
Risk Factors for Eating Disorders

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The authors review research on risk factors for eating disorders, restricting their focus to studies in which clear precedence of the hypothesized risk factor over onset of the disorder is established. They illustrate how studies of sociocultural risk factors and biological factors have progressed on parallel tracks and propose that major advances in understanding the etiology of eating disorders require a new generation of studies that integrate these domains. They discuss how more sophisticated and novel conceptualizations of risk and causal processes may inform both nosology and intervention efforts.

Keywords: eating disorders, epidemiology, cultural risk factors, genetic risk factors

Eating disorders rank among the 10 leading causes of disability among young women (Mathers, Vos, Stevenson, & Begg, 2000), and anorexia nervosa has the highest mortality rate of all mental disorders (Millar et al., 2005; Sullivan, 1995; Zipfel, Lowe, Reas, Deter, & Herzog, 2000). Fueled by these ominous indicators of their clinical significance, efforts to identify risk or causal factors for eating disorders are indicated for at least four critical reasons. First, identification of causal mechanisms satisfies the need to understand why certain people develop the problem in question and others do not. Indeed, some experts believe that such an understanding helps decrease the stigma associated with a mental disorder: If the cause is seen as out of the individual's control, less blame is assigned than if the disorder is seen as "volitional" (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000). A *Newsweek* magazine story titled "Fighting Anorexia: No One to Blame" stressed recent findings on genetic vulnerability to explain risk for the development of anorexia nervosa (Tyre, 2005).

Second, ideally, nosology is based on etiology, yet the current classification schema for eating disorders, as articulated in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV]; American Psychiatric Association, 1994), is based solely on the observed clustering of signs and symptoms. The eating disorder criteria remain the subject of considerable debate, in large part because they fail to result in clearly defined subgroups or to account for changing symptomatology over the course of the illness. Most individuals who experience a clinically significant eating disorder do not meet diagnostic criteria for anorexia nervosa or bulimia nervosa but, rather, meet criteria for *eating disorder not otherwise specified* (EDNOS), a diagnosis intended to capture a residual group (Hoek &

van Hoeken, 2003; Hudson, Hiripi, Harrison, & Kessler, 2005; Striegel-Moore et al., 2005). Binge-eating disorder (BED) is the most widely studied specific example of an EDNOS (for review, see M. J. Devlin, Goldfein, & Dobrow, 2003). Also, diagnostic crossover is common, especially from anorexia nervosa to bulimia nervosa (Tozzi et al., 2005) or from bulimia nervosa to BED (Fichter, Quadflieg, & Hedlund, 2005). Risk-factor studies present an untapped source of information of potential value for revising the current classification system.

Third, treatment is best accomplished when we know the causes of a disorder. The current evidence base for treatment of anorexia nervosa, in particular, is weak (Berkman, Bulik, Brownley, & Lohr, in press). A classic example from the history of psychiatry is the treatment of neurosyphilis. As recently as the mid-19th century, advanced neurosyphilis with psychiatric manifestations was a ticket to treatment in an asylum. With the discovery of the *Spirochaeta pallida* (*Treponema pallidum*; Schaudinn & Hoffman, 1905), the search for biological cures ultimately led to the discovery of the efficacy of penicillin in the treatment of this sexually transmitted disease, leading to a rapid decline in incidence and changing treatment forever. Although the story with anorexia nervosa is unlikely to be so dramatic, this transformation of syphilis treatment from asylums to penicillin illustrates the power of understanding etiology in the search for effective interventions.

Fourth, identification of risk factors is important for determining high-risk groups for targeted interventions, designing prevention program content, and informing public policy. For example, a growing literature on cross-generational transmission of eating disorders, termed *cycles of risk* (Bulik, Reba, Siega-Riz, & Reichborn-Kjennerud, 2005), suggests that targeted prevention strategies should be tested that focus on the offspring of women with eating disorders. Through the lens of sociocultural models, many prevention programs feature media literacy as a key component (Stice & Shaw, 2004). Finally, legislative efforts have included the introduction of laws prohibiting false advertising by the diet industry.

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The single best predictor of risk for developing an eating disorder is being female, prompting the question *why women?* And indeed, most studies have been restricted to girls or women; yet, clearly, female sex is not a sufficient condition for explaining risk, prompting a second question: *Which women in particular?* (Striegel-Moore, Silberstein & Rodin, 1986). In the present review, we describe the state of knowledge of risk and causal factors for eating disorders as gleaned from studies on human populations. This illustrates that the “prototypical” eating disorder case is a young, White, middle- or upper-middle class woman residing in Europe or North America, reflecting in part the legacy of defining eating disorders on the basis of White girls or women and the common practice in the field of relying on patient samples (biasing samples toward White, educated, and more affluent individuals). Our understanding of risk factors will remain woefully incomplete until research definitions and sampling strategies accommodate diversity in the population within and across nations. We also address how risk-factor knowledge contributes to destigmatization of eating disorders, suggests revisions for the next *DSM* and *International Classification of Diseases* (World Health Organization, 1992), and informs treatment and prevention interventions. We conclude with recommendations for future studies and future thought about eating disorders.

Changing Perspectives on Risk Factors for Eating Disorders

Eating disorders (compared with many other psychiatric disorders) are newcomers to the official psychiatric nomenclature. Since their earliest appearances in the literature, the field has been bifurcated in the search for etiological ex-

planations. In this section, we review briefly the history of our understanding of anorexia nervosa and bulimia nervosa.

Anorexia Nervosa

Anorexia nervosa was introduced as a new illness in the late 19th century in separate yet almost simultaneous accounts by British psychiatrist William Gull (1874, 1888) and French physician Charles Lasègue (1873). Although both Gull and Lasègue characterized anorexia nervosa as a “nervous” disease, each man emphasized different aspects of his patients’ clinical presentation. Gull’s clinical description elaborated on the physiological correlates that resulted from a “perversion of the will” and attributed them to “simple starvation” without detailed discussion of what might have caused this “perversion.” Lasègue described anorexia nervosa as a “hysteria of the gastric center” and paid considerable attention to the psychological or social factors that he believed were involved in the development of this disorder (cited in Brumberg, 2000, pp. 118–119). At the risk of oversimplification, Gull focused more on biological processes and disruptions, whereas Lasègue focused more on psychosocial and psychological roots. In both cases, however, the treatment was somatic, involving nutritional restoration. Nearly a century later, Hilde Bruch (1973, 1979) introduced a biopsychosocial conceptualization of anorexia nervosa that emphasized developmental factors and family dynamics. Theoretical models proliferated in the 20th century ranging from the purely biological (Kaye, Frank, et al., 2005) to the biopsychosocial (Connan, Campbell, Katzman, Lightman, & Treasure, 2003) to the purely cultural (Bordo, 1993; Orbach, 1986), yet studies of risk factors typically have not tested comprehensive etiologic models that incorporate biological, psychosocial, and environmental factors (for reviews, see Bulik et al., 2005; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Striegel-Moore & Cachelin, 2001).

Bulimia Nervosa

Russell (1979) described bulimia nervosa as “an ominous variant of anorexia nervosa,” and although he widely has been credited with introducing this disorder into the nomenclature, a few years prior, Boskind-Lodahl (1976) offered a feminist formulation of the *binge-purge syndrome* as a culture-bound syndrome that arose from Western culture’s obsession with female thinness in particular and the restrictions of female gender role stereotypes in general. As with anorexia nervosa, numerous theoretical models proliferated, yet most risk-factor studies have tested variables representing various risk domains rather than testing a particular theoretical model (Jacobi et al., 2004; Stice, 2002; Striegel-Moore et al., 1986; for review, see Striegel-Moore & Cachelin, 2001). Nevertheless, too often, discussion of the etiology of eating disorders becomes polarized into “cultural” versus “biological” explanations that ignore the fact that biological and environmental variables are inextricably linked.



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Advances in Conceptualizing Risk

Fundamentally, risk-factor research seeks to understand the factors that cause an outcome of interest (a full discussion of the epistemological and methodological challenges of such research is beyond the scope of this article; see, e.g., Haynes, 1992). A risk factor is a characteristic (e.g., allele), event (e.g., teasing), or experience (e.g., growing up in a culture that values extreme thinness) that precedes the onset of the outcome of interest (e.g., an eating disorder) and that, “if present, is associated with an increase in the probability (risk) of a particular outcome over the base rate of the outcome in the general (unexposed) population” (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997, p. 377). For causality to be inferred, it further needs to be shown that the association between the risk factor and the outcome is not due to confounding influences, that the results are replicable, and that there are plausible explanations for the processes mediating the relation between the hypothesized factor and the outcome (Kazdin et al., 1997). No one study can meet all of these requirements; rather, risk-factor research involves piecing together the puzzle by drawing on multiple studies using a range of designs and methods.

As discussed in previous comprehensive reviews (Jacobi et al., 2004; Stice & Shaw, 2002; Striegel-Moore & Cachelin, 2001), the state of knowledge concerning the risk and causal factors of eating disorders is frustratingly incomplete. Notwithstanding the fact that they are central to advancing etiological models, most epidemiological studies have not included eating disorders among the psychiatric disorders to be assessed in the population (Wittchen & Jacobi, 2005). In the United States, the first epidemiological study of a nationally representative sample was con-

ducted only in 2001–2003 (Hudson, Hiripi, Pope, & Kessler, 2007). Few studies have moved beyond the first (important, yet preliminary) step of demonstrating statistical association between the factor and the outcome to the second step of establishing the factor’s precedence, and fewer studies yet have used an experimental manipulation as the ultimate test of causal hypotheses (Jacobi et al., 2004). For example, anorexia nervosa has been shown to be associated with abnormalities in the serotonergic system during the acute illness stage as well as after recovery (Kaye, Bailer, Frank, Wagner, & Henry, 2005). This research suggests that the serotonin system is a potentially fruitful area of further study, yet before they can be considered to be of etiological significance, it needs to be established whether these abnormalities simply represent concurrent symptoms or consequences (biological scars) of the eating disorder. Similarly, measuring personality characteristics or family functioning in acutely ill or recovered patients does not permit disaggregation of risk factors from clinical correlates or consequences of experiencing the disorder. We caution that in decades past, researchers proposed the now-debunked hypotheses that risk for schizophrenia and autism arose from particular mothering styles (Caplan & Hall-McCorquodale, 1985) on the basis of data gathered with designs ill-suited for testing risk-factor hypotheses. Retrospective assessment of putative risk factors (e.g., asking about an individual’s history of weight-related teasing) is fraught with methodological problems such as difficulty with accurately timing the exposure relative to the age of onset of the disorder and inaccurate or selective recall of critical events. We focus our review on the second generation of research, which comprises genetic studies, studies with prospective and experimental designs, and evidence concerning fixed markers. We do not review the voluminous literature of cross-sectional or retrospective studies of personality characteristics or parenting styles and family functioning, a literature that has been critically discussed in numerous reviews (e.g., Jacobi et al., 2004; Ward, Ramsay, & Treasure, 2000; Wonderlich, Lilienfeld, Riso, Engel, & Mitchell, 2005).

Research Approaches to Uncovering Risk Factors for Eating Disorders

The core features of eating disorders include disturbance in body image (e.g., overvaluation of thinness, weight or shape concerns), over- or undercontrol of eating (e.g., severe dietary restriction, binge eating), and extreme behaviors to control weight or shape (e.g., compulsive exercise, purging). This suggests a research focus on biological structures and processes involved in appetite, satiety, and weight regulation and on cultural factors that shape attitudes and behaviors related to body image and eating. For decades, the primary focus of risk-factor research has been sociocultural and family influences on etiology of eating disorders, yet very few specific, replicated candidate environmental risk factors have emerged (Jacobi et al., 2004). In contrast, studies over the past decade have consistently detected genetic effects. Furthermore, our ability to mea-

sure genes has outpaced our ability to measure environments that might influence eating disorder risk. In this article, we ask what is known about risk factors for the development of anorexia nervosa and bulimia nervosa from research addressing the cultural and biological origins of risk. Most research studies of these two risk domains have progressed on separate tracks, with biological factors being explored mostly in genetic studies and cultural factors explored mostly in longitudinal or experimental studies. We conclude with an illustration of how an integration of research approaches will help overcome the bifurcation of the field and advance understanding of the causes of eating disorders.

Culture and Risk for Eating Disorders

Sociocultural models of eating disorders have emphasized “Western” culture’s female beauty ideal of extreme thinness and objectification of the female body as specific risk factors for the development of an eating disorder. The cultural models describe these steps: exposure to the thin ideal; internalization of the ideal; and experience of a discrepancy between self and ideal, which in turn leads to body dissatisfaction, dietary restraint, and restriction. In some individuals, restraint and/or restriction leads to overeating, in turn amplifying body image concerns and, thus, precipitating further restraint and/or purging (Polivy & Herman, 1985; Striegel-Moore et al., 1986). Objectification of the female body contributes to risk by teaching girls and women that they are valued primarily for their looks, reinforcing the need to pursue attractiveness (Moradi, Dirks, & Matteson, 2005).

To explain why some but not all girls or women develop an eating disorder in this cultural climate, additional variables have been proposed that serve to amplify or mitigate against risk arising from the thin beauty ideal (Striegel-Moore et al., 1986). These include social pressure to be thin (this may take the form of being exposed to media images, peer teasing, or admonishments to lose weight, to name a few), high social class (with, presumably, more attention being paid to and more resources being available for working toward the beauty ideal), personality traits such as perfectionism (making one more eager to comply with the social norms), high social anxiety (increasing one’s susceptibility to social feedback), elevated weight or obesity (moving one’s body further away from the ideal), high impulsivity (making maintenance of restrictive eating more challenging and amplifying risk for binge eating), individual differences in biological response to starvation, and individual differences in the reward value of starvation or eating. These models cannot explain why some individuals go on to develop binge eating whereas others engage only in extreme dietary restriction and other forms of inappropriate weight-control behaviors (e.g., excessive exercise).

Four lines of evidence have been considered as supporting the cultural models: (a) the preponderance of female cases of anorexia nervosa and bulimia nervosa; (b) the rising incidence of anorexia nervosa and bulimia nervosa in

girls or women coinciding with the decreasing body-size ideal for women; (c) cross-cultural differences in the incidence or prevalence of eating disorders, with higher incidence–prevalence in cultures that value extreme female thinness; and (d) the significant prospective relationship between internalization of the thin ideal and disordered eating.

Eating Disorders: Primarily a Female Psychopathology

Worldwide, females with an eating disorder outnumber males by a sizeable margin in every study (Hoek, 2006; Wittchen & Jacobi, 2005). For example, the National Comorbidity Survey Replication (Kessler et al., 2004; Kessler, Chiu, Dernier, & Walters, 2005), which examined eating disorders in a subset of this representative sample of household residents in the United States ages 18 years and older, found lifetime prevalence rates of 0.3% in men and 0.9% in women for anorexia nervosa and 0.5% in men and 1.5% in women for lifetime bulimia nervosa. In a representative sample of Oregon high school students (ages 14–18 years), none of the boys and 0.74% of the girls met criteria (current or past) for anorexia nervosa, and 0.14% of boys and 1.6% of girls met criteria for bulimia nervosa (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993).

Sex differences are far less pronounced in BED or when eating disorders are defined more broadly to include partial syndromes (Hudson et al., 2007; John, Meyer, Rumpf, & Hapke, 2006; Woodside et al., 2001). Indeed, recurrent binge eating has been shown to be as common in men as in women (Hay, 1998; Lewinsohn, Seeley, Moerk, & Striegel-Moore, 2002; Reagan & Hersch, 2005; Woodside et al., 2001). Men who binge eat are significantly less likely than women to report extreme weight-control behaviors such as purging (Hay, 1998), and they often use different compensatory methods with different goals (e.g., reducing body fat and increasing muscularity; Anderson & Bulik, 2004). This may account in part for the lower prevalence of bulimia nervosa in males compared with females and also suggests that the diagnostic criteria themselves may be sex-biased. Hence, the eating disorders for which weight concerns are defining features are considerably more common among females than among males. In contrast, sex appears to be a less potent marker of risk for eating disturbances that are not marked by weight and shape concerns (as currently defined) and extreme compensatory behaviors.

Adolescence: The period of greatest vulnerability. With remarkable consistency, research has shown that anorexia nervosa and bulimia nervosa typically occur during adolescence and that onset thereafter is uncommon (Garfinkel et al., 1995; Hudson et al., 2005; Kendler et al., 1991; Striegel-Moore et al., 2005; Woodside et al., 2001). It is of note that onset of BED does not seem to follow this pattern: Onset has been reported to occur well into adulthood (Hudson et al., 2005, 2007; Kinzl, Traweger, Trefalt, Mangweth, & Biebl, 1999).

Socioeconomic status. Worldwide, studies have shown that in affluent countries, higher status individuals on average are thinner than lower status individuals (Sobal & Stunkard, 1989). On the basis of these observations, anorexia nervosa has long been described as a disease of affluence (Bruch, 1973), and a few studies have found anorexia nervosa to be more common among individuals of high socioeconomic status (Lindberg & Hjern, 2003; McClelland & Crisp, 2001). In contrast, no relationship was found between parental education and bulimia nervosa in the only epidemiological study examining this association (Kendler et al., 1991). Studies that combined anorexia nervosa and bulimia nervosa (Favaro, Ferrara, & Santonastaso, 2003; Lewinsohn, Striegel-Moore, & Seeley, 2000; Moya, Fleitlich-Bilyk, & Goodman, 2005; Wittchen, Nelson, & Lachner, 1998) found no association between socioeconomic status and eating disorders; yet in combining all eating disorders, the possible differences between anorexia nervosa and bulimia nervosa concerning socioeconomic status were obscured.

Males with an eating disorder. The cultural models emphasize the female beauty ideal and gender role socialization in explaining the sex imbalance in the risk for eating disorders; accordingly, homosexual males may be at elevated risk because of gay culture's increased emphasis on physical appearance relative to that of heterosexual male culture (Boroughs & Thompson, 2002; Silberstein, Mishkind, Striegel-Moore, Timko, & Rodin, 1989). We were able to locate only one risk-factor study that addressed this issue (omitting several studies with inadequate comparison groups). Specifically, a survey of 4,374 adolescent boys (mean age = 14.3 years) found that boys who described themselves as gay or bisexual reported making more efforts to look like boys or men in magazines than did boys who described themselves as heterosexual. There were no differences in the frequency of dieting efforts reported across sexual orientation, but the gay-bisexual boys were more likely to binge eat than the heterosexual boys (Austin et al., 2004). Given that trying to look like boys or men in magazines does not necessarily represent a causal continuity with eating disorders, and given the young age of this sample, little can be concluded regarding the nature of homosexuality as a risk factor for the development of eating disorders in males.

Race/ethnicity. It long has been assumed that eating disorders are rare among ethnic minority populations, in part because in clinical practice or in treatment trials, few ethnic minority individuals have been found (Smolak & Striegel-Moore, 2001). Regardless of types of psychiatric disorders, patient samples are biased toward an overrepresentation of White women (Kessler et al., 2005). Studies in the United States have found that ethnic minority women compared with White women are disproportionately less likely not only to seek care but, when they do seek care, to receive treatment for the eating disorder (Becker, Franko, Speck, & Herzog, 2003; Cachelin & Striegel-Moore, 2006; Striegel-Moore et al., 2003; Wilfley, Pike, Dohm, Striegel-Moore, & Fairburn, 2001).

We were able to locate only one study in the United States that examined race/ethnicity as a marker of eating disorders in a community sample that used a rigorous diagnostic screening and assessment procedure. In a cohort of 2,054 young adult African American (Black) and White American women (mean age = 21 years), all eating disorders were less common among the Black women, and there were no Black women with anorexia nervosa (compared with 1.5% of the White women; Striegel-Moore et al., 2003). However, the two groups differed in parental socioeconomic status, making it difficult to determine whether the results reflected the fact that few of the Black girls came from affluent households and accounted for the differences in prevalence of anorexia nervosa. In the same sample, an empirically based classification of eating disorders found that the clustering of eating disorder symptoms varied by ethnicity: Purging behaviors in the absence of binge eating were significantly more common among the White women, whereas binge eating in the absence of purging was more common among the Black women (Striegel-Moore et al., 2005). Other studies have found no racial/ethnic differences in the prevalence of recurrent binge eating (Reagan & Hersch, 2005; Smith, Marcus, Lewis, Fitzgibbon, & Schreiner, 1998; Striegel-Moore, Wilfley, Pike, Dohm, & Fairburn, 2000). The lack of epidemiological data capturing the population diversity in the United States is a significant gap in our knowledge about eating disorders. On the basis of the preliminary evidence, White race/ethnicity may be a marker for eating disorders involving severely restrictive or compensatory behaviors but not for binge eating.

In conclusion, female sex is a potent and well-replicated fixed marker for anorexia nervosa and bulimia nervosa (Jacobi et al., 2004), but it appears to be less strongly a predictor of BED. Adolescence marks the period of greatest risk for onset. Few studies offer clues about the importance of other demographic characteristics, yet there are hints that variables such as socioeconomic status or race/ethnicity may differentially signify risk for anorexia nervosa, bulimia nervosa, and BED. If confirmed, this would support a nosological distinction of syndromes involving severe dietary restriction or purging from syndromes involving binge eating without compensatory behaviors.

Secular Trends in the Incidence of Eating Disorders

Ideal female body size significantly decreased during the 20th century, with marked decreases in media images having been documented for the period between 1958 and 1988 (e.g., Wiseman, Gray, Mosimann, & Ahrens, 1992). To examine whether there has been a corresponding increase in eating disorder incidence (absent epidemiological data), experts have examined changes in the number of new patients being treated. Hoek and van Hoeken (2003) found that the incidence of anorexia nervosa increased markedly from 1930 through 1970 and has remained unchanged since then. Similarly, the prevalence of anorexia nervosa in Swe-

den, studied in 31,406 twins, was significantly higher in individuals born between 1945 and 1958 than in individuals born between 1935 and 1944 (Bulik et al., 2006). Likely, this increased incidence is not simply the result of greater public awareness or availability of specialized treatment resources for anorexia nervosa in recent years: Anorexia nervosa did not receive much public attention, nor were there many eating disorder treatment facilities or experts, until the latter quarter of the 20th century (Brumberg, 1988).

Bulimia nervosa was not officially recognized until 1979, precluding an exploration of changes in incidence across the period of time when the female body ideal drastically shrank in size (1950–1980). Epidemiological studies suggest a secular increase in the incidence of bulimia nervosa during the latter half of the past century: Lifetime prevalence was significantly lower among age cohorts born before 1944 (Hudson et al., 2007), 1950 (Bushnell, Wells, Hornblow, Oakley-Browne, & Joyce, 1990), or 1960 (Kendler et al., 1991) than among more recent age cohorts. Moreover, studies identifying new patients receiving medical care in the Netherlands (Hoek et al., 1995), the United Kingdom (Treasure et al., 1996), and the United States (Soundy, Lucas, Suman, & Melton, 1995) reported significant increases in the incidence of bulimia nervosa between 1985 and 1995.

The evidence of increased incidence of eating disorders points to the importance of cultural factors in their etiology, but it does not preclude the important role of biological factors. A parallel example in a related field is the rapid escalation of the prevalence of obesity. Although the genetic underpinnings for obesity are undisputable (Maes, Neale, & Eaves, 1997; Perusse et al., 2005), the rapid increase in prevalence can only be attributable to environmental shifts, because the gene pool cannot change that quickly. Intriguingly, this argues for careful consideration of other mechanisms of gene–environment interplay rather than genetic main effects—such as genetic sensitivity to changes in the environment—which, as yet, remain unstudied in eating disorders.

Cross-Cultural Studies

Some have described eating disorders as “culture-bound syndromes,” either in the sense that they occur uniquely in a limited number of cultures (Prince, 1985) or in the sense that they represent the culturally shaped (i.e., culture-specific) expression of an underlying (culturally universal) disease (Swartz, 1985). Within this framework, anorexia nervosa and bulimia nervosa, as defined in the *DSM-IV*, should not be found in cultures that do not subscribe to the social norms about female thinness and the importance that women pursue beauty to affirm their sense of femininity and secure interpersonal or vocational success (Rodin, Silberstein, & Striegel-Moore, 1985).

We have already noted the dearth of epidemiological studies; consequently, the data needed to answer the question of whether risk for eating disorders varies across cultures are incomplete at best. Eating disorders have been

shown to occur across the globe (Becker & Fay, 2006; Keel & Klump, 2003; Makino, Tsuboi, & Dennerstein, 2004), yet few studies have used designs suitable for testing the hypothesis that eating disorders are less common in *non-thin-ideal* cultures than in *thin-ideal* cultures. We caution that with one notable exception (Hoek et al., 2005; Hoek, van Harten, van Hoeken, & Susser, 1998), these studies have relied on samples of convenience, used diagnostic assessments that were not validated for use in the population under investigation, and failed to take socioeconomic status into account. Given these limitations, it is not surprising that prevalence estimates vary considerably across studies (Keel & Klump, 2003; Makino et al., 2004).

Consistent with the cultural models, Hoek et al. (1998, 2005) found that the overall incidence of anorexia nervosa was much lower on Curaçao, an island on which the majority population is Black and values large female bodies, than in the Netherlands or the United States, but the incidence among White or mixed-race women was on par with that in the comparison countries. Specifically, all of the Curaçao cases were of mixed race (and, as such, were navigating two cultures) or were White. The authors noted that all mixed-race cases had been abroad, were more affluent than the population average, and described (in in-depth qualitative interviews) how they were aspiring to lead a “Western” lifestyle (Katzman, Hermans, van Hoeken, & Hoek, 2004).

Experts have emphasized that globalization has brought the thin female beauty ideal and related social norms to ever larger numbers of cultures and peoples and that, as a consequence, eating disorders ought to be expected to increase worldwide (Catina & Joja, 2001; Gordon, 2001). When, after 51 years of unsuccessful Nigerian participation in the Miss World beauty pageant, a Nigerian contestant finally won the title in 2001, her success was attributed to the fact that Nigeria for the first time had sent a contestant who was not considered beautiful by local standards because she was too thin (Onishi, 2002). Whether this introduction of the thin ideal in a country that has long cherished fat women will result in an increase in eating disorders is yet to be seen; the findings from Curaçao suggest that this may happen. Research has lagged behind in capturing the impact of the increasing penetration of the thin beauty ideal into cultures that traditionally have valued ample female bodies. Although ethnographic accounts offer intriguing examples of adverse effects of exposure to the thin beauty ideal on body-image concerns (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002), the few studies that have explored the impact of cultural transition of subpopulations (e.g., comparing individuals who moved from a nonthin-ideal culture to a thin-ideal culture with those who remained behind) or of entire populations through acculturation on body image and eating behavior are methodologically limited in much the same ways as the aforementioned cross-cultural studies and have produced conflicting results (for review, see Becker & Fay, 2006).

In conclusion, the literature on cross-cultural differences has accumulated clear evidence that eating disorders

do occur across the globe, yet it has been less successful in providing unambiguous evidence for the role of culture in risk, because of methodological limitations. Accelerating globalization offers the perfect opportunity to study the impact of changing body ideals on eating disorders risk as well as the potential to elucidate mechanisms whereby such an effect would occur.

Internalization of the Thin Beauty Ideal Increases Risk for Eating Disorder Symptoms

The cultural theories of eating disorders have excellent face validity, yet a critical question is by what mechanism(s) the cultural factor of thin ideal contributes to eating disorder risk. Recognizing the near ubiquitous exposure to images of and social norms regarding the importance of attaining the thin ideal in Western cultures, Rodin et al. (1985) proposed internalization of the thin ideal as one such mechanism. Thin-ideal internalization encompasses an awareness of the ideal and its social meaning and adoption both of the ideal and belief in its personal relevance (Cafri, Yamamiya, Brannick, & Thompson, 2005; Striegel-Moore et al., 2004).

Longitudinal studies and experiments have been used to test the relation between thin-ideal internalization and eating disorders. In both designs, various outcome measures have been used to accommodate the low incidence of full-syndrome eating disorders. Indeed, we found no study with *any* new-onset cases of anorexia nervosa in girls, and the literature documents only one incident case of bulimia nervosa in the context of a longitudinal risk-factor study (McKnight Investigators, 2003). Consequently, thus far the literature is silent concerning the contribution of thin-ideal internalization, body dissatisfaction, or dieting to risk for the development of full-syndrome anorexia nervosa or bulimia nervosa.

Some investigators have coped with the challenge of low incidences by broadening the case definition by combining full- and partial-syndrome cases, either by disorder (Kotler, Cohen, Davies, Pine, & Walsh, 2001) or across anorexia nervosa and bulimia nervosa (Ghaderi & Scott, 2001; Martinez-Gonzalez et al., 2003; McKnight Investigators, 2003; Patton, Selzer, Coffey, Carlin, & Wolfe, 1999). Although there is some evidence of etiological continuity between partial and full syndromes within disorders (Garfinkel et al., 1996; Kendler et al., 1991; Walters & Kendler, 1995), as we illustrated earlier in the case of fixed markers, etiological continuity should not be assumed across the eating disorders. Another approach involves using a continuous outcome measure that is derived by adding various affective, cognitive, and behavioral symptoms of all eating disorders in a composite score (Stice & Shaw, 2002). A meta-analysis of prospective and experimental risk-factor studies found that effects were weaker when outcome was defined using continuous composite-symptom scores than when outcome was defined categorically (Stice & Shaw, 2002).

A third, more granular approach disassembles diagnostic categories into component symptoms (which invari-

ably are more common than full syndromes) and identifies risk factors associated with one or more particular component parts of a syndrome (e.g., binge eating; Field, Camargo, Taylor, Berkeley, Frazier, et al., 1999; Field et al., 2002; Johnson & Wardle, 2005; Killen et al., 1994; Stice, Killen, Hayward, & Taylor, 1998; Stice, Presnell, & Spangler, 2002). This approach is of interest not only for the pragmatic reason of increased sample size but because it is perhaps fallacious to assume that the risk factors for all of the symptoms of our a priori defined syndromes are necessarily the same. By the same token, however, it cannot be assumed that the risk profile for a symptom necessarily reflects the risk profile for the entire syndrome. Results therefore need to be interpreted within the context of the design.

Longitudinal studies, consistently, have shown that thin-ideal internalization, body dissatisfaction, elevated body weight, and dieting predict risk for onset or worsening of eating pathology and that the effects of these risk factors are considerable (McKnight Investigators, 2003; Stice, 2002). For example, in a large cohort of 14–15-year-old Australian children, girls reporting severe dieting were 18 times more likely to develop partial-syndrome bulimia nervosa at a 6-month follow-up than were girls who did not diet (Patton et al., 1999). A study of 1,177 adolescent girls found that body dissatisfaction and dieting predicted increased bulimic symptoms at a 12-month follow-up (Johnson & Wardle, 2005). The Growing Up Today Study, which followed over 11,000 boys and girls (ages 9–14 years at study entry), reported that a composite measure of thin-ideal internalization, social pressures or teasing about weight, and dieting was predictive of onset of purging in girls at the one-year follow-up (Field, Camargo, Taylor, Berkey, & Colditz, 1999) and of onset of binge eating from the one-year to the two-year follow-up (Field et al., 2002).

A meta-analysis (Groesz, Levine, & Murnen, 2002) of 25 experimental studies of the effect of exposure to media images of thin women on female participants (most of them college students) found that body dissatisfaction significantly increased after exposure (in within-group designs) or relative to women who were exposed to control images (in between-groups designs). Overall, effect sizes were modest, and effects were stronger in studies of high school girls compared with college women and stronger among participants who reported great body dissatisfaction prior to the experimental exposure than those who were satisfied with their body image. Experimental studies also showed that female students reported significant increases in body dissatisfaction when they were exposed to peer modeling of thin-ideal internalization or social pressure about being thin (Krones, Stice, Batres, & Orjada, 2005; Stice, Maxfield, & Wells, 2003). Such studies have typically involved no follow-up to determine whether the effects on body image were transient or lasting. Only one experimental study has examined the longer term effects of exposure to media images (receiving a year-long subscription to a fashion magazine), and it found no long-term effects on body dissatisfaction, dieting, or bulimic behavior (Stice, Span-

gler, & Agras, 2001). Stice, Trost, and Chase (2003) demonstrated, however, that an intervention designed to reduce thin-ideal internalization produced significant decreases in thin-ideal internalization and significant improvements in negative affect and bulimic symptoms. This work is promising in that it suggests that the adverse effects of exposure to the thin ideal may be countered with a psychological intervention.

Taken together, the experimental evidence suggests that media exposure to the thin ideal, or social pressure about thin-ideal internalization, increases body-image concerns at least in the short run. Experimental studies offer a more rigorous test of etiological hypotheses than longitudinal studies and permit a more clear separation of the elements in the hypothesized causal sequence from exposure to the thin ideal to thin-ideal internalization and so on. Indeed, longitudinal studies that combine elements of this sequence may yield inflated estimates of the role of the thin ideal as a risk factor, because composite predictors confound predictor and outcome variables (e.g., body dissatisfaction and dieting are both links in the risk chain and symptoms of an eating disorder). Exposure to the thin ideal and social pressures to internalize it are nearly ubiquitous, as exemplified by the extraordinarily high number of hours spent with electronic media (Escobar-Chaves et al., 2005), making it difficult to demonstrate the effect of such exposure on eating disorder risk in short-term studies. Future experimental studies need to test whether repeated exposure to interventions that counter the cultural messages of the importance of thinness are protective against the onset of eating disorder symptoms and to examine the impact of such interventions over longer time periods.

The advantages of experimental designs notwithstanding, we see a need also for future longitudinal studies. Our review echoes the conclusions reached by others that the complex etiology of eating disorders calls for multifactorial risk models that accommodate risk factors from multiple domains. Experimental designs can only test a small number of hypothesized risk factors at a given time. In contrast, longitudinal studies permit exploration of multiple factors. Stice et al. (2002), using signal detection analysis (a methodology that permits identification of subgroups with distinct risk profiles), found that a subset of girls developed binge eating in the absence of significant body-image concerns; these girls' path to binge eating involved significant elevation of depressive symptoms and low self-esteem. The role of culture in increasing risk for the development of low self-esteem has not yet been examined empirically as it relates to eating disorders. Conversely, another subgroup was characterized by high thin-ideal internalization and elevated body mass index (BMI). This work supports a more complex understanding of risk that describes multiple risk pathways toward eating pathology and helps explain why a given risk factor, when tested at the population level, may only be linked weakly or inconsistently with risk yet potent when experienced in combination with other risk factors.

The Role of Genetic Factors

After reviewing the plethora of research on sociocultural factors and their influence on the development of eating disorders, we are still left with one pressing unanswered question. Namely, if all young girls are exposed to these sociocultural pressures, why does only a small fraction go on to develop anorexia nervosa and bulimia nervosa? The methods we have described thus far are capable of identifying patterns and trajectories, but they have not yet evolved sufficiently methodologically to identify actual mechanisms of risk. In contrast, genetic research is well positioned to identify and explicate mechanisms. As we review the state of knowledge, we are faced with three possible models.

First, understanding the genetic underpinnings of anorexia nervosa and bulimia nervosa may answer this vexing question in much the same way it was stated—by helping us identify why some individuals, by virtue of their genetic legacy, are more vulnerable to sociocultural pressures to attain a thin ideal. This would be a Gene \times Environment interaction model whereby varying genotypes would render individuals differentially sensitive to environmental events. For example, an individual with Genotype A might experiment with her first extreme diet, find the experience aversive and uncomfortable, and reject the behavior on the basis of it not being at all reinforcing. In contrast, an individual with Genotype B might experience that first episode of severe caloric restriction to be highly reinforcing by reducing her innate dysphoria and anxiety, providing her with a sense of control over her own body weight and resulting in her receiving positive social attention for weight-loss attempts. That individual might then adopt the behavior in a persistent manner because of its multiple reinforcing effects, which would then set the stage for the development of anorexia nervosa.

A second model would be that we could, theoretically, discover genetic main effects. We could find that the sociocultural theories of eating disorders represent mere illusory correlations—convenient and highly face valid yet temporally and culturally limited explanations for a perplexing phenomenon, the plausibility of which obscures the contribution of much stronger genetically driven processes of appetite and weight dysregulation.

Third, we might uncover what could best be described as a *mixed model*. This hypothetical model would reflect an artifact of our diagnostic system that may obscure our ability to detect genes by clustering together a grab bag of symptoms of varying degrees genetic and environmental etiology to create the syndromes we know today as anorexia nervosa and bulimia nervosa. Genetic main effects could primarily account for the core symptoms we see in eating disorders that have persisted throughout history—such as maintenance of low body weight, binge eating, and self-induced vomiting—but the pervasive culture of the times may have “filled in the gaps” by providing an explanatory context and padding the definition of syndromes with environmentally mediated and contextually plausible

symptoms. For example, the maintenance of low body weight seen in anorexia nervosa has been observed for centuries, and indeed, the core phenotype of persistent low body weight appears to have genetic underpinnings (Grice et al., 2002), yet the psychological “fillers” that have been added to create the syndrome of anorexia nervosa have changed over time. Historically, in European cultures, the core symptom of low weight was cradled in an explanatory system of saintliness, purity, and asceticism (Bell, 1985). In current European culture, the core symptom is couched with more temporally and contextually relevant criteria which reflect thin-ideal internalization. The core symptom has persisted over the centuries, but the psychological and contextual factors in which we embed the symptom have changed. Also supporting this model is the presentation of anorexia nervosa in many Asian cultures in which the core symptoms of low weight and food refusal exist in the absence of fear of fatness (S. Lee, 1995; Ngai, Lee, & Lee, 2000). This mixed model suggests that our diagnostic categories may be heterogeneous mixtures of genetically influenced symptoms and cultural overlays that may not accurately reflect underlying biology. This third model could have major implications for methodological approaches to genetic research and contribute to furthering our refinement of psychiatric nosology of the eating disorders.

First Steps: Family Studies of Eating Disorders

Despite a late start relative to study of other psychiatric disorders, considerable advances have been made in elucidating the role of genetic factors both independently and in concert with environment in the etiology of eating disorders. Extensive family history studies have focused on the basic question of whether eating disorders aggregate in families. With familial transmission of anorexia nervosa, bulimia nervosa, and BED established (Fowler & Bulik, 1997; Hudson et al., in press; Hudson, Pope, Jonas, & Yurgelun-Todd, 1983; Hudson, Pope, Jonas, Yurgelun-Todd, & Frankenburg, 1987; Y. Lee et al., 1999; Lilienfeld et al., 1998; Strober, Freeman, Lampert, Diamond, & Kaye, 2000, 2001), more recent studies have focused on the extent to which genes play a role, which genes most likely are involved in conferring vulnerability, and their mechanism of influence. Finally, new designs are emerging that couple genetic and environmental risk factors to unveil risk profiles.

Twin Studies of Eating Disorders

Methodological considerations. Twin and adoption studies are the most effective ways to determine the patterns of influence of genetic and environmental factors on disease liability. In the absence of adoption studies, the field of eating disorders has relied heavily on twin studies to elucidate these factors. The fundamental principles of twin methodology use the similarities and differences between monozygotic (MZ) and dizygotic (DZ)

twin pairs to identify and delineate genetic and environmental causes for a particular trait. Twin analyses are based on the liability-threshold model (Falconer, 1965; Pearson, 1901), which assumes that an unobserved or latent variable that indexes liability to a trait is fundamental to the observed trait. Moreover, the twin model is based on the equal environments assumption (EEA)—namely, that MZ twins are not treated more similarly than DZ twins on factors of etiological relevance to the trait of interest. For example, although MZ twins might share the same bedroom more frequently than do DZ twins, sharing a bedroom is unlikely to be a factor of major etiological relevance to eating disorders. To date, no notable violations of the EEA have been observed, lending further credence to the observed results (Bulik, Sullivan, Wade, & Kendler, 2000; Kendler, Neale, Kessler, Heath, & Eaves, 1993; Klump, Holly, Iacono, McGue, & Willson, 2000).

Because twin study methodology can be complex, a brief primer is in order: The variance in liability to any trait can be partitioned into additive genetic, shared environmental, and unique environmental factors. *Additive genetic effects* refer to the cumulative effects of many genes of small-to-moderate effect (i.e., heritability). *Shared environmental factors* contribute to twin similarity and reflect environmental factors influencing both members of a twin pair. *Unique environmental factors*, which include error of measurement, refer to those environmental events to which only one member of a twin pair is exposed. Although rarely acknowledged, it is critical to note that any heritability estimate is a product of the prevalence of the trait under question—MZ twin concordance and DZ twin concordance—and is specific to a given population at a particular point in time. Critical to understanding twin studies is the notion that there is not *one* definitive heritability of any trait or disorder. A classic example of the effect of prevalence on heritability estimates of a trait was reported by Kendler, Thornton, and Pedersen (2000). In this study, regular tobacco use was studied in male and female twins born between 1890 and 1958 in Sweden. In men, over time, both the prevalence and heritability (~61%) of regular tobacco use remained constant across birth cohorts. For women, tobacco use was rare in early cohorts because of social restrictions. Thus, in women born before 1925, rates of tobacco use were low, and twin resemblance was environmental in origin. In later cohorts, as social restrictions were lifted, the prevalence of regular tobacco use increased in women, and the heritability estimates mirrored those of males (~63%). Functionally, social restrictions inhibited the expression of underlying genetic predispositions to regular tobacco use in women.

Twin and genetic studies of eating disorders have approached the task of quantifying genetic and environmental contributions to liability using categorical definitions of illness (e.g., diagnostic categories like anorexia nervosa, bulimia nervosa, and BED), subphenotypes (e.g., binge eating, purging, undue influence of self-evaluation on shape and weight), or purported endophenotypes (e.g., temperament or personality factors believed to be relevant to

the etiology of the disorders). One major shortcoming of family and twin studies of eating disorders is that they have virtually all been conducted on primarily European populations. We know little about the heritability of these disorders and traits in other races and ethnic groups.

Twin studies of anorexia nervosa. In studies that have used multiple strategies to boost statistical power to overcome the problem that anorexia nervosa is an uncommon disorder, the heritability estimates from anorexia nervosa have been 48% (Kortegaard, Hoerder, Jørgensen, Gillberg, & Kyvik, 2001), 58% (Wade, Bulik, Neale, & Kendler, 2000), and 76% (Klump, Miller, Keel, McGue, & Iacono, 2001). The contribution of the shared environment was found to be negligible, and the remaining variance was primarily attributable to unique environmental factors. Only recently has a sufficiently powerful study reported heritability estimates for the narrow *DSM-IV* definition of anorexia nervosa. In 31,406 Swedish twins born between 1935 and 1956, Bulik et al. (2006) identified all cases of anorexia nervosa by clinical interview of the Swedish Twin Registry, hospital discharge diagnosis of anorexia nervosa, or cause-of-death certificate. They reported a heritability estimate of 56% (95% confidence interval [CI]: .00–.87), with a small contribution (5%) of shared environment (95% CI: .00–.64) and 38% of variance in liability attributable to unique environment (95% CI: .13–.84). Despite varying definitions and wide confidence intervals, all of these studies point to a substantial contribution of additive genetic effects to anorexia nervosa.

Twin studies of bulimia nervosa. Twin studies of varyingly broad definitions of bulimia nervosa using univariate, bivariate, and measurement model techniques (Bulik, Sullivan, & Kendler, 1998; Kendler et al., 1991; Kortegaard et al., 2001; Walters et al., 1992, 1993) also have consistently yielded high heritability estimates, ranging from 50% (Walters et al., 1992) to 83% (Bulik et al., 1998), with unique environmental factors accounting for the remaining variance. In line with the third model presented above (the mixture model), bulimia nervosa has also been dissected into its component symptoms, revealing substantial genetic effects on the symptoms of self-induced vomiting (73%) and binge eating (49%–51%; Reichborn-Kjennerud et al., 2003; Sullivan, Bulik, & Kendler, 1998) but suggesting no genetic contributions to the symptom of placing undue emphasis on shape and weight where shared and unique environment were the sole contributors to variance in liability in the best fitting models (Reichborn-Kjennerud et al., 2004).

Twin studies of BED. Twin studies have reported substantial heritability of the symptom of binge eating (Bulik et al., 1998; Bulik, Sullivan, & Kendler, 2003) with the presence of purging behaviors not controlled for statistically. One population-based study of Norwegian twins between 18 and 31 years of age reported a heritability estimate of 41% for a syndrome that approximated BED, termed binge eating in the absence of compensatory behaviors, with the remaining variance attributable to nonshared environment.

Molecular Genetic Studies

Methodological considerations. Two types of molecular genetic designs have been applied to eating disorders: association studies and linkage studies. Association studies compare cases displaying a trait of interest with controls who do not display the trait. All individuals are genotyped for the relevant candidate gene or genes. Statistical analysis is used to compare allele or genotype frequencies (Sasieni, 1997) in cases versus controls (Sham, 1998). Association studies are optimal when the pathophysiology of a trait points to specific candidate genes. The second approach, linkage analysis, requires a large sample of multiplex pedigrees or extreme sibling pairs (Allison, Heo, Schork, Wong, & Elston, 1998). Anonymous genetic markers across the genome are used to identify chromosomal regions that may contain genes that influence the trait of interest. Linkage approaches narrow the focus on the genome. Genes located under the linkage peaks are then further explored using association approaches to determine whether they may indeed be associated with the trait of interest.

Genetic association studies can be dizzying to the reader unfamiliar with genetic research. The parade of initially exciting and later nonreplicated findings has been dubbed the *Proteus effect* (Ioannidis & Trikalinos, 2005) and underscores the methodological and statistical challenges of finding a needle in a haystack while dealing with issues of multiple comparisons and changing prior probabilities. To focus the research, those involved in association studies of eating disorders have homed in on systems known to influence feeding, appetite, and mood as well as genes that have been identified as potentially contributory to other psychiatric disorders.

Molecular genetic studies of anorexia nervosa. The palette of association studies conducted to date is rich and far too detailed to review here. Several comprehensive reviews and updates of association studies in eating disorders have been published (Gorwood, Kipman, & Foulon, 2003; Hinney, Remschmidt, & Hebebrand, 2000; Klump & Gobrogge, 2005; Mazzeo, Slof-Op 't Landt, van Furth, & Bulik, 2006; Slof-Op 't Landt et al., 2005). Briefly, given the known involvement in both mood and appetite, genes in the serotonergic system (e.g., serotonin receptor 2A, 2C, the serotonin transporter gene) and the dopaminergic system (e.g., D3 receptor, D4 receptor, dopamine transporter) as well as several other genes involved in functions believed to be central to the etiology of eating disorders (e.g., brain-derived neurotrophic factor, catechol-O-methyltransferase, estrogen receptor) have been explored, with associations observed and frequently nonreplicated and interpretation clouded by the impact of small sample sizes.

Linkage studies for anorexia nervosa (Bacanu et al., 2005; B. Devlin et al., 2002; Grice et al., 2002; Kaye et al., 2000) have yielded significant results and underscored the importance of detailed phenotyping. A linkage study of a heterogeneous sample of individuals with broadly defined

eating disorders yielded absolutely no signals of interest across the genome. However, restricting that sample to relative pairs exhibiting the classic restricting subtype anorexia nervosa in the absence of binge-eating behavior yielded evidence for the presence of a susceptibility locus on chromosome 1 (Grice et al., 2002). Additional approaches that enhanced the focus of the linkage analysis by incorporating key behavioral covariates into linkage analyses (B. Devlin et al., 2002)—namely, drive for thinness and obsessionality—isolated several regions of interest on chromosomes 1, 2, and 13. The second step in this analysis involves exploring the identified regions of interest to determine whether genes exist in those regions that could plausibly be related to the disorder and testing them for association. Two genes under these linkage peaks, serotonin 1D (HTR1D) and delta opioid (OPRD1) receptor, exhibited significant association with anorexia nervosa (Bergen et al., 2003). The subsequent step is to determine the actual functions of identified genes and how they might contribute to risk for anorexia nervosa.

Molecular genetic studies of bulimia nervosa. In association studies, the choices of candidate genes in bulimia nervosa have closely paralleled those for anorexia nervosa (Gorwood et al., 2003; Hinney et al., 2000; Klump & Gobrogge, 2005; Mazzeo et al., 2006; Slob-O'p 't Landt et al., 2005). One intriguing advance that again shows the importance of detailed phenotyping has emerged from the laboratories of Steiger et al. (2005). This group examined factors associated with the serotonin transporter (5-HTTLPR) gene in 59 women with eating disorders involving binge eating and purging (e.g., bulimia nervosa; anorexia nervosa, binge-purging subtype). Detailed personality information was also available on these women. In this sample, the S allele was not associated with eating disorder symptoms but was associated with borderline personality disorder and related symptoms such as impulsivity, affective instability, and insecure attachment. Individuals with the S allele also had a significantly lower density of paroxetine-binding sites, suggesting that they might not respond as well to selective serotonin reuptake inhibitors—which are commonly prescribed to treat bulimia nervosa. The authors also speculated that the paroxetine-binding densities could reflect Gene \times Environment interactions, because chronic food restriction in animals is associated with 5-HT dysregulation. The authors suggested that chronic dieting typical of patients with these syndromes could trigger the expression of this genetic polymorphism. This emerging body of research presages the potential impact of pharmacogenetics in the treatment of eating disorders and highlights another manner in which genes and environment may interact to influence ultimate phenotypic expression.

Only one linkage study of bulimia nervosa has been reported (Bulik, Devlin, et al., 2003). Significant linkage was achieved on chromosome 10p13, and suggestive evidence for linkage was reported for 10p14 and 14q22-23. Again, working toward the most refined phenotype for linkage analysis and focusing on information from twin

studies that highlighted the high heritability of self-induced vomiting (Sullivan, Bulik, & Kendler, 1998), the authors performed a second linkage analysis in a subset of 133 families, in which at least two affected individuals reported regular vomiting. This refinement led to the narrowing of the linkage peak on chromosome 10p13, which enabled a more focused search for loci of interest.

Molecular genetic studies of BED. Association studies have also been initiated to shed light on genetic mechanisms underlying binge eating, with one study reporting BED to be associated with mutations in the melanocortin 4 receptor gene (Branson et al., 2003), a gene implicated in the development of overeating and obesity (Farooqi et al., 2003; List & Habener, 2003); however, a subsequent study failed to replicate this finding (Hebebrand et al., 2004).

Genetic Epidemiology and Risk: Questions for the Future

It is intriguing to reevaluate the existing genetic literature on the basis of the three models forwarded. Most notably, Reichborn-Kjennerud et al. (2004) suggested that there may be distinct sources of familial resemblance (e.g., genetic and environmental contributions) for different symptoms of bulimia nervosa as codified in the *DSM-IV*. Both binge eating and vomiting, which comprise Criteria A and B of *DSM-IV* bulimia nervosa, appear to represent more genetically mediated symptoms, with the heritability of binge eating reported to be between 49% and 51% (Reichborn-Kjennerud et al., 2003; Sullivan, Bulik, & Kendler, 1998) and that of vomiting at 72%. In contrast, Criterion C of *DSM-IV* bulimia nervosa, undue influence of shape and weight concerns on self-evaluation, does not appear to be heritable and shows substantially greater shared environmental effects. Most notably, weight concerns (Wade, Martin, & Tiggemann, 1998) and placing undue importance on weight as an indicator of self-evaluation are best accounted for by models that include only shared and unique environmental effects. Moreover, sex differences may also exist in genetic and environmental contributions to individual symptoms, as illustrated by Keski-Rahkonen et al. (2005), who showed differences in patterns of heritability between males and females on drive for thinness and body-dissatisfaction scores. Their results suggest that the heritability patterns of eating disorder-related attitudes are highly sex specific. Thus, a *DSM-IV* diagnostic category such as bulimia nervosa might actually represent an occasionally co-occurring yet etiologically diverse mixture of genetically and environmentally influenced symptoms that differ by sex. From a genetic perspective, focusing on compound syndromes that include multiple symptoms of varying genetic etiology could impede searches for susceptibility loci. Focused attention on heritable endo- and subphenotypes could assist with isolating genetic factors that influence the core behaviors, and such behaviors could then be reweaved into more complex designs that elucidate gene-environment interaction and correlation.

Neonatal and Neurodevelopmental Variables and Anorexia Nervosa

Nonetheless, the future of genetic research in eating disorders may well rest with sophisticated approaches to Gene \times Environment interplay. One theory has been forwarded as an example of such interplay (Bulik et al., 2005), and we present one prototype methodological example from the affective disorders literature to illustrate one way in which genes and environment may interact.

The theory involves the role of neonatal complications and neurodevelopmental challenges in anorexia nervosa and how genetically mediated maternal variables can influence intrauterine environment. The basis of this theory is the observation that neonatal complications increase risk for anorexia nervosa. Cnattingius, Hultman, Dahl, and Sparen (1999) linked the Swedish birth registry with the Swedish psychiatric inpatient registry and found that girls born prematurely (especially if they were small for gestational age [SGA]) and those born with cephalhematoma were at increased risk for developing anorexia nervosa later in life. Similar results were reported in a different study by Favaro, Tenconi, and Santonastaso (2006) in 114 individuals with anorexia nervosa, 73 individuals with bulimia nervosa, and 554 controls in Padua, Italy. Favaro et al. (2006) reported that maternal anemia, diabetes mellitus, preeclampsia, placental infarction, neonatal cardiac problems, and hyporeactivity were all significant independent predictors of the later development of anorexia nervosa. In addition, the more neonatal events, the higher the risk of developing anorexia nervosa.

In both studies, the authors suggested that subtle brain damage at birth could result in early feeding difficulties and increased risk for anorexia nervosa. However, alternative mechanisms, related to genetically mediated maternal eating behavior, could also be at play. For example, Bloomfield et al. (2003) demonstrated that moderate maternal food restriction in sheep around the time of conception results in precocious fetal cortisol surge and preterm birth. Human females with prolonged food-deprivation episodes are also more likely to deliver at a shorter gestational age, which is probably mediated through higher corticotrophin-releasing hormone levels (Herrmann, Siega-Riz, Hobel, Aurora, & Dunkel-Schetter, 2001).

Notably, in follow-up studies of women with anorexia nervosa, even those who no longer met diagnostic criteria tended to maintain relatively low body weight and cognitive features characteristic of anorexia nervosa (perfectionism and cognitive restraint; Sullivan, Bulik, Fear, & Pickering, 1998). This could reflect lingering subthreshold symptoms and continuing efforts toward food restriction and weight control. It is possible that the preterm births and SGA babies in these women observed in these studies are secondary to the persistence of genetically mediated eating disorder symptoms that “fly under the radar” diagnostically yet are sufficient to impact fetal growth and development. Along these lines, although few data exist, the wide availability of fertility treatments may increase the likelihood

that individuals with eating disorders who have experienced reproductive complications of the eating disorder will conceive. The shame and secrecy associated with an eating disorder, as well as the fear of being turned away by fertility specialists, may result in underestimates of the prevalence of eating disorders in fertility clinics (Norre, Vandereycken, & Gordts, 2001). This could mean that more individuals in partial recovery will become pregnant and, tragically, not receive adequate perinatal counseling to ensure adequate nutrition during pregnancy. Indeed, several studies have confirmed that women with eating disorders have higher rates of pregnancy complications, cesarean deliveries, and postpartum depression (Bulik et al., 1999; Franko et al., 2001; Franko & Spurrell, 2000; Franko & Walton, 1993). In summary, women with histories of anorexia nervosa tend to maintain low BMIs even after recovery and tend to give birth to lower birth-weight babies, potentially increasing their offspring’s risk for the development of later chronic disease (Barker, 1992; Barker et al., 1993), including possibly anorexia nervosa (Cnattingius et al., 1999). Thus, an intricate cycle of genetic factors (influencing weight gain during pregnancy) and environmental factors (fetal undernutrition in utero) coupled with the direct heritability of eating disorders could be acting in concert to influence offspring eating disorders risk.

How Genes Can Help Us Understand Environment

We have no doubt that the remarkable historical changes in traits such as eating disorders, fertility, and obesity reflect environmental changes. However, individuals are differentially genetically susceptible to environmental shifts. How can discovering “liability genes” help researchers to understand the impact of environment? Caspi et al. (2002, 2003) reported two examples involving genetic variants that alter gene expression, a variant in the gene MAOA encoding the neurotransmitter-metabolizing enzyme monoamine oxidase A and a variant in the promoter region of the gene SLC6A4 encoding the serotonin transporter. These authors showed that a child’s response to maltreatment early in life depended critically on variation at MAOA: Maltreated children were stochastically more likely to develop behavior problems when they had low levels of MAOA expression than when expression was high. For SLC6A4 variation, they showed that individuals carrying one or two copies of the “short” allele of the promoter polymorphism, which produces lower expression of SLC6A4, were more likely to exhibit more symptoms of depression and suicidality after experiencing stressful life events than were individuals carrying two copies of the “long” allele. With appropriate caution, this model may provide a beacon for the future. This approach will be most appropriate in the presence of clearly defined a priori hypotheses and selections of environmental risk factors and genetic variants. Given the abundance of potential environmental factors from which to choose, as well as the number of genes likely to be implicated, the dangers of multiple testing yielding false-positive results are considerable. If we were to iden-

tify similar examples of Gene \times Environment interactions in the field of eating disorders, it would be the first step in unraveling the manner in which genes and environment act together in eating disorders—a definitive step away from what has been a field bifurcated by the nature versus nurture dichotomy for far too long.

Overall Conclusion

Evidence has accumulated in support of both biological (genetic and early developmental trauma) and cultural factors contributing to the increased risk for the development of eating disorders or associated behaviors and attitudes. Research on biological factors and cultural factors has progressed largely along parallel tracks. Future studies must explore these two classes of risk factors in tandem while remaining cognizant of the broader cultural context in which the disorders emerge. Entire classes of risk factors (e.g., personality, familial environment factors) have not yet been studied prospectively. Multimethod research approaches are needed to advance our understanding of the etiology of eating disorders. Comprehensive epidemiological studies are needed that capture more fully the clinical presentation of eating disorders in demographically diverse populations. Empirically based classification systems should be validated using hypothesized risk factors. Researchers need to exploit more fully the availability of longitudinal data that have been collected in samples that have been followed from birth or early childhood to generate hypotheses about the contribution of risk variables like personality feature or early family environment to the development of eating pathology. In addition, new longitudinal studies need to be initiated that are designed to test etiological hypotheses. Clearly, samples sizes numbering in the tens of thousands will be required to ensure adequate power for examining risk for the onset of full-syndrome disorders. The existing literature also points to the promise of experimental studies with high-risk populations (e.g., offspring of mothers with eating disorders) for testing the contribution of certain risk factors.

Our review points to several conclusions about stigma, nosology, treatment, and prevention of eating disorders. The evidence shows that we live in a culture that values thinness and that exposure or social pressure to conform to this norm contributes to body image concerns. The cultural context of thin idealization provides an all-too-easy explanation of eating disorders as the consequence of pursuit of beauty, contributing to a perception that these problems are self-inflicted and undeserving of serious consideration. The evidence is fairly strong that biological factors contribute to risk factors for anorexia nervosa. The dissemination of such findings may finally dispel the damaging and disrespectful myth that anorexia nervosa is vanity run amok. Moreover, the almost wholesale exclusion from research of individuals from racial or ethnic minority groups and the marginalization of males cannot be justified with (unsubstantiated) arguments that eating disorders affect only White girls or women and contribute to stigma-

tization of members of these underresearched groups when they present with an eating disorder.

This review also points to significant flaws in our nosology. With the majority of individuals seeking treatment being captured under an umbrella of a residual diagnosis (EDNOS), we have failed to characterize adequately the core pathology profiles that comprise the eating disorders. In part, this may be a result of our need to find satisfying and plausible explanations for perplexing symptom clusters. For decades, our patients with anorexia nervosa have said “it’s not about looking like a model.” We nonetheless persist with the explanatory framework of the thin-ideal internalization regardless of eating disorder type. Although there is no question that thin-ideal internalization is damaging, the mechanism whereby it influences risk for eating disorders remains unknown. Perhaps an example of *effort after meaning* (Bartlett, 1932), we have created, for example, a composite picture of anorexia nervosa that includes low body weight (which may be more biologically mediated) and undue influence of weight on self-evaluation (which may be more environmentally mediated) under one diagnostic label. Similarly, bulimia nervosa includes binge eating and self-induced vomiting (both apparently genetically mediated) and undue influence of weight on self-evaluation (apparently more environmentally mediated). Our hodgepodge diagnoses may not reflect underlying biological processes, may not adequately capture the population of interest, and may impair our ability to identify either genetic or environmental risk factors for disease. Thus, we encourage a critical reappraisal of our current nosology as both biological and sociocultural research unfolds.

The findings also point to population groups at high risk (e.g., offspring of mothers with anorexia nervosa, children with certain neonatal complications) and suggest the need to test targeted preventive interventions for these groups. The genetic branch of eating disorders research is in its infancy. Extensions of genetic research exploring not only the genetics of risk but also the genetics of course of illness and treatment response (both biological and psychological interventions), as well as more sophisticated models of Gene \times Environment interplay, will launch the next generation of genetic investigations. Moreover, although not reviewed here, greater cross-talk between animal and human researchers will help us unveil new biological mechanisms that influence eating disorders, and greater cross-talk between obesity and eating disorders researchers could only serve to enrich and inform both fields.

The corpus of findings from the sociocultural theory of eating disorders literature underscores the importance of preventive and policy interventions designed to decrease exposure to or attenuate the impact of thin-ideal messages. Even if we cannot prove that these factors cause eating disorders, we can safely say that they negatively influence the self-perceptions of young women. This fact has become even more critical in the face of the obesity epidemic, around which television shows, prevention programs, community and workplace interventions, and school-based pro-

grams designed for the explicit purpose of weight loss or weight control have arisen rapidly without adequate testing for their impact on individuals who are vulnerable to eating disorders. Public health interventions countering overweight and obesity can be carried out in a manner that addresses simultaneously the needs of those individuals at risk for overweight and obesity and those individuals at risk for eating disorders (Neumark-Sztainer, 2005). Yet the rapidity with which such programs have arisen has outpaced our ability to evaluate both their beneficial effects and their potentially adverse side effects.

Perhaps most critical, as a field, we have also been a victim of marginalization. The U.S. Centers for Disease Control has failed to collect rigorous epidemiologic data regarding eating disorders and disordered eating behavior. The World Health Organization has not published facts on disability-adjusted life years (DALYs) for anorexia nervosa or bulimia nervosa, whereas DALYs for similarly prevalent conditions (e.g., obsessive-compulsive disorder) have been reported. We attribute much of this marginalization to pervasive misperceptions about the volitional nature of eating disorders, which has impacted research; third-party reimbursement; and most tragically, families and sufferers who have known all along that eating disorders are far more grave than merely a choice to pursue thinness.

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