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# Environmental and genetic risk factors for eating disorders: What the clinician needs to know

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## Synopsis

Patients and families are often aware of research on genetic factors influencing eating disorders. Accurate interpretations of research on environmental and genetic risk factors can be empowering to patients and families; however, misinterpretations could prove detrimental. The clinician who is not versed in genetic research may feel ill-prepared to discuss the nuances of genetic research with patients and families. In this paper the authors discuss what is known about genetic and environmental risk factors with an emphasis on gene-environment interplay in order to increase clinicians' comfort level with discussing these complex issues with their patients.

### Keywords

Anorexia nervosa; bulimia nervosa; binge eating disorder; genetic; environment; risk

The significant role genetic factors play in the development of eating disorders is becoming increasingly clear [1–3]. Family studies of anorexia nervosa and bulimia nervosa have consistently found a higher lifetime prevalence of eating disorders among relatives of eating disorder probands than among relatives of controls [4–6]. Further, numerous twin studies [3, 7–10] suggest that liability to anorexia and bulimia nervosa is significantly influenced by additive genetic factors. Although somewhat less developed, family and twin studies point towards binge eating disorder being both familial [11] and influenced by additive genetic factors [12,13]. Of note, although many molecular genetic studies have been conducted, they have yet to yield an unambiguous replicated finding (see [14–16] for reviews). The emergence of powerful and novel approaches such as genomewide association studies, could bring significant advances to the field. Given that the effect size of single gene variants is likely to be small, large collaborative investigations are required in order to identify risk alleles. We propose that advances in genetic research will assist us not only with identification of risk alleles, but will unlock new methodological approaches to help identify differential risk to environmental risk factors dependent on genotype.

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Although genetic epidemiology of eating disorders has emerged with force in the past decade, environmental risk factors have received the bulk of research and clinical attention [17–19]. In particular, sociocultural influences, such as unrealistically thin media images have been hypothesized to promote disordered eating and body dissatisfaction [20,21] on the causal pathway to eating disorders. However, it is clear that, although virtually all women are exposed to these sociocultural influences, only a very small proportion develop clinical eating disorders [22].

Ultimately, the elucidation of causal models for eating disorders will no doubt include various types of genetic and environmental interplay. One of these is gene-environment correlation (or $r_{GE}$), which has been described as occurring "when the exposure to positive or negative environmental influences is not randomly distributed with respect to genetic differences" ([1], pp. 12–13). Three types of G-E correlations have been described in the literature [1,23]: passive, evocative, and active. Passive gene-environment (G-E) correlations occur because children receive genes from the same individuals who create their family environment (unless they are adopted). Thus, the same parents who may be passing down genes that influence liability to eating disorders may also be modeling eating disordered behaviors (e.g., restriction, compulsive exercise), and attitudes (e.g., body dissatisfaction, drive for thinness) to their children. Children in these circumstances could be receiving a "double-dose" of eating disorder risk, as a result of both genetic and environmental exposures.

G-E correlations may also be evocative, or evoked by the individual with a genetic predisposition to a given disorder [1]. For example, an individual with a genetic predisposition to an eating disorder may disproportionately seek out appearance-related comments from parents or peers. These comments may be positive or negative; what is particularly important about them is that they reinforce the individual's tendency to over-value appearance, and may promote the initiation or maintenance of eating disordered behaviors. So, for example, a young girl who is dissatisfied with her body may constantly ask her parents for reassurance that she does not look fat. The resultant environment may seem very appearance focused, but that emphasis may actually be evoked by this reassurance-seeking behavior.

Finally, active G-E correlations occur when an individual with genetic vulnerability to an eating disorder seeks out environments that reinforce a strong emphasis on appearance, such as modeling or gymnastics [1]. Thus, the preponderance of disordered eating in ballet companies (e.g., [24,25]) may not only be due to the influence of ballet on disordered eating, but may also reflect the fact that individuals prone to eating disorders select themselves into that high risk environment.

A second type of gene-environment interplay is gene x environment interaction (G x E). Unlike $r_{GE}$, in which an individual's genotype influences the *likelihood of exposure* to environmental risk factors, G x E interaction occurs when an individual's genotype influences his/her *vulnerability, or response to environmental risk factors* ([26,27]. For example, the influence of a weight-preoccupied coach on the eating disordered behaviors of the athletes s/he coaches is likely to vary as a function of the athletes' genotype.

The analysis of G-E interplay is complex, and, as Klump et al. [23] have noted, our ability to unravel the nature of gene x environment interplay will be enhanced once we have identified true risk alleles for eating disorders. Ultimately, genetic research may prove to be the key to unlocking our understanding of environmental risk factors for eating disorders by helping us enhance our specificity in identifying *which risk factors* affect *which individuals*, resulting in *which eating disordered behaviors.*

Although genetic research in eating disorders is frankly in its infancy, patients, families, and clinicians are aware of this research and face challenges in incorporating this knowledge into

either their personal conceptions of their (or their family member's) illness, or in the case of clinicians, helping patients and their families to understand the implications of this knowledge. Simplistic nature versus nurture dichotomies are easy to understand, yet rarely capture the complexity of reality, and definitely fail to do so in the case of eating disorders. Clinicians and researchers must become educated in the nuances of gene x environment interplay and avoid perpetuating purely environmental or purely genetic conceptualizations of eating disorder etiology [23].

Many excellent reviews of environmental risk factors [17,18,28,29] for eating disorders and genetic influences on eating disorders [14–16] exist. The interested reader is directed to those sources for comprehensive treatments of each. In this paper, we will not duplicate these excellent efforts, but rather will highlight what is known about risk factors for eating disorders within the conceptual framework of gene and environment interplay. In addition, we will discuss the clinical implications of these correlations and interactions, including ways of incorporating this knowledge into practice and directions for future treatment and prevention efforts.

## A Plausible Scenario of How Genes Might Influence Eating Disorders

A question commonly posed by clinicians, families and patients alike is, how do genes work in influencing risk for eating disorders? The lay conception of genetics tends to over-emphasize the deterministic aspect of genetic risk. Modeled after Mendelian one gene-one disorder examples (e.g. Huntington's), the misperception emerges that there is one gene for anorexia nervosa and if you have that gene you are destined to develop the condition. Clinicians are well-positioned to dispel these myths and offer more realistic albeit complicated explanations for complex inheritance patterns. By definition, eating disorders are complex traits. That means that their inheritance pattern in families does not follow traditional Mendelian patterns, and that they are influenced by multiple genetic and environmental factors of small to moderate effect. There is not one gene for anorexia nervosa or one gene for bulimia nervosa. More likely there are a number of genes that code for proteins that influence traits that index vulnerability to these disorders. Complicating the risk picture even further, these genes exist in concert with other genetic factors that may confer protection against eating disorders, along with main effects of risk and protective environments, as well as gene x environment interplay as we discuss in the following section.

## Integrating Risk Factor Research with a Genetic Epidemiological Perspective on Eating Disorders G-E

### Relationships Potentially Influencing Eating Disorder Etiology

For decades, parenting styles have been unrightfully blamed for causing eating disorders. Considerable care must be taken when discussing gene x environment interplay not to convey the message that somehow parenting is to blame for these pernicious illnesses. Conversely, a purely genetic explanation should not be taken to mean that parents need not examine their parenting style and the influence that might have on their children. The context for the following discussion is that parenting does matter. Moreover, parenting style is in part influenced by the parents' genes, and the effect of parenting style is similarly influenced by the offsprings' genotype. For an example outside of the eating disorders world, alcoholism has a known genetic component. An alcoholic father's erratic and authoritarian parenting is in part influenced by his genetic predisposition to alcoholism. His two children may be differentially influenced by that parenting style due to their own genetically influenced constitution—one may be vulnerable and sensitive to rejection and verbal abuse whereas the other may remain effectively

impervious to rejecting parenting. We will now explore some of the identified risk factors for eating disorders through this complex lens.

**Parental Role Modeling of Eating Disordered Behavior—**In the clinic, it is not uncommon to see either frank eating disorders or shadows of disordered eating, appearance focus, exercise obsession, body preoccupation, or personality traits associated with eating disorders such as perfectionism or obsessionality in parents of individuals with eating disorders. Of course, this is not always the case, and there are many examples of families in which these traits are not evident. Through their relationships with food and their own bodies, parental role modeling of unhealthy eating behaviors and attitudes likely represents an example of passive G-E correlation. The literature suggests that such modeling has an effect on offspring, as case studies indicate that children do indeed imitate their mothers' purging and restricting behaviors [30–32]. Studies conducted in non-treatment-seeking samples have found similarities between mothers' and daughters' restraint and dieting behaviors among children as young as 10 years old [33]. Mothers' comments about their own weight and appearance are associated with the body esteem of their fourth and fifth grade daughters and sons [34], and mothers who complain about their own weight are more likely to have daughters who are weight concerned. Moreover, maternal drive for thinness and restraint are associated with the development of overeating behaviors among children in their first five years of life [35]. Thus, although not solely causal, the principle of children being more likely to "do as you do" rather than "do as you say" renders parental role modeling of healthy eating and weight behaviors important to the task of creating a protective environment—especially in genetically vulnerable offspring.

**Problematic Feeding Behaviors—**A second area where parental genotype and environment intersect dramatically is in the feeding behaviors of children. Mothers tend to retain primary responsibility for nurturing their offspring. For most mothers this is a joyous part of motherhood; however, for women with eating disorders this can represent an emotionally charged and highly challenging part of the job. Clinicians working with mothers with eating disorders should be keenly aware of the potential difficulties they may encounter in what should be this very natural mother-child interaction. Studies have shown that mothers with eating disorders are more likely to use food for nonnutritive purposes, such as rewarding, comforting, or punishing their children [36,37]. One retrospective study found that adults whose parents used food in this manner were more likely to exhibit disordered eating behaviors than those who did not recall such parenting behaviors, even when factors such as current Body Mass Index (BMI), childhood weight status, ethnicity and age were controlled [38].

Mothers' own eating restraint and disinhibition also appear to influence their approach to feeding their children. In two studies of five year old girls, mothers' restraint and weight concerns were positively associated with the degree of restriction they imposed on their daughters' eating [39,40]. Maternal disinhibition of eating was also related to children's eating behavior. For example, Johnson and Birch [41] found that parental disinhibition was inversely associated with children's ability to self-regulate energy intake. For both sexes, a tendency to overeat in the presence of food-related cues may be transmitted across generations.

**Parental Over-Emphasis on Child Weight and Shape—**In addition to having concerns about their own shape and weight, women with eating disorders may similarly be over-concerned about their children's weight, even when it is well within normal limits [30,36,37]. One study [36] found that 15% of mothers with a history of bulimia nervosa had attempted to "slim down" their normal weight infants. Similarly, Waugh and Bulik [42] found that 20% of the mothers with eating disorder histories in their sample tried to change their children's appearance. Maternal restriction of children's eating is a concern, as previous research [41] suggests that maternal control of children's eating interferes with the development of dietary

self-regulation. These effects have been found among children as young as two years of age [41].

In another example of how genes and environment may interact, mothers' disordered eating attitudes appear to influence their perceptions of their children's appearance. For example, Stein and colleagues [43] found that mothers' satisfaction with their children's size was inversely related to the severity of their own eating disorder symptomatology. Further, other research has found that mothers' comments about their children's weight were associated with children's body esteem and the frequency of children's weight loss attempts [44].

The influence of maternal perceptions on daughters' eating disordered behavior appears to continue into adolescence and young adulthood. For example, mothers whose daughters engaged in eating disordered behavior were more likely to view their daughters as overweight [45,46] than were mothers of non-eating disordered daughters (controlling for weight differences between groups). Further, this appearance pressure is not necessarily limited to weight, as mothers of daughters with eating disorder symptomatology also rated their daughters as less attractive than the daughters rated themselves [46]. These results are concerning, as they suggest that daughters who engage in eating disordered behaviors may not only lack a maternal role model of healthy eating, but also feel maternal pressure to lose weight and enhance their appearance. This of course could reflect several complex intergenerational processes. On one hand, mothers may harbor threshold or subthreshold eating disturbances themselves, and their comments or behaviors could reflect their underlying pathology. Alternatively or complementarily, the daughters' pathology could render them more sensitive to maternal comments that in other situations may be perceived as culturally normative. These environmental experiences could facilitate the expression of an existing genetic predisposition for eating disorder symptomatology.

**Life Events and Distress Tolerance—**Historically, research on parenting and its relation to eating disorders has focused on family/parent characteristics, an important component of which is termed "shared environment" in twin research. Shared environment refers to those familial experiences that act to increase similarity amongst family members. However, genetic epidemiological research has suggested that the majority of environmental variance in eating disordered symptomatology is influenced by "unique environment," or environmental factors experienced by only one member of a twin pair or family [23,47]. Unique environmental influences act to increase dissimilarity amongst family members. However, even the definitions and existence of shared and unique environmental factors are disputed. For example, adverse life events, such as abuse experiences to which only one member of a twin pair was subjected, might exemplify unique environmental factors salient to the development of an eating disorder. Yet, it has been posited that the frequency and intensity of defining unique environmental events [48], as well as the impact of these unique life experiences are at least partially under genetic control [49]. For example, Klump and colleagues [23] stated, "Genetic differences … may provide the mechanisms by which nonshared environment exerts its influence" (p. 123). Thus, it seems appropriate to review research on adverse life events and their association with eating disorder etiology from a genetic epidemiological perspective.

As noted above, adverse life events may be one significant way in which the unique environment can act to increase eating disorder risk. The association between negative life events and the onset of eating disorders has received significant clinical and research attention [23,50–52]. However, as Schmidt and colleagues [51] have noted, the majority of studies in this area have used case reports to identify life events, and did not include non-eating disordered comparison groups. However, one case-control study [52] found that some adverse experiences were more common among women with bulimia nervosa in the year prior to illness onset (e.g., a major move, illness, pregnancy, physical abuse, and sexual abuse). Further, the more life

events an individual experienced in the last year, the more likely she was to have bulimia nervosa. However, the frequency of several other life events (e.g., bereavement, illness of a close relative, friend or partner, and beginning or end of a new romantic relationship) did not differentiate women with and without the disorder. In addition, nearly one-third of women with bulimia nervosa did not experience any of the life events assessed in the previous year. Thus, although there appears to be some association between experiences of adverse life events and bulimia onset, there is also significant variability among affected individuals with respect to recent experiences.

The impact of recent life events on the development of binge eating disorder (BED) was investigated by Pike et al. using a case-control sample [50]. Women with BED experienced more adverse life events in the year prior to the onset of their eating disorder (compared to nonclinical and psychiatric controls), and the likelihood of BED was positively associated with the frequency of negative events. Compared to both the non-clinical and psychiatric control groups, women with BED more frequently reported major changes in life circumstances and relationships. Physical abuse, perceived risk of physical abuse, safety concerns, stress, and experiences of weight and shape-related criticism were also more common in the BED group (vs. non-clinical controls).

The impact of stressful life events is obviously at least partially dependent upon how individuals perceive, or think about these experiences, and how they attempt to cope with them. Thus, studies on the impact of life events on eating disorder etiology should also be considered in light of the significant body of research supporting the influence of cognitive appraisal on psychological outcomes [53,54]. According to appraisal theory, the impact of life events depends on how they are appraised by the individual [53,54]. The appraisal process involves both determining if a given situation is threatening to the individual (primary appraisal) and if so, evaluating whether his/her resources are sufficient to manage this threat (secondary appraisal). It seems plausible that both genetically-influenced traits (e.g., trait anxiety, fearfulness), and environmental factors (social support, SES) would influence primary and secondary appraisal. This hypothesis is supported by the results of a twin study [55] which found that specific coping behaviors (turning to others and problem solving) were significantly influenced by genetic factors.

Distress tolerance is another construct related to appraisal and coping processes. The degree to which a given individual possesses the ability to tolerate distress also is likely influenced by a complex combination of genetic and environmental factors. According to Corstorphine et al., distress tolerance is, “the ability to endure and accept negative affect, so that problem-solving can take place…[and it] manifests as high emotional vulnerability and an inability to regulate emotion” [56], p. 91). Although no extant research has investigated the heritability of the specific construct of distress tolerance, related variables, such as neuroticism, have been found to be strongly familial [57,58] and to predict the later development of anorexia nervosa [59]. Further, emotion regulation difficulties have been found in women with eating disorders of all subtypes (e.g., [60,61]). A recent study [56] found that eating disordered attitudes were associated with scores on two components of a distress tolerance measure: high avoidance of affect and low acceptance and management of problems.

Research in other clinical areas has indicated that distress tolerance skills can be improved with the implementation of a specific cognitive-behavioral intervention (e.g., Dialectical Behavioral Therapy [DBT] for Borderline Personality Disorder, [62]). Thus, interventions aimed at individuals at high-risk for eating disorders (and thus potentially genetically prone to low distress tolerance) should perhaps include elements of DBT focusing on affect regulation and coping skills training. Thus, in this manner, an environmental intervention could directly address genetic vulnerabilities. Future studies of G-E correlations should include the construct

of distress tolerance, in order to evaluate its interaction with life events and specific genetic vulnerabilities.

## Evocative G-E Correlations Potentially Influencing Eating Disorder Etiology

**Interaction of Temperamental Style and Environment—**Genes may also influence risk for eating disorders through the way in which one's genotype evokes certain responses from the environment. For example, considerable work has explored temperamental style in individuals with eating disorders. In the current context, we are interested not only in how temperament may be associated with eating disorders risk, but also in the various ways that temperament influences interactions with the environment. As suggested above, personality characteristics (such as neuroticism) can make us more or less vulnerable to environmental insults; but these traits can also influence our tendency to evoke certain responses from the environment.

Recent work on temperamental factors influencing eating disorder etiology indicates that anorexia nervosa is associated with a temperamental style characterized by perfectionism, a need for order and exactness, harm avoidance, and sensitivity to praise and reward [63–66]. Bulimia nervosa shares many of these same characteristics, but commonly coupled with features of novelty seeking and impulsivity [64,65,67]. These temperamental traits have been shown to be influenced by genetic factors [68,69]. However, these temperamental characteristics also influence how individuals interface with their environment. Thus, it is plausible that individuals genetically susceptible to eating disorders due (to some degree) to their temperament, may hold themselves to extremely high standards, and through their own actions, actively seek out praise or evaluation of their efforts from others. Comments evoked from others, even if positive, may in turn reinforce perfectionist tendencies, including those relevant to eating disordered behavior.

On the other hand, parents may treat even identical (or monozygotic, MZ) twins differently, which could also influence eating disorder susceptibility. For example, in a study of MZ twins discordant for bulimia nervosa, Wade et al. [70] found that affected twins reported higher parental expectations than their unaffected co-twins. It is not possible from this study to determine if parents actually did hold one twin to higher standards, or, if this finding reflects the retrospectively recalled experience of the affected twin. That is, the answers may reflect the affected twin's attempt to make sense of her childhood experiences and current symptomatology. Longitudinal studies are needed to clarify the role of parental expectations (both actual and perceived) in eating disorders etiology.

**Teasing and Weight Shape Related Criticism—**Another environmental issue relevant to eating disorders risk is weight related teasing, and critical comments about weight (particularly those made by parents) which have been identified as risk factors for a range of eating disordered behaviors (e.g., [18,50,70,71]), although not all studies have identified this association (e.g., [19]). The association between teasing and risk for eating disorders is unlikely to be a simple one and a complete understanding of risk mechanisms needs to consider gene x environment relationships.

Klump et al. [23] suggested that weight-related teasing may reflect an evocative G-E correlation relevant to eating disorders. For example, teasing experienced by an overweight adolescent may be in part evoked by his or her simply being overweight. Thus, in this case, a trait significantly influenced by genetic factors (BMI), influences the comments an individual is exposed to in the environment.

Another possible way in which genetic and environmental factors may interact to influence teasing–related risk is that teasing experiences may be appraised more negatively among

individuals with different genetically-influenced temperaments. Individuals genetically predisposed to eating disorders and neuroticism for example, may be particularly prone to ruminate about weight-related teasing or critical comments about weight, thereby exacerbating the negative effects of these occurrences. Supporting this observation, clinicians report that many individuals with eating disorders recall triggering events that prompted their eating disorder. Although highly salient and even defining to these individuals, many of these events fall within normative experience and could easily be brushed off by individuals less sensitive to their salience. As in the case with the overweight teen above eliciting comments about weight, it is also possible that individuals genetically vulnerable to eating disorders may elicit comments about their appearance through their frequent seeking of reassurance (and these comments may, in some cases, be critical). These evoked responses may reinforce their eating disordered tendencies or behaviors.

Further, it should also be considered that, as noted above, even MZ twins may be treated differently by their parents, particularly with respect to appearance. Research on MZ twins discordant for anorexia and bulimia nervosa found that affected twins report receiving more critical comments regarding their weight than their non-affected co-twins [70]. As these twins are virtually identical genetically, this finding may represent a true nonshared environmental difference. Nonetheless, specific genetic vulnerabilities (e.g., to a higher-than average BMI, or sensitivity to praise) may still influence the etiology of eating disorders in this case. It is also possible that these findings are influenced by recall bias, as participants were not surveyed about their childhood experiences until they were already adults. Thus, perhaps affected twins affected were more likely to view parent comments regarding weight as salient to their current functioning.

## Active G-E Correlations Potentially Influencing Eating Disorder Etiology

**Media—**One potential pitfall of genetic research on eating disorders is the misinterpretation that environmental factors such as the media do not matter. Western media's idealization of an ultra-thin female body type has long been viewed as an important sociocultural risk factor for eating disorders [72,73]. However, given the ubiquity of this influence in Western cultures, other factors must influence vulnerability to the thin cultural ideal. As Bulik [1] suggests, genetically vulnerable individuals might seek out experiences, such as exposure to thin-ideal media images, which reinforce their negative body image. This hypothesis is supported by a longitudinal study which found that adolescent girls whose eating disorder symptomatology increased over a 16 month period also reported significantly greater fashion magazine reading at Time 2, compared with Time 1 [74].

**Peer Group Selection—**Similarly, individuals genetically predisposed to eating disorder symptomatology such as thin-ideal internalization might also actively choose to affiliate with peers who place a similar high value on weight and appearance [23]. One potential example of this form of active selection could be the decision to join a sorority (particularly for European-American women). European-American sorority members report high levels of eating disorder symptomatology, including weight preoccupation, drive for thinness, and body dissatisfaction [75]. A longitudinal study [76] found that sorority and non-sorority members did not differ on three measures of disordered eating (EDI Drive for Thinness, Body Dissatisfaction, and Bulimia) at Time 1 and Time 2 (first and second year of undergraduate, respectively). However, by Time 3 (third year of undergraduate), non-members' drive for thinness scores had decreased, while members' scores on this measure remained roughly the same, and this difference was statistically significant. Thus, the authors concluded that characteristics of the sorority environment could contribute to the persistence of a higher degree of drive for thinness. Although this study did not include a measure of actual or putative genetic vulnerability to eating disorders, it is plausible to speculate that an environment that promotes

the maintenance of eating disordered characteristics would be particularly problematic for a genetically vulnerable individual.

## Potential Environmental Buffering Effects for High-Risk Groups

We have focused primarily on environmental risk factors and how they could plausibly interact with various facets of genetic vulnerability to increase risk for eating disorders. Given our inability to modify genetic risk in this point in time, it is critical to present a balanced picture of the environment. Just as both risk and protective genetic factors can exist, so can both risk and protective environmental factors. Also, protective factors may function differentially depending on the genotype of the exposed individual. Given the field's focus on pathology, much less is known about differential effects of buffering environmental factors based on genetic differences.

**Family meals and breakfast eating—**One fascinating and modifiable environmental factor that has emerged as a possible buffer against the development of eating disorders in adolescent girls is family meals [77]. Likewise, breakfast eating may also play a role in preventing the development of eating problems. For example, Fernández-Aranda and colleagues [78] found that women with eating disorders were less likely to have eaten breakfast regularly during childhood compared to non-eating disordered controls. Although retrospective, these findings are consistent with those of a large ($n$ = 2216), longitudinal study which found that breakfast eating frequency was inversely associated with dieting and weight-control behaviors, and positively related to dietary quality and physical activity in adolescents [79]. Overall, these studies offer preliminary insight into potential buffers against eating disorders; however, research in this area has not yet progressed to assess the differential effect of these protective factors in individuals at high-risk for eating disorders versus the general population.

**Distress tolerance/anxiety management—**Another factor that offers promise as a potential buffer against the development of eating disorders is the enhancement of emotion regulation skills. As noted above, individuals with eating disorders experience relatively high levels of perceived stress and difficulties regulating emotion [56,61]. Thus, interventions aimed at enhancing emotion regulation skills might be of particular benefit to high-risk groups. However, research incorporating mindfulness techniques has not specifically targeted high-risk groups. For example, a recent study investigated the effectiveness of a primary prevention program incorporating elements of mindfulness (e.g., yoga), targeting fifth-grade girls [80]. This program integrated mindfulness into an empirically-based curriculum, which also included other elements, such as media literacy, and the promotion of dissonance regarding idealization of an ultra-slim body type. Compared to a control group, girls in the intervention reported lower body dissatisfaction and uncontrolled eating, and higher social self-concept at post-testing. However, there were no significant changes on other variables assessed including drive for thinness, perceived stress, physical self-concept and perceived competence. Nonetheless, these outcomes do provide some support for the inclusion of mindfulness-based activities in prevention. In contrast, a study with undergraduate women [81] did not find any differences between participants in a yoga program and a control group on eating disorder symptoms at post-testing. Future studies should target high-risk groups, to evaluate the efficacy of mindfulness-based techniques within this specific sample.

**Reducing Weight and Shape-Related Attentional Biases—**Previous studies have found that individuals with eating disorders manifest attentional biases (or selective attention) toward weight and shape-related information [82,83]. These biases are likely influenced by both genetic and environmental factors. However, a recent study [83] found that these biases were reduced in women with eating disorders following completion of a specific form of

cognitive-behavioral therapy. Future research should investigate the effectiveness of incorporating some of these specific cognitive-behavioral techniques into prevention programs targeting high-risk individuals.

In summary, simplistic nature versus nurture explanations could never suffice to capture the many ways in which genes and environment may interact to influence eating disorder risk. Moreover, many of these processes are likely to be operative over time in any given individual and risk may be cumulative. The critical message for the clinician working with eating disorders is not necessarily to educate patients and parents about all potential forms of gene x environment interplay, but definitely to guard against simplistic explanations that lead to inaccurate conclusions and inappropriate solutions.

## Clinical Implications

After reviewing the various and complex ways that genes and environment can interact and correlate, the clinician, although enlightened, will want to know how best to incorporate this knowledge into clinical practice. Given the state of the science of treatment for eating disorders —especially in the absence of any effective medications for anorexia nervosa—the question remains how best to work with the patient and family to bolster protective environmental influences, reduce evoked environmental exposures, and develop strategies in the patient to minimize the deleterious effects that sensitivity to the environment can create.

**Focus on the parents—**One prong in the approach to incorporating genetics into clinical work focuses on the parents of individuals with eating disorders. Whether the parents are involved in parent training, traditional family therapy, or other types of supportive interventions, they can be educated about genetic factors influencing eating disorders. A sensitive explanation that incorporates knowledge about complex genetic etiology (not the one gene-one disease model) and about how genes and environment interact can serve to relieve guilt in parents who have been blamed for creating the illness in their offspring (or alternatively erroneously assumed that their parenting was to blame). A genetic and biological explanation can help parents understand that their child's resistance is not just stubbornness or deviousness, but that in his or her recovery, their child is fighting an uphill battle against his or her biology. This knowledge can empower parents to understand and can decrease frustration. Care should be taken that parents do not transform this knowledge into a new form of guilt (i.e., feeling guilty for passing on risk genes), as the roll of the genetic dice is one thing over which we have no control. Likewise, care should be taken not to allow the genetic information to impart complete absolution on parents, as parenting can always improve and positive parenting changes should also be prescribed as part of treatment. Finally, symptomatic parents should be referred for treatment. Although few data exist, clinically, it is an uphill battle for offspring to recover from an eating disorder if they are faced on a day to day basis with a parent who is actively suffering from the illness.

**Focus on the next generation—**Intervention with mothers with eating disorders can be viewed as a form of targeted prevention. Eating disorder prevention is a complicated endeavor. Although primary prevention deserves additional research attention, interventions targeting individuals at risk of developing eating disorders may be a particularly promising focus of prevention efforts [84,85]. Such intervention could serve to improve maternal efficacy and confidence in feeding their children, increase modeling of healthy eating behaviors and attitudes, decrease mothers' anxiety regarding parenting behaviors, and increase self-efficacy and confidence in general parenting skills. In the long term, this type of intervention may assist mothers with developing a healthy buffering environment which minimizes modeling of behaviors and attitudes associated with disordered eating.

Results from focus groups [86] and clinical case studies [87] suggest that mothers with eating disorders are eager to learn about how best to care for their children, especially with respect to feeding. However, they report that the level of assistance they desire is not routinely offered by their health care providers. In one of the only published interventions conducted with this population, Stein et al. [59] studied 80 mothers with eating disorders and their four to six month old infants to test whether a 13 session intervention of video-feedback treatment in conjunction with cognitive behavioral self-help was more effective than cognitive behavioral self-help alone in reducing mealtime conflict and other aspects of maternal-child interaction. Those mothers in the video-feedback group exhibited significantly less conflict than control mothers as well as significant improvements in infant autonomy and several other interaction measures. In addition, maternal eating psychopathology was reduced across both groups. Such interventions could help break the "cycle of risk" associated with eating disorders [16], by providing parents with useful buffering strategies.

**Focus on the patient—**Patients read enormous amounts about their illness and are often aware of the genetic research on eating disorders yet they struggle to understand what the data mean for them and the challenges they face every day during recovery. Helping patients to understand the genetic literature is a first step. Although they might not initially see its relevance to their situation, helping them map how disordered eating and temperamental traits track in their families by using techniques such as labeling family trees can provide a useful context for understanding genetic and environmental contributions to their current situation. An understanding of genetic and environmental interplay can provide them with an explanatory model for not only their illness, but also for understanding their sensitivity to the environment. It can help provide them with the motivation to acquire skills that may help buffer them from the environment and combat their biology most effectively.

## Summary

Although several decades ago there was significant debate about the influence of "nature" versus "nurture" on the development of psychological traits and outcomes, it is now generally accepted that both genes and environment interact to influence personality and behavior. However, in the clinical setting, genetic influences on clients' presentation of their personal histories, including characteristics of their family-of-origin environment, their perceptions of stressful life events, and their experiences within their social network, are not generally viewed from a genetic-epidemiological perspective. Similarly, risk factor research typically includes a broad range of individuals with varying genetic vulnerability to eating disorders. Thus, it is not possible to determine from the results of these large-scale studies *what risk factors* are particularly potent for *which individuals* with *what specific genetic vulnerability* to eating disorders. Moreover, given the relatively low base rate of clinical eating disorders in the population [22], it is possible that some potent environmental risk factors may be overlooked if they are not associated with eating disorder symptomatology in the general population. Thus, although genetic research has not yet progressed to a point where it can be informative for either treatment or prevention, it is time to develop interventions aimed at promoting buffering rather than predisposing environments. Such interventions could help families transform a double disadvantage into a single disadvantage. Given the current state of knowledge in the genetics and eating disorder fields such interventions may be the best approach to reducing risks for these pernicious and devastating disorders.

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