency, or rate of recovery (Haynes, 1992). There are debates about the most productive way to construct such models. While some individuals believe that causal models should be limited in scope, with only a few broad-based and well measured constructs (e.g., Junker & Pilkonis, 1993), others emphasize the need for models to contain a larger number of causal variables which are comprehensive, synthetic, and dynamic (Barnett & Gotlib, 1988; Everly, 1986; Haynes, 1992). There appears to be little debate, however, that scientific thinking must accompany sophisticated quantitative methodologies if psychological science is to produce findings which may be replicated over time.

The relationship between personality traits (and personality disorders) and eating disorders has received considerable empirical testing in spite of the fact that conceptual models of the nature of this relationship have been limited

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and poorly defined. Empirical studies suggest that certain personality traits or disorders are fairly common in eating disordered individuals (see reviews by Cassin & von Ranson, 2005; Vitousek & Manke, 1994), predict the course of the eating disorder (e.g., Dancyger, Sunday, Eckert, & Halmi, 1997), are associated with treatment outcome (e.g., Bulik, Sullivan, Carter, McIntosh, & Joyce, 1999), and may run in families of eating disordered individuals (e.g., Lilenfeld et al., 1998). Only rarely have such findings been explicitly presented as a test of a particular conceptual model of the relationship between eating disorders and personality variables (e.g., Lilenfeld et al., 2000). Furthermore, although there have been reviews of both the conceptual (e.g., Sohlberg & Strober, 1994; Wonderlich & Mitchell, 1997) and the empirical literature (e.g., Cassin & von Ranson, 2005; Vitousek & Manke, 1994), there has yet to be a systematic review which both specifies theoretical models of the relationship between personality and eating variables and critically evaluates the level of empirical support for such models.

In the present review, we briefly describe each of the conceptual models thought to be most relevant to the relationship between personality and eating disorders. This is not intended to be an exhaustive review of all possible explanatory models of comorbidity (see Klein & Riso, 1993), but rather a detailed focus on those models that have been most studied in the field. The present review builds upon two recent reviews in the eating disorders field (Jacobi, Hayward, deZwaan, Kraemer, & Agras, 2004; Stice, 2002), both of which focused upon risk factors. The current review focuses specifically upon personality risk factors, as well as the numerous other ways in which personality may be conceptually related to eating disorders. In addition, we examine the methodological approaches that may be utilized to evaluate these models. Next, we review empirical studies which have tested these models, whether explicitly or implicitly, and examine the relative empirical support among the various models. Finally, we offer methodological and conceptual suggestions for future research in this area.

1. Models of the personality/eating disorder relationship

The study of personality dimensions or disorders, and their relationship to other psychiatric or psychological disorders, is necessarily complicated by numerous measurement and methodological issues associated with personality variables. These include issues such as stability of personality traits (Conley, 1985; Pedersen & Reynolds, 1998), state effects on personality trait measurement (Hirschfeld & Klerman, 1979; Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950; Vitousek & Manke, 1994), high levels of personality disorder comorbidity and significant heterogeneity within each personality disorder diagnosis (Livesley, 2001), questionable validity of personality disorder assessment in adolescents (Shiner, 1998), and the debate over whether personality disorders are best conceptualized categorically or dimensionally (Clark, Livesley, & Morey, 1997; Ekselius, Lindstroem, von Knorring, & Bodlund, 1994; Widiger, 1993).

In spite of these challenges, it is important to consider how personality might relate to specific psychopathologies. Below, we review a number of such conceptual models (depicted in Fig. 1) thought to be most relevant to the relationship between eating disorders and personality disorders, many of which have been discussed previously in greater detail (e.g., Clark, Watson, & Mineka, 1994; Klein & Riso, 1993; Lyons, Tyrer, Gunderson, & Tohen, 1997; Wonderlich & Mitchell, 1997). These models have been applied to numerous forms of comorbid psychopathology, yet have never been systematically evaluated in an effort to understand the relationship between eating disorders and personality. In our review of the empirical literature testing these models, we included both studies of personality (generally assumed to lie on a continuum) and personality disorders (generally assumed to be categorical). The continued debate about the most appropriate way to measure personality variables (Livesley & Jang, 2000; Westen & Shedler, 2000) requires that we be inclusive of both approaches in this review.

1.1. Predispositional model

This model depicts a relationship in which a personality construct precedes and increases the risk of developing an eating disorder. Furthermore, this model assumes that the personality disturbance and eating disorder are independent conditions in which the etiology and pathophysiology of the two disorders are distinct (Lyons et al., 1997). The assumption of independence helps to separate the predispositional model from the spectrum model, which will be described shortly. The predispositional model was implied in Bruch's (1973) early writings regarding the premorbid characteristics of the anorexic child as "compliant, perfectionistic, and dependent." More recently, this model was utilized by Strober (1991) to account for the etiological relevance of temperament factors in anorexia nervosa. He

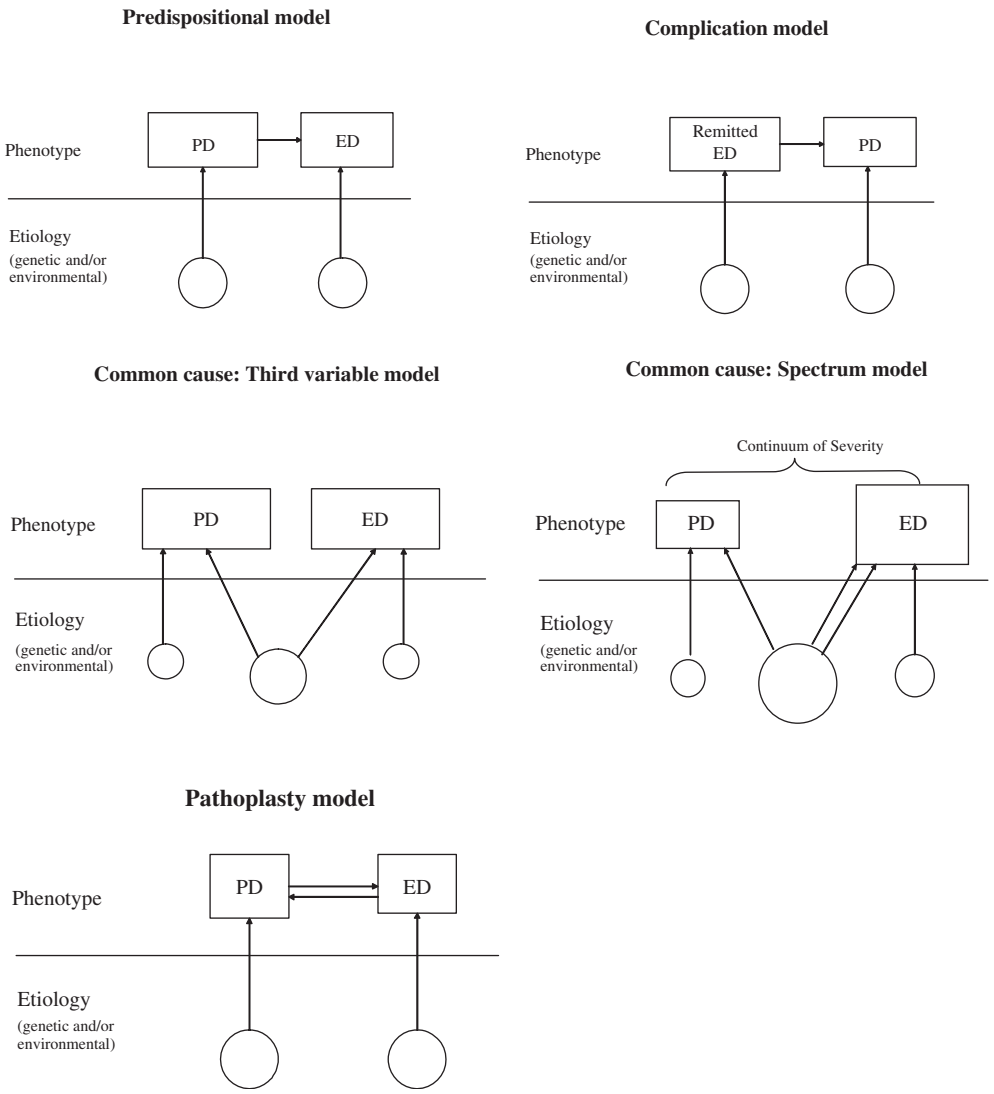


Fig. 1. Five theoretical models of the eating disorder–personality (disorder) relationship.

posited that personality traits such as low novelty seeking, high harm avoidance, and high reward dependence (Cloninger, Svrakic, & Przybeck, 1993) may mediate environmental events associated with adolescence in a manner which increases the risk of developing anorexia nervosa. However, it is unclear if Strober's theorizing reflects a true predispositional model or, alternatively, a spectrum model. If his model implies that temperament is an independent entity which simply increases the risk for eating disorders, then it is indeed a predispositional model. On the other hand, if he is suggesting that a harm avoidant temperament and anorexia nervosa represent variants of the same underlying psychopathology, then this would be a spectrum model. This highlights the need to determine whether personality traits and eating disorders are independent entities, which is a prerequisite for the predispositional model.

1.2. Complication model

This model does not assume that personality variables precede or increase the risk of developing eating disorders, as in the predispositional model. Rather, variation in personality variables is thought to be the product or *complication* of the eating disorder itself. One specific example within this general class of models is the *state-*

effect model, which refers to eating disorder symptomatology causing short-term personality changes as a result of the current eating disorder. A well-known example which supports this model is the landmark study by Keys et al. (1950), in which semi-starved subjects were found to exhibit many symptoms associated with eating disorders, which included personality constructs such as obsessionality. These "traits" emerged in response to the semi-starved state.

By contrast, another variant within this general class of complication models is the *scar-effect model*, which refers to eating disorder symptomatology resulting in long-term personality changes, due to an individual having previously experienced the disorder. Thus, while the state-effect model refers to the impact of the eating disorder on personality during the eating disorder episode, the scar-effect model refers to the enduring effect on personality even after resolution of the eating disorder.

1.3. Common cause model

This model implies that personality traits/disorders and eating disorders are different conditions which are both caused by the same underlying factor or factors. A genetic analogy is the concept of "pleiotropy," in which a particular genotype gives rise to two or more different phenotypic expressions (Plomin, DeFries, & McLearn, 1990). For example, PKU is attributed to a single gene most noted for causing mental retardation. However, individuals with PKU also tend to have lighter hair and skin color. This is likely due to the pleiotropic (multiple) effects of the gene (Plomin et al., 1990).

The first type of common-cause model may be referred to as the *third-variable model*. In this model, the two disorders are considered independent and arising primarily from the same underlying cause. For example, it may be posited that some underlying genetic factor increases the risk of both anorexia nervosa and obsessive compulsive personality disorder. Alternatively, it could be posited that a psychosocial risk factor (e.g., early trauma) increases the risk of both a personality disturbance (e.g., borderline personality disorder) and an eating disorder (e.g., bulimia nervosa) (Lyons et al., 1997).

The second type of common-cause model is referred to as the *spectrum model*. While this model is not consistently defined in the literature, a typical assumption is that the personality trait and eating disorder are considered to be quantitative, rather than qualitative, variations of the same underlying etiology and pathophysiology, and consequently are not independent disorders. Spectrum models posit that particular personality variables and eating disorders frequently coexist because they represent quantitatively different variations of the same fundamental disorder, both in terms of etiology and pathophysiology. While this model is commonly employed to account for the relationship between schizotypal personality disorder and schizophrenia (Lenzenweger, 1998), the same logic may be applied to eating disorders. For example, it is possible to think of a particular personality trait (e.g., perfectionism) or disorder (e.g., obsessive compulsive personality disorder) as a variation of the same psychopathology associated with certain eating disorders. Recent speculation that anorexia nervosa emerges from a temperamental substrait (Lilenfeld & Kaye, 1998; Strober, 1995) may be consistent with such a spectrum perspective. The assumption, in this case, would be that anorexia nervosa and the temperamental disturbance are variations on the same psychopathological theme and do not represent independent entities. Such subtleties regarding independence of the disorders are at the heart of the distinction between third-variable and spectrum models.

1.4. Pathoplasty model

The pathoplasty model is not a causal model. Instead, the model implies that once personality traits and eating disorders are established, they may interact in a way that modifies the presentation and course of each condition. For example, obsessive-compulsive personality traits may have a significantly different impact on the clinical features and course of bulimia nervosa than would impulsive personality traits. Some (e.g., Klein & Riso, 1993) have noted that differentiating the pathoplasty model from the predispositional model is difficult. For example, if a particular personality trait served as a predispositional risk factor for eating disorders, it is likely to continue to operate as a pathoplastic factor over time, although it may exert different effects as time passes. It will be important for theoreticians to specify whether or not they consider a particular personality factor to have true causal significance versus pathoplastic significance in a given model.

2. Methodological approaches used for model testing

2.1. Prospective design

The ideal test of the *predispositional model* is with a prospective design. The necessary features for such a study include assessment at least at two different points in time in which the subjects change status on the outcome of interest (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997). A prospective design overcomes some of the inherent limitations of cross-sectional and/or correlational designs. A true "risk factor" (e.g., personality variable) must precede the outcome variable of interest (e.g., eating disorder); such temporal ordering is only possible to study with a prospective design (Jacobi et al., 2004; Kazdin et al., 1997; Kraemer et al., 1997). However, due to the relatively low base rates of eating disorders, prospective studies are extremely difficult to execute with this population.

Longitudinal designs with subjects who may already display the outcome variable, and thus are not true prospective longitudinal designs, may provide a test of the *pathoplasty model*, particularly in terms of the pathoplastic influence of personality on eating disorder course and outcome. For instance, if one were to find that those subjects with higher premorbid positive affect scores were more likely than subjects with low positive affect to recover from their eating disorder, this would imply that this personality trait affects the course (the outcome in this case) of the eating disorder. Similarly, controlled treatment studies that examine the predictive significance of personality on treatment outcome provide a longitudinal test of the pathoplasty model.

A true prospective design is also the ideal test of the *complication model*. A personality trait that was not present premorbidly, but emerges after the development of the eating disorder, would demonstrate support for this model. However, in order to distinguish state-effect from scar-effect models, one would need to follow subjects to resolution of the eating disorder. Those personality traits which disappear when the eating disorder resolves would support the state-effect model. Those which persist would support the scar-effect model.

2.2. Recovered design

Perhaps because of the costs associated with prospective designs, as well as the low base rates of eating disorders, recovered designs are often used to test the *predispositional model*. A recovered design entails identifying individuals who were previously ill with the condition of interest, but now no longer meet criteria to be considered currently afflicted.

The fundamental idea with such a design is that the personality characteristics of the recovered population may reflect a relatively enduring trait which was present premorbidly and served to increase risk for the disorder. Importantly, the recovered design removes the potentially powerful effects of the eating disorder symptoms themselves on personality, which are emphasized in the complication model, so that enduring personality traits are not simple epiphenomena associated with eating disorder status. However, recovered designs are limited because of the inability to distinguish "scar effects" on personality, due to the prior eating disorder, from true premorbid personality traits. That is, personality traits in a recovered individual may represent a complication of having had the previous illness (i.e., a "scar" of the illness) or, alternatively, they may be interpreted as reflecting premorbid personality status. Another problem with this design is the potential for residual eating disorder symptomatology to continue to affect personality traits. Different studies have used varying definitions of recovery, thus affecting the ability to make conclusions about "scars" from the illness. It is well known that a starvation-state produces changes in one's personality (Keys et al., 1950; Vitousek & Manke, 1994). The extent to which these subtle changes may persist after resolution of acute eating disorder symptomatology has yet to be determined. Thus, a recovered design provides an alternative, yet less rigorous test of the predispositional model, than a prospective design.

Recovered designs, while not quite as good as prospective designs, do provide a reasonably robust test of *complication models*. Specifically, personality elevations in currently ill subjects compared to recovered subjects, would provide support for the *state-effect model* (e.g., Ames-Frankel et al., 1992), while personality elevations in recovered subjects compared to never ill subjects may support the *scar-effect* model. However, this pattern of results could also be explained by the predispositional model. As previously discussed, it is impossible to tease apart scar effects from premorbid traits using the recovered study design alone. Therefore, prospective, longitudinal designs are ultimately needed to distinguish between these two potential interpretations of recovery study data.

2.3. Retrospective design

Retrospective reports by subjects and their parents (or other informants) are a third, less robust way to test the *predispositional model*. That is, these studies are an alternative way to identify assumed "premorbid" factors, although there are substantial problems with this approach, as outlined below. In this type of study, retrospective reports are used in an effort to establish the time line between an antecedent (e.g., personality trait) and an outcome (e.g., eating disorder). With retrospective assessment, individuals are asked to recall events (or personality functioning) at an earlier point in time. The goal of the assessment is to generate information about the disorder of interest (e.g., eating disorder) and the risk factor (e.g., personality trait) after both have occurred. The design strategy typically used is a case-control study in which a target sample is identified (e.g., eating disordered or depressed) and compared to a control sample (e.g., normal or psychopathology control group). Potential predictors or risk factors (e.g., personality traits) that may differentiate the groups are assessed retrospectively.

The retrospective design was previously used in early studies examining the relationship between personality and mood disorders. For example, Hirschfeld and Klerman (1979) conducted a study in which they asked subjects to complete personality questionnaires "according to their usual self." The subjects were asked to recall a premorbid interval of personality functioning while they were in the midst of a depressed episode. Strictly speaking, retrospective reports permit one to identify only correlates (Jacobi et al., 2004; Kazdin et al., 1997). In this case, the correlate is recall of previous personality functioning. The problems with this technique include selective recall, inaccurate recall, and other retrospective reporting inaccuracies (Kazdin et al., 1997). This method can be improved by having collateral informants report on subjects' prior personality characteristics, although family members or other informants may also be subject to the same problems of biased reporting.

2.4. Cross-sectional design

Cross-sectional designs, although commonly utilized, are clearly very limited in their ability to evaluate all but one of the previously described models. While the ideal test of the pathoplasty model is with a prospective design, as discussed previously, an alternative, much less rigorous test of this model could also be obtained with a traditional, cross-sectional comparison of eating disordered individuals with differing levels of a particular personality trait (e.g., "high-impulsive" versus "low-impulsive" bulimic individuals). If, for example, the pathoplasty model applies to impulsivity and eating disorders, these individuals would be expected to have a different clinical presentation (e.g., suicidal or not) depending upon the degree to which they possess the trait of impulsivity.

2.5. Family study design

Family studies can help distinguish among *predispositional*, *complication*, and *common-cause models*. There is substantial evidence that eating disorders, as well as most other forms of psychopathology, cluster in families (Gershon et al., 1984; Halmi et al., 1991; Hudson, Pope, Jonas, Yurgelun-Todd, & Frankenburg, 1987; Kassett et al., 1989; Lilenfeld et al., 1998; Logue, Crowe, & Bean, 1989; Stern et al., 1992; Strober, Freeman, Lampert, Diamond, & Kaye, 2000; Strober, Lampert, Morrell, Burroughs, & Jacobs, 1990). Likewise, personality traits also appear to be familial (e.g., Carmichael & McGue, 1994; Livesley, Jang, & Vernon, 2003). Thus, family members have, on average, greater liability for the illness that affects the proband.

To test for shared familial etiology between an eating disorder and a personality disorder, one would ideally use a family study design with the following four groups: eating disorder probands with the personality disorder, eating disorder probands without the personality disorder, non-eating disorder probands with the personality disorder, and non-eating disorder probands without the personality disorder (i.e., never-ill controls). Unfortunately, such a study has yet to be completed. If an eating disorder and personality disorder share a common cause, one would expect elevated rates of both the eating disorder and personality disorder among the relatives of all three clinical groups compared to the relatives of never-ill controls.

Furthermore, one can actually discriminate between the two types of common-cause models (third-variable vs. spectrum) using the family study design. Support for the "third-variable model" would be found if the *comorbid condition* (i.e., eating disorder+personality disorder) were equally common among relatives of all three clinical groups (i.e., probands with pure eating disorder, pure personality disorder, comorbid eating disorder+personality

disorder) and higher than among relatives of control probands. This would be the case if we assume that the risk factors for the two disorders are largely overlapping; thus, having both disorders does not confer any greater risk for both conditions. Alternatively, if we assume that risk factors for the two disorders are only partially overlapping and there were instead a substantial amount of unique risk factors for each disorder, then the comorbid condition would, in fact, be highest among relatives of the comorbid probands. So, this prediction is dependent upon the extent to which one assumes the two disorders have overlapping risk factors and the extent to which there are remaining unique risk factors.

Support for the "third-variable model" also would be found if the *personality disorder* were equally common among relatives of pure personality disorder and comorbid eating disorder+personality disorder probands, both of which should be higher than among relatives of pure eating disorder probands, which should be higher than among relatives of control probands. The reason for this is that there are shared and unique vulnerability factors; the personality disorder probands and their relatives have both the shared vulnerability factors between eating disorder and personality disorder, as well as the unique vulnerability factor(s) for personality disorder. However, the eating disorder probands and their relatives have only the shared risk factor(s) between eating disorder and personality disorder and no unique personality disorder risk factors since the probands have a pure eating disorder.

Finally, applying the same logic, support for the "third variable model" also would be found if the *eating disorder* was equally common among relatives of pure eating disorder and comorbid eating disorder+personality disorder probands, both of which should be higher than among relatives of pure personality disorder probands, which should be higher than among relatives of control probands.

When the etiological factors between two disorders are largely overlapping, and there is phenomenological similarity between them, then they may lie on the same continuum. Specifically, support for the "spectrum model" would be found if the *comorbid condition* were equally common among relatives of pure eating disorder and comorbid eating disorder+personality disorder probands, both of which should be higher than among relatives of pure personality disorder probands, which should be higher than relatives of control probands. This is because we are assuming that the eating disorder (e.g., anorexia nervosa) is the more severe form of pathology and the personality disorder (e.g., obsessive-compulsive personality disorder (OCPD)) is the less severe form; however, both are assumed to lie on the same continuum or "spectrum." This is analogous to the assumption that schizophrenia and schizotypal personality disorder lie on the same continuum, with the former being the more severe condition (Lenzenweger, 1998). Thus, the relatives of anorexic probands will have a higher "loading" of whatever risk factor(s) contribute to this pathology. Hence, rates of anorexia+OCPD (or either one alone) will be highest in the relatives of anorexic probands (whether they have "pure" anorexia or anorexia comorbid with OCPD). Certainly, an argument could be made that in some instances, the personality disorder may be the more severe manifestation of the disorder, in the case of borderline personality disorder and bulimia nervosa, for instance. The same logic applies, but the predictions would be in the opposite direction.

Support for the "spectrum model" also would be found if the *personality disorder* were equally common among relatives of pure eating disorder and comorbid eating disorder+personality disorder probands, both of which should be higher than among relatives of pure personality disorder probands, which should be higher than among relatives of control probands. Again, the reason for this is that the relatives of eating disorder probands have a higher "loading" of whatever risk factor(s) contribute to the pathology (again, assuming in this case that the eating disorder is the more severe condition on the continuum).

Finally, applying the same logic, support for the "spectrum model" also would be found if the *eating disorder* was equally common among relatives of pure eating disorder and comorbid eating disorder+personality disorder probands, both of which should be higher than among relatives of pure personality disorder probands, which should be higher than among relatives of control probands. In this case, frequency of eating disorder among relatives would be the same for both the "third-variable" and "spectrum" models.

Hypotheses based upon a dimensional measurement of personality could also be tested with a family study approach. For example, to test for shared etiology between an eating disorder and a personality trait measured dimensionally, rather than categorically, one would study rates of eating disorders and levels of the personality trait of interest among the relatives of the following four groups: eating disorder probands with high levels of the trait, eating disorder probands with low levels of the trait, non-eating disorder probands with high levels of the trait, and non-eating disorder probands with low levels of the trait. Again, no studies of this type have been completed to date.

The four proband groups discussed previously (i.e., pure eating disorder, pure personality disorder, comorbid eating and personality disorder, control) and their relatives may also allow us to distinguish between *common cause and predispositional* models if we are considering familial risks only. Specifically, personality disorder elevations in the never-eating disordered family members of eating disordered probands compared to the never-eating disordered family members of control probands would be consistent with the predispositional or the common cause (specifically, the spectrum) model. One would then need to examine rates of eating disorders among the non-personality disordered relatives of personality disorder probands in order to discriminate between predispositional and spectrum models. Elevated rates of eating disorders among these relatives (compared to eating disorder rates among non-personality disordered relatives of control probands) would support the spectrum model. In contrast, if the personality disorder were a predisposing factor for the eating disorder, but did not share a common cause, then rates of eating disorders among these two groups of relatives would not significantly differ.

A combination of a family study and recovered study design is another way to evaluate support for the *predispositional and complication models*. Personality elevations in the previously eating disordered (i.e., recovered) family members of eating disordered probands compared to the never-eating disordered family members of eating disordered probands would support either the scar-effect or the predispositional model. However, personality elevations among the currently eating disordered family members of eating disordered probands compared to the recovered family members of eating disordered probands would definitively support the state-effect model.

2.6. Multivariate twin study design

Finally, multivariate twin studies are an alternative, powerful methodology to use to evaluate the *common-cause model*. By examining associations between personality traits (or disorders) and eating disorders (or disordered eating) among monozygotic compared to dizygotic twin pairs, one can not only gather information about whether these conditions share a common underlying cause, but also to what extent the nature of the shared cause is accounted for by shared genes and/or shared environment. Thus, after evaluating associations between personality traits and disordered eating, this study design allows us to determine the extent to which genetic and environmental factors underlie these relationships. Specifically, one can examine whether common genes and/or environmental factors account for the total variance in disordered eating or whether there are genetic and environmental influences on disordered eating that are independent of those for personality.

The relative strength of all six reviewed methodological approaches for testing the four theoretical models are briefly summarized in Table 1.

3. Empirical tests of the models

This review is confined to studies using one of the previously defined methodological approaches, such that an evaluation of at least one of the aforementioned models of the relationship between personality and eating disorders

Table 1
Methodological approaches used to test theoretical models of the eating disorder and personality relationship

Model	Methodological approaches for testing model	Strength of test
Predispositional	Prospective study design	***
	Recovered study design	**
	Family study/recovered study design combination	**
	Retrospective study design	*
Complication (state effect; scar effect)	Prospective study design	***
	Recovered study design	**
	Family study/recovered study design combination	**
Common cause (spectrum; third variable)	Family study design	***
	Multivariate twin study design	***
Pathoplasty	Prospective study design	***
	Cross-sectional study design	*

Note. *Adequate; **good; ***excellent.

was possible. Thus, many cross-sectional descriptive studies of personality traits and/or pathology among individuals with eating disorders were excluded (unless their design allowed for evaluation of the pathoplasty model), because these data could not discriminate among the alternative models described above. A summary of the empirical studies reviewed below appears in Table 2.

3.1. Predispositional model

3.1.1. Prospective studies

Several true prospective studies of personality have been conducted in the field of eating disorders. This methodological design is time-consuming and costly, yet is the most powerful way of evaluating the hypothesis that certain premorbid personality traits increase the risk of later developing an eating disorder. Because the prevalence of eating disorders is low, most of these prospective studies predicted abnormal eating attitudes or "disordered eating" rather than diagnosable eating disorders.

Two of the first such longitudinal, epidemiological studies with adolescent girls found no personality predictors of disordered eating over time (Attie & Brooks-Gunn,1989; Patton, Johnson-Sabine, Mann, & Wakeling, 1990). In contrast, the prospective study by Leon and colleagues found that poor interoceptive awareness predicted eating disorder risk status one year later among an epidemiological sample of junior high and high school students (Leon, Fulkerson, Perry, & Early-Zald, 1995). In addition, these authors later found that negative affect (which encompassed a broad array of variables, including "ineffectiveness," as well as negative emotionality) predicted increased eating disorder risk status after a three to four year follow-up period (Leon, Fulkerson, Perry, Keel, & Klump, 1999). Only some of these adolescents had a diagnosable eating disorder at the follow-up assessment, but all had clinically significant symptoms. Importantly, there were very few subjects with eating pathology at time 1 and the results did not change when these subjects were excluded from analyses.

A few studies have examined the relationship between childhood temperament and the later development of eating problems during adolescence. Similar to the findings of Leon et al. (1999), Martin and colleagues (2000) found that high negative emotionality measured from infancy onward was most associated with eating disorder risk status at 12–13 years of age. Unfortunately, they did not evaluate presence of eating pathology at Time 1. Killen et al. (1996) found that the temperamental factors of distress and fear (i.e., neuroticism), as well as ineffectiveness and poor interoceptive awareness, predicted partial eating disorder syndromes over a four-year period of time in a large community sample of high school girls. These findings held true after controlling for initial eating disorder symptoms.

There have been two recent, large, true prospective studies with subjects free of an eating disorder at baseline. One followed females aged 12–21, while another followed women aged 18–30. Both studies also found that neuroticism (negative emotionality) predicted the development of eating disorders over time (Cervera et al., 2003; Ghaderi & Scott, 2000).

Several additional prospective studies have been conducted with specific populations considered to be at increased risk for eating disorders. Garner, Garfinkel, Rockert, and Olmsted (1987) found that only drive for thinness and body dissatisfaction (not traditionally considered personality traits) predicted the development of eating disorders over a two to four year period among 35 adolescent girls enrolled in a highly competitive ballet school. Striegel-Moore, Silberstein, French, and Rodin (1989) found that the personality trait of ineffectiveness (i.e., feeling inadequate, insecure, worthless and not in control of one's life) was associated with a worsening of disordered eating symptoms (e.g., dieting, bingeing, purging) among female college students from the beginning to the end of their freshman year. Vohs, Bardone, Joiner, Abramson, and Heatherton (1999) found that perfectionism assessed during the senior year of high school predicted the development of bulimic symptoms during the freshman year in college. Importantly however, perfectionism functioned as a risk factor only when the individual considered herself to be overweight and also had low self-esteem (Vohs et al., 1999). The latter two studies controlled for initial eating pathology, while the Garneret et al. (1987) study did not.

Finally, Rastam (1992) conducted a study that might best be classified as "quasi-prospective." Her colleague performed a blind, systematic chart review of the premorbid personality functioning (i.e., DSM-III-R Axis II diagnoses were assigned) of 51 adolescents with anorexia nervosa (some of whom were identified through a population-based study of over 4000 children and some of whom were referred for evaluation of a potential eating disorder) in an attempt to identify their personality functioning before the onset of the eating disorder. Anorexic

Table 2
Findings from empirical tests of theoretical models of the eating disorder and personality relationship

Model tested	Methodological design	Authors	Participants	Findings
Predispositional	Prospective	Attie and Brooks-Gunn (1989)	193 adolescent girls	*No personality predictors* of disordered eating over a 2 year period
	Prospective	Patton et al. (1990)	176 adolescent girls	*No personality predictors* of disordered eating over a 1 year period
	Prospective	Leon et al. (1995)	664 adolescent girls and 668 boys	*Poor interoceptive awareness* predicted increased ED risk status among girls 1 year later
	Prospective	Leon et al. (1999)	726 adolescent girls and 698 adolescent boys	*Negative affect* predicted increased ED risk status among girls 3–4 years later
	Prospective	Martin et al. (2000)	597 girls and 631 boys	*Negative emotionality* and *persistence* assessed during childhood predicted increased ED risk status, especially among girls
	Prospective	Killen et al. (1996)	825 adolescent girls	*Neuroticism*, *ineffectiveness* and *poor interoceptive awareness* predicted partial syndrome EDs over a 4 year period
	Prospective	Cervera et al. (2003)	2509 females aged 12–21	*Neuroticism* predicted ED development (AN, BN, EDNOS) over an 18 month period
	Prospective	Ghaderi and Scott (2000)	856 females aged 18–30	*Neuroticism* predicted ED development over a 2 year period
	Prospective	Garner et al. (1987)	35 adolescent girls from a competitive ballet school	*No personality traits* (just drive for thinness and body dissatisfaction) predicted ED development over a 2–4 year period
	Prospective	Striegel-Moore et al. (1989)	403 female and 546 male college freshman	*Ineffectiveness* predicted worsening of disordered eating symptoms over a 1 year period
	Prospective	Vohs et al. (1999)	342 females	*Perfectionism* predicted bulimic symptoms from high school senior year to college freshman year only among those with low self-esteem and overweight self-perception
	Quasi-prospective	Rastam (1992)	51 adolescents with AN and 51 matched psychopathology control adolescents	More premorbid *OCPD traits* (assessed by blind chart review) among AN than control group
	Recovered	Bastiani et al. (1995)	19 females with RAN (11 ill; 8 weight restored for 4 weeks); 10 healthy CW	*Perfectionism* elevated in those recovered from RAN
	Recovered	Srinivasagam et al. (1995)	20 females recovered from AN (≥1 year); 16 healthy CW	*Perfectionism, obsessive-compulsive traits (symmetry and exactness)* elevated in those recovered from AN
	Recovered	Kaye et al. (1998)	30 females recovered from BN (≥1 year); 31 healthy CW	*Perfectionism*, *ineffectiveness*, *poor interoceptive awareness*, *obsessive-compulsive traits (symmetry and exactness)* elevated in those recovered from BN
	Family+Recovered	Lilenfeld et al. (2000)	47 females with BN (37 ill; 16 recovered for ≥1 year); 44 healthy CW	*Perfectionism*, *poor interoceptive awareness* elevated in those recovered from BN
	Recovered	Stein et al. (2002)	11 females recovered from BN (for ≥1 year); 15 healthy CW	*Perfectionism*, *ineffectiveness*, *stress reaction and negative emotionality* elevated in those recovered from BN

	Family+Recovered	Casper (1990)	25 females recovered from AN were assessed 8–10 years after treatment, as well as their never-ED sisters ($n=15$) and CW ($n=23$)	Women recovered from AN had elevated *ineffectiveness*, *stress reactivity*, *restraint*, *harm avoidance*, *conformance to authority*, *worse interoceptive awareness*, *and lower well-being*, *positive emotionality and danger seeking* than CW. Women recovered from AN had elevated *ineffectiveness*, *interpersonal distrust*, *and lower restraint*, *danger seeking and impulsivity* than their sisters.
	Recovered	Pollice et al. (1997)	22 underweight ANs (13 RAN, 9 ANBP) females assessed <1 month of inpatient admission and again after short term weight-restoration (<1 month >90% IBW); a different group of 26 AN (13 RAN, 13 ANBP) long-term (6 months–10 years) weight restored; 18 CW	*Obsessionality* worst among ill women, better when short-term restored, even better when long-term restored, and best among CW
	Recovered	Gillberg et al. (1995)	See Rastam (1992)	*Obsessive-compulsive and avoidant personality disorders* elevated in women with adolescent-onset AN, *some* of whom were recovered after 5 year follow-up period (6 years after AN onset)
	Recovered	Matsunaga et al. (2000)	10 women recovered from AN, 28 women recovered from BN, and 16 women recovered from AN+BN (all for ≥1 year)	Persistence of *Cluster C personality disorders* among all groups and increased *Cluster B symptomatology* among those with prior bulimic symptoms
	Retrospective	Fairburn et al. (1997)	Community female sample of 102 BN, 204 healthy CW, and 102 with other psychiatric disorders	Premorbid (by retrospective report) *perfectionism and negative self-evaluation* distinguished BN women from both control groups
	Retrospective	Fairburn et al. (1999)	Community female sample of 67 AN, 102 BN, 204 healthy CW, and 102 with other psychiatric disorders	Premorbid (by retrospective report) *perfectionism* distinguished AN women from both control groups; premorbid *negative self-evaluation* distinguished AN women from BN women and both control groups
	Retrospective	Anderluh et al. (2003)	44 AN, 28 BN, 28 CW	Retrospectively reported childhood *obsessive-compulsive personality traits* predicted AN and BN development
	Family+Retrospective	Karwautz et al. (2002)	45 families with 2 sisters discordant for AN	Retrospectively reported elevated rates of premorbid *Cluster C personality disorders*, *harm avoidance*, *persistence* and *perfectionism*, as well as decreased *novelty seeking* and *self-directedness* among anorexic compared to never-ill sisters
	Family+Recovered	Lilenfeld et al. (2000)	Summarized above	*Perfectionism, ineffectiveness and interpersonal distrust* elevated in the never-ED relatives of ED individuals compared to the never-ED relatives of CW
Complication	Recovered	Keys et al. (1950)	Healthy young men whose food intake was restricted, resulting in the loss of ~25% of total weight	*Rigidity and obsessionality* developed in response to a semi-starved state
	Recovered	Pollice et al. (1997)	Summarized above	Summarized above
	Recovered	Kaye et al. (1998)	Summarized above	Summarized above
	Recovered	Srinivasagam et al. (1995)	Summarized above	Summarized above

(continued on next page)

Table 2 (*continued*)

Model tested	Methodological design	Authors	Participants	Findings
	Recovered	Strober (1980))	22 adolescent females with first-episode AN reassessed after 6 months of inpatient treatment	*Extraversion* increased (and a trend toward decreased *neuroticism*) after weight restoration
	Recovered	Stonehill and Crisp (1977)	45 AN patients	*Extraversion* increased and *neuroticism* decreased after weight restoration
	Recovered	Crisp et al. (1979)	18 AN patients	*Extraversion* increased and *neuroticism* decreased after weight restoration
	Recovered	Ames-Frankel et al. (1992)	34 inpatients and 49 outpatients with BN entering treatment	*Cluster B and Cluster A personality disorder dimensional scores* decreased following binge frequency reduction
	Recovered	Kennedy et al. (1990)	19 AN, 16 BN, 9 AN+BN	*Cluster A (paranoid, schizoid, schizotypal), B (borderline) and C (avoidant, dependent, passive-aggressive) personality dis order dimensional scores* decreased from intake to discharge
	Family+Recovered	Casper (1990)	Summarized above	Summarized above
	Family+Recovered	Lilenfeld et al. (2000)	Summarized above	Elevated *absorption* and *alienation* among ill vs. recovered BN women. Diminished *interoceptive awareness*, *heightened stress reactivity* and *perfectionistic doubting of actions* among previously ill relatives of BN vs. their never ill relatives
Common cause	Family	Lilenfeld et al. (1998)	26 RAN (93 1st degree relatives) and 44 CW (190 1st degree relatives)	Elevated rates of *OCPD* among relatives of RAN probands, regardless of OCPD comorbidity in the proband
	Family	Strober (1998)	152 RAN (510 1st degree relatives) and 181 CW (523 1st degree relatives)	Elevated rates of *OCPD* among relatives of RAN probands, regardless of OCPD comorbidity in the proband
	Family+Recovered	Casper (1990)	Summarized above	Higher *stress reactivity* among sisters of women recovered from AN than CW
	Family	Fassino et al. (2002)	50 RAN (23 fathers, 25 mothers) and 60 CW (20 fathers, 20 mothers) from the community	Low *self-directedness* in AN probands and both parents compared to CW probands and parents. Fathers of AN probands also had elevated *harm avoidance* and *reward dependence*, but low *persistence* compared to CW fathers.
	Family	Fassino et al. (2003)	28 BN (23 fathers, 28 mothers) and 29 CW (27 fathers, 29 mothers) from the community	Low *self-directedness* in BN probands and their mothers compared to CW probands and mothers. Fathers of BN probands had low *persistence* compared to CW fathers
	Family	Woodside et al. (2002)	107 AN, 107 mothers, 78 fathers; 248 CW	Elevated *perfectionism* among mothers of AN probands and fathers of RAN probands compared to CW
	Family	Steiger et al. (1995)	15 RAN, 10 AN-BP, 40 BN, 10 EDNOS, and 173 1st degree relatives	*No personality traits* elevated among relatives of ED probands
	Family	Carney et al. (1990)	25 BN (46 1st degree relatives) and 25 CW (61 1st degree relatives)	*No personality traits* elevated among relatives of ED probands

	Twin	Wade et al. (2000)	537 monozygotic and 344 dizygotic female twin pairs	*Neuroticism* and a general measure of disordered eating were modestly positively correlated, primarily due to nonshared (unique) environmental risk factors. Disordered eating did not share genetic risk factors with any personality variables
	Twin	Klump et al. (2002)	256 female adolescent twin pairs	Shared genetic variance accounted for a modest proportion of the association between disordered eating and *negative emotionality, positive emotionality* and *constraint*
Pathoplasty	Prospective/ longitudinal	Strober et al. (1997)	95 adolescents hospitalized for AN	*OCPD* traits predicted worse outcome over a 10–15 year period
	Prospective/ longitudinal	Sutandar-Pinnock et al. (2003)	49 patients hospitalized for AN	Less *perfectionism* predicted better response to treatment and better outcome at 6–24 month follow-up
	Prospective/ longitudinal	Fahy et al. (1993)	39 outpatient women with BN	*Personality disorder* predicted poorer response to CBT (but nonsignificantly so after controlling for depression and BMI)
	Prospective/ longitudinal	Rossiter et al. (1993)	71 women with BN	*Cluster B personality disorder* traits predicted poorer treatment response to CBT (at 4 months and 1 year post-treatment), medication or both combined
	Prospective/ longitudinal	Johnson et al. (1990)	40 treatment-seeking women with BN (21 with comorbid BPD)	More BN patients with *BPD* continued to meet BN criteria 1 year after beginning treatment, compared to those without BPD
	Prospective/ longitudinal	Wonderlich et al. (1994)	8 RAN, 8 ANBP, 14 BN	*BPD* (when measured dimensionally but not categorically) predicted a more negative outcome after 5 year follow-up
	Prospective/ longitudinal	Grilo et al. (2003)	23 BN and 69 EDNOS females, most of whom were treatment-seeking or currently in treatment	Neither presence nor severity of comorbid *personality disorders* were related to the natural course of the ED
	Prospective/ longitudinal	Bulik et al. (1999)	98 BN women	Increased *self-directedness* predicted rapid response to CBT for BN at 1 year follow-up
	Prospective/ longitudinal	van der Ham et al. (1998)	49 adolescent ED patients	*Maturity fears* at admission among those with RAN predicted poor outcome after 4 years
	Prospective/ longitudinal	Edwin et al. (1988)	39 RAN and 27 ANBP discharged inpatients	Elevated MMPI *Hysteria* scale scores predicted better outcome among RAN patients over 8–60 month follow-up period
	Prospective/ longitudinal	Dancyger et al. (1997)	52 AN female inpatients	*MMPI* scores on nearly all *clinical scales* at discharge for hospitalized anorexic patients were correlated with outcome 10 years later
	Prospective/ longitudinal	Schork et al. (1994)	59 AN female inpatients	*MMPI* scores on nearly all *clinical scales* at discharge for hospitalized anorexic patients were correlated with outcome 10 years later
	Cross-sectional	Bulik et al. (2000)	70 AN females referred for treatment (assessed an average of 15 years after AN onset) and 98 community CW	Higher *harm avoidance* and lower *self-directedness* found among chronically ill AN patients vs. those who had fully recovered
	Cross-sectional	Rosenvinge and Mouland (1990)	30 AN female inpatients (assessed an average of 14 years after hospitalization)	More *histrionic personality disorder* among the good outcome group and more *BPD* among the poor outcome group at follow-up

Note. ED = Eating Disorder, AN = Anorexia Nervosa, BN = Bulimia Nervosa, EDNOS = Eating Disorder Not Otherwise Specified, RAN = Anorexia Nervosa-Restricting type; ANBP = Anorexia Nervosa-Binge eating/purging type, IBW = Ideal Body Weight, OCPD = Obsessive Compulsive Personality Disorder, CBT = Cognitive Behavioral Therapy, BMI = Body Mass Index, BPD = Borderline Personality Disorder, MMPI = Minnesota Multiphasic Personality Inventory.

individuals were significantly more likely than a matched psychopathology control group to have previously displayed features of obsessive compulsive personality disorder, such as rigidity, inflexibility, and perfectionism. However, it is not clear whether all adolescents were free of an eating disorder at the time the chart notes containing personality information were made.

In summary, these prospective studies represent the best attempts to identify personality traits that may be predisposing factors for the later development of eating disorders. These identified traits include negative emotionality (neuroticism), poor interoceptive awareness, perfectionism, ineffectiveness, drive for thinness, and obsessive-compulsive personality traits. However, the existing studies are limited by several factors. First, few of these prospective studies have conducted a thorough assessment of premorbid personality traits with a comprehensive instrument such as the Multidimensional Personality Questionnaire (Tellegen, 1982) or the NEO Personality Inventory (Costa & McCrae, 1992). Second, many of these studies did not rule out the possibility that subjects may have had eating pathology while their "premorbid" personality assessment was conducted. Thus, if eating disorder symptoms are not controlled at index and had a scarring effect, prospective and recovered designs would spuriously conclude such persisting personality traits to be predispositional. Third, the follow-up periods are often brief. Finally, outcome measures vary substantially and only three prospective studies (Cervera et al., 2003; Ghaderi and Scott, 2000; Garner et al., 1987) were able to predict the development of diagnosable eating disorders, as opposed to "disordered eating." Again, these first two large, epidemiological studies found that neuroticism predicted the development of eating disorders over time.

3.1.2. Recovered studies

Because of the logistical problems inherent in conducting true prospective research with eating disorders, several researchers have employed a recovered design to test the predispositional model. Personality traits that are present after recovery from the eating disorder are presumed to be premorbid factors.

There are several personality traits that have been implicated in prospective research and also found to be elevated among eating disordered individuals after recovery. These include perfectionism (Bastiani, Rao, Weltzin, & Kaye, 1995; Kaye et al., 1998; Lilenfeld et al., 2000; Srinivasagam et al., 1995; Stein et al., 2002; Vohs et al., 1999), ineffectiveness (Casper, 1990; Kaye et al., 1998; Stein et al., 2002; Striegel-Moore et al., 1989), poor interoceptive awareness (Casper, 1990; Kaye et al., 1998; Leon et al., 1999; Lilenfeld et al., 2000), negative emotionality/neuroticism (Casper, 1990; Cervera et al., 2003; Ghaderi & Scott, 2000; Killen et al., 1996; Leon et al., 1999; Martin et al., 2000; Pollice, Kaye, Greeno, & Weltzin, 1997; Stein et al., 2002), restraint (Casper, 1990; Rastam, 1992), and obsessionality (Kaye et al., 1998; Pollice et al., 1997; Rastam, 1992; Srinivasagam et al., 1995). Additional personality traits that have been found to be elevated among recovered individuals, but not tested in prospective designs, include harm avoidance and conformance to authority (Casper, 1990). Casper (1990) also found similarly low levels of positive emotionality and high levels of traditionalism and constraint among both acutely ill restricting anorexic patients and those who were recovered, suggesting that these traits are not "state-effects" and may possibly be risk factors.

A few studies have examined personality disorders, as opposed to personality traits, among recovered individuals. Gillberg, Rastam, and Gillberg (1995) found elevated rates of obsessive-compulsive and avoidant personality disorders among a cohort of individuals with adolescent-onset anorexia nervosa, *some* of whom were recovered from their illness. Matsunaga and colleagues (2000) also found the persistence of Cluster C personality disorders among women who were recovered from either anorexia or bulimia nervosa for at least one year. In addition, they found increased Cluster B symptomatology only among those recovered individuals who had previously evidenced bulimic symptoms when they were ill.

In summary, these recovered studies suggest that potential predispositional factors include negative emotionality/neuroticism, poor interoceptive awareness, perfectionism, ineffectiveness, restraint, and obsessionality, all of which have also been implicated in prospective study designs. In contrast, harm avoidance and conformity to authority have only been tested in recovered designs thus far and would need prospective assessment in order to try to disentangle premorbid factors from scar effects. With regard to personality disorders, Cluster C disorders are common among recovered eating disorder patients, while Cluster B disorders and symptoms are common among those recovered from bulimic symptoms specifically. Additional prospective and family study work suggest that obsessive compulsive personality disorder may be a predisposing factor for restricting-type anorexia nervosa in particular.

3.1.3. Retrospective studies

Retrospective reports can investigate potential predisposing personality traits by utilizing a case-control design. Personality traits that are more common among those who have developed an eating disorder, compared to matched controls, can be considered potential predisposing factors if the traits are reported to have been present before the onset of the illness. Fairburn and colleagues conducted two such studies utilizing a community sample. "Premorbid" (as deemed by retrospective report) perfectionism and negative self-evaluation were found to distinguish those with anorexia nervosa and those with bulimia nervosa, from psychiatric, as well as healthy controls (Fairburn, Cooper, Doll, & Welch, 1999; Fairburn, Welch, Doll, Davies, & O'Connor, 1997). Another more recent study also found retrospectively reported childhood obsessive-compulsive personality traits to have high predictive value for the development of anorexia and bulimia nervosa when compared to healthy control women (Anderluh, Tchnaturia, Rabe-Hesketh, & Treasure, 2003). Finally, a study in which the comparison group consisted of the sisters of anorexic individuals found that the ill sister retrospectively reported having elevated rates of premorbid Cluster C personality disorders, harm avoidance, persistence, and perfectionism, and decreased rates of novelty seeking and self-directedness compared to her never-ill sister (Karwautz, Rabe-Hesketh, Collier, & Treasure, 2002).

3.1.4. Family studies

While not a replacement for prospective longitudinal designs, the combination of a family and recovered study design allows for a rigorous evaluation of the predispositional model (Grilo, 2002). Personality traits that are elevated in the never-eating disordered family members of eating disordered individuals may represent predisposing factors. Lilenfeld and colleagues (2000) found evidence of this among the first-degree relatives of bulimic women compared to the first-degree relatives of control women for the three traits of perfectionism, ineffectiveness, and interpersonal distrust. The first two of these traits have also been previously identified in prior premorbid and recovered studies as potential predisposing factors for the development of eating disorders (Bastiani et al., 1995; Casper, 1990; Kaye et al., 1998; Srinivasagam et al., 1995; Stein et al., 2002; Striegel-Moore et al., 1989; Vohs et al., 1999).

3.2. Complication model

3.2.1. Recovered studies

Recovered studies are a rigorous test of the complication model. Personality trait elevations in currently ill, compared to recovered subjects, would provide strong support for the state-effect model.

Experimental studies of imposed starvation with previously healthy individuals suggest that rigidity and obsessionality may result from a starved state (Keys et al., 1950). Indeed, obsessionality appears to be highest among currently ill subjects, next highest among those who are recovered from an eating disorder, and lowest among those who were never ill (Kaye et al., 1998; Pollice et al., 1997; Srinivasagam et al., 1995). Neuroticism has been shown to decrease and extraversion to increase after weight restoration among individuals with anorexia nervosa (Crisp, Hsu, & Stonehill, 1979; Stonehill & Crisp, 1977; Strober, 1980). In addition, emotional lability and other indices of behavioral disinhibition decrease following reductions in bingeing and purging (Ames-Frankel et al., 1992; Kennedy, McVey, & Katz, 1990), suggesting that impulsive traits may be at least partially secondary to active bulimia nervosa (Grilo, 2002).

Finally, elevated ineffectiveness, drive for thinness, restraint, harm avoidance, conformance to authority, and poor interoceptive awareness has been observed among long-term recovered anorexic subjects compared to never-eating disordered women (Casper, 1990), but it is impossible to definitively determine which may be "scar effects" and which may be predisposing factors at the present time. However, ineffectiveness, poor interoceptive awareness, drive for thinness and restraint have all been implicated in prospective research, suggesting that these are more likely to be predisposing factors. Future prospective research should examine these traits in order to more definitively make this distinction.

3.2.2. Family studies

Family studies can be utilized to evaluate complication model hypotheses only if recovery data are embedded within the study design. Lilenfeld et al. (2000) found that emotional responsivity to the environment ("absorption")

and a sense of alienation were elevated among currently ill, compared to recovered bulimic probands, suggesting that these may be "state effects" of the illness. In contrast, diminished interoceptive awareness, heightened stress reactivity, and perfectionistic doubting of actions were found among the previously eating disordered relatives of bulimic probands compared to their never ill relatives, suggesting that these may be "scar effects" of the illness. These traits were considered more likely to be scar effects than predisposing factors because they were not significantly greater among the never-ill relatives of bulimic probands compared to the never-ill relatives of controls. Thus, these personality traits may be consequent to, and/or exacerbated by, having experienced an eating disorder.

3.3. Common cause model

3.3.1. Family studies

Family studies are one of the strongest tests of the hypothesis that personality traits/disorders and eating disorders share an underlying familial cause. Elevated rates of both the eating disorder and personality disorder among the relatives of probands who have the comorbid condition (i.e., eating disorder plus personality disorder) and both pure groups (eating disorder only; personality disorder only) compared to the relatives of normal control probands would provide support for the common cause model. Unfortunately, no studies in the field have yet tested the hypothesis in this way as has been done, for instance, with major depression and personality disorders (McGlashan et al., 2000). However, two research groups have attempted to utilize a modification of this approach. These two studies have included the comorbid group, the pure eating disorder group and the normal control group, but were missing the pure personality disorder group (Lilenfeld et al., 1998; Strober, 1998). Both have found identical support for the common cause model for obsessive-compulsive personality disorder (OCPD) and restricting-type anorexia nervosa. The relatives of anorexic probands were found to have elevated rates of OCPD (compared to the relatives of normal controls), regardless of the presence of OCPD in the proband herself, suggesting that these two disorders share some common familially based etiological factor(s) (e.g., Lilenfeld et al., 1998; Strober, 1998). However, because there was no pure OCPD proband group, it is not possible to distinguish between the two types of common-cause models, namely "third-variable" and "spectrum" models.

Several studies have attempted to test the hypothesis of shared etiology between an eating disorder and a personality trait measured dimensionally, rather than categorically. However, a true test of this has never been done, which would require studying relatives of the following four groups: eating disorder probands with high levels of the trait, eating disorder probands with low levels of the trait, non-eating disorder probands with high levels of the trait, and non-eating disorder probands with low levels of the trait. In a study which attempts to address this question with a different design, Casper (1990) studied the personality traits of women who had recovered from anorexia nervosa, their never-eating disordered sisters, and control women. The former anorexic subjects were found to be less impulsive and more restrained than their sisters. Interestingly, the recovered anorexic patients' sisters reported greater stress reactivity than normal controls, suggesting that this may be a familial trait of potential shared etiological importance with anorexia nervosa.

A recent family study of temperament and character traits among parents of individuals with anorexia and bulimia nervosa found that probands and parents all had low levels of self-directedness, while fathers specifically had low levels of persistence (Fassino et al., 2002, 2003). Fathers of anorexic individuals also had high levels of harm avoidance and reward dependence. However, this study did not assess eating disorders in family members. Therefore, while it is possible that there is shared etiology between these personality traits and eating disorders, this study design does not allow for a rigorous evaluation of this hypothesis. Another similar investigation from a larger, multi-site genetic study of eating disorders found that mothers of anorexic probands and fathers of probands with restricting-type anorexia nervosa specifically had elevated levels of perfectionism compared to control parents (Woodside et al., 2002). Again, eating disorders were not ascertained in parents.

In contrast, Steiger, Stotland, Ghadirian, and Whitehead (1995) and Carney, Yates, and Cizadlo (1990) found no elevated personality traits among the first-degree relatives of eating disorder probands compared to the first-degree relatives of control probands. It should be noted, however, that the Steiger et al. (1995) study involved assessment of relatively few personality traits and the Carney et al. (1990) study was limited by low response rates and no direct contact with relatives.

3.3.2. Mulitvariate twin studies

Multivariate twin studies are another powerful test of the hypothesis that personality traits/disorders and eating disorders share an underlying familial cause. Furthermore, these data can additionally yield information regarding the extent to which the nature of the shared cause is accounted for by shared genes and/or shared environment. Yet, very few multivariate twin studies have been conducted which have focused upon the relationship between personality and a continuous measure of disordered eating, and none have studied diagnosable eating disorders.

Wade and colleagues (2000) found modest, positive relationships between neuroticism and a general measure of disordered eating, which was determined to be primarily the result of common "nonshared" environmental factors (i.e., experiences that are unique to siblings reared in the same family, such as differential parental treatment or life events experienced by one sibling but not the other), rather than common genetic factors. In contrast, Klump, McGue and Iacono (2002) found that shared genetic factors were more likely than environmental factors to contribute to personality (i.e., negative emotionality, positive emotionality, and constraint) and disordered eating phenotypic associations, though this shared genetic variance was admittedly limited. Methodological differences, such as differences in personality constructs (Church, 1994) and measures of eating pathology examined, as well as the nature of the sample and time period upon which the assessment was focused, may account for these two discrepant study findings.

3.4. Pathoplasty model

3.4.1. Prospective studies

Several studies have evaluated the effects of personality on the course and outcome of eating disorders using a prospective design, which is the ideal test of the pathoplasty model. These studies have all found that personality disorders are associated with poorer response to treatment, with the exception of histrionic personality disorder in anorexia nervosa (Steinhausen, 2002). A long-term prospective study of severe anorexia nervosa in adolescents found obsessive-compulsive personality disorder traits to predict worse outcome over a 10–15 year period (Strober, Freeman, & Morrell, 1997). Another six to 24 month prospective study of individuals hospitalized for anorexia nervosa found that lower levels of perfectionism, specifically, was associated with better response to treatment and better outcome at follow-up (Sutandar-Pinnock, Woodside, Carter, Olmsted, & Kaplan, 2003).

Several outcome studies of bulimia nervosa have found personality disorder (especially borderline) comorbidity to predict poorer response to cognitive behavioral therapy (Fahy, Eisler, & Russell, 1993; Johnson, Tobin, & Dennis, 1990; Rossiter, Agras, Telch, & Schneider, 1993). One five year follow-up study found that borderline personality disorder predicted a more negative outcome when measured dimensionally, but not when measured categorically (Wonderlich, Fullerton, Swift, & Klein, 1994). However, for all of these studies, it is possible that borderline personality disorder is more strongly prognostic for general psychiatric symptoms rather than eating disorder symptoms specifically (Steiger & Stotland, 1996). In fact, a recent prospective examination of bulimia nervosa and other eating disorders found that neither the presence nor severity of comorbid personality disorders were related to the natural course of the eating disorder (Grilo et al., 2003).

Several studies have examined the pathoplastic effects of personality traits rather than personality disorders. Bulik et al. (1999) found that higher levels of self-directedness (i.e., being responsible and disciplined; Cloninger, Przybeck, Svrakic, & Wetzel, 1994) predicted rapid response to CBT for bulimia nervosa. van der Ham, van Strein, and van Engeland (1998) found that strong maturity fears predicted poor outcome among restricting anorexic patients after four years.

Finally, two prospective studies examined the effect of Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1948) scores on outcome after inpatient treatment. The first of these studies found that elevated Hysteria scale scores predicted *better* outcome among restricting-type anorexic patients over an eight to 60 month follow-up period (Edwin, Andersen, & Rosell, 1988). The finding of improved outcome in anorexic patients with histrionic personality traits was also highlighted in a recent review (Steinhausen, 2002). Another prospective study found that MMPI scores on nearly all clinical scales at the time of discharge were correlated with outcome after ten years for previously hospitalized anorexic patients (Dancyger et al., 1997; Schork, Eckert, & Halmi, 1994). However, admission MMPI scores were not related to ten year follow-up scores, suggesting that the long-term

outcome of anorexia nervosa was largely unrelated to the severity of psychopathology during the acute phase of the illness.

3.4.2. Cross-sectional studies

A cross-sectional design can provide an alternate, less rigorous test of the pathoplasty hypothesis. Bulik, Sullivan, Fear, and Pickering (2000) found that chronically ill anorexic patients had higher harm avoidance and lower self-directedness, compared to anorexic patients who had fully recovered. Rosenvinge and Mouland (1990) found significantly greater rates of histrionic personality disorder among the "good outcome" group and significantly greater rates of borderline personality disorder among the "poor outcome" group at the time of follow-up from a retrospective outcome study of previously hospitalized individuals with anorexia nervosa. Unfortunately, the cross-sectional study design renders it impossible to determine whether these personality traits/disorders were present premorbidly and contributed to recovery (or lack thereof) or, alternatively, whether they emerged during the course of the illness. However, because these findings support previous results from prospective research, it is likely that they may indeed affect the course and outcome of the eating disorder.

4. Conclusions

4.1. Summary

Prospective studies and other research have implicated negative emotionality, perfectionism, drive for thinness, poor interoceptive awareness, ineffectiveness, and obsessive-compulsive personality traits as likely predispositional factors, which may increase the risk for developing an eating disorder. A recent comprehensive review highlights evidence specifically for negative emotionality, perfectionism, and drive for thinness as risk factors (Stice, 2002). Interoceptive awareness, ineffectiveness, and obsessive-compulsive personality traits were not reviewed because the studies which examined these traits did not meet the rigorous criteria utilized for inclusion in Stice's (2002) meta-analytic review. Another recent comprehensive review supports evidence for all of the above traits as risk factors, with the exception of perfectionism and obsessive-compulsive personality disorder, which were classified as retrospective correlates (Jacobi et al., 2004) because the methodology used in the studies which implicated these traits as risk factors were not true prospective studies. However, growing evidence from quasi-prospective, retrospective, and family study research strongly suggests that perfectionism and other obsessive-compulsive personality traits may indeed be predisposing factors for eating disorders.

No studies have been able to adequately disentangle competing hypotheses well enough to make definitive statements about personality traits that may be "complications" of the eating disorder. The personality traits of stress reactivity, alienation, and absorption (i.e., emotional responsivity to the environment) have been implicated as consequent to and/or exacerbated by the eating disorder (Lilenfeld et al., 2000). Obsessionality and rigidity are traits that have long been considered potentially predisposing (DuBois, 1949), but are also likely exacerbated by the state of the illness (Pollice et al., 1997).

Very limited research conducted thus far is able to adequately evaluate whether an eating disorder and a personality disorder share a common familial cause. Few family studies examining eating disorders and personality disorders have been conducted, and the few that have been completed did not include a pure personality disorder group (i.e., without an eating disorder). However, both family studies conducted to date have found the same pattern of results implicating a common cause for restricting-type anorexia nervosa and obsessive compulsive personality disorder (Lilenfeld et al., 1998; Strober, 1998). That is, these two disorders may be alternative manifestations of the same underlying etiology (Serpell, Livingstone, Neiderman, & Lask, 2002). The two multivariate twin studies which have been conducted thus far have yielded conflicting findings with regard to whether, and to what extent, personality traits (e.g., neuroticism) and disordered eating share common genes or common environmental factors.

Finally, the notion that personality disorders or traits may influence the course and outcome of an eating disorder (i.e., pathoplasty) has been evaluated. Many studies have examined personality disorders as a general category rather than examining specific personality disorders, limiting conclusions that can be made. Those few studies that have examined individual personality disorders have consistently found that a Cluster B personality disorder, particularly borderline personality disorder, is associated with poorer treatment outcome among eating disorder patients. Obsessive-compulsive personality disorder traits are a poor prognostic feature among anorexic patients (Steinhausen,

2002). By contrast, histrionic personality disorder traits are associated with better treatment outcome in this group. In addition, recent research suggests that greater self-directedness (e.g., having a clear sense of oneself and one's goals) may predict better outcome from eating disorders. Finally, elevated harm avoidance and maturity fears have been linked specifically to poor outcome in anorexia nervosa.

4.2. Future directions

First, more sophisticated, longitudinal, prospective research is needed. Most prospective research in the field of eating disorders, thus far, has been limited by variable predictor and outcome measures, modest sample sizes, short follow-up periods, and failure to control for baseline eating pathology. Prospective research is time-consuming and costly, but necessary to identify true predispositional personality factors. Second, more family study work is needed in order to identify those personality disorders and traits that may share a common familial etiology with eating disorders. Specifically, it is critical to include a pure personality disorder group, along with the pure eating disorder, comorbid eating disorder–personality disorder, and normal control groups. This research design is also costly and labor-intensive; however, this work will aid in directing the focus of burgeoning genetic research in the field (e.g., Kaye et al., 2000). Finally, more epidemiologically based research is needed. Because personality disorders are notoriously over represented in clinical samples (Fairburn, Welch, Norman, O'Connor, & Doll, 1996), generalizability will necessarily be limited if research is restricted to clinical populations.

Personality is just one piece of the eating disorder puzzle, but likely a very important one. The field is making great strides in this area and it is our hope that the current review will serve as a summary of where we are and what still remains to be done in order to further our understanding of the many possible relationships between eating disorders and personality.

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