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Eating disorders: What are the risks?

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Eating disorders are characterized by severe disturbances in eating behavior and overconcern with body weight and shape. Onset is typically during adolescence. DSM-IV recognizes three eating disorders: anorexia nervosa (AN), bulimia nervosa (BN), and eating disorder not otherwise specified (EDNOS). While AN and BN are defined by specific criteria sets, EDNOS is heterogeneous and includes individuals who narrowly miss full criteria for AN and BN, as well as those presenting with a range of other symptom clusters, including binge eating disorder (BED). Eating disorders are difficult to treat, often associated with a chronic and relapsing course, high rates of psychiatric comorbidity and medical morbidity, and, specifically in AN, increased mortality risk. AN and BN have estimated prevalence rates of 0.5–1.0% and 1–4%, respectively; however the most common eating disorder presentation is EDNOS.¹ The etiology of these disorders is poorly understood: Are eating disorders sociocultural phenomena, neurobiological disorders, inherited conditions, or a combination of the three?

Research studies designed to identify risk factors for the development of eating disorders are critical. Such research can assist in identifying high risk groups to target for early intervention and advance the development of diagnostic, prevention, and treatment programs. Additionally, increasing understanding of the etiology of these disorders may decrease stigma associated with eating disorders. Yet this type of research is methodologically challenging. By definition, a risk factor temporally precedes and predicts the onset of a subsequent outcome.² The low base rate of eating disorders in the population, the multiple ages at which the disorders may onset, and the wide range of candidate risk factors and the difficulty and expense of their measurement all challenge risk factor research.

In this issue of the *Journal*, two research groups present longitudinal population-based cohort studies designed to examine risk for eating disorders. Nicholls and Viner³ consider a set of putative risk factors for the development of AN in a British cohort study. These included birth and infancy variables and follow-up data at five years and ten years, with assessment of lifetime eating disorders at age 30. Allen, Byrne, Forbes, and Oddy⁴ examine risk for the development of a range of threshold and subthreshold eating disorder profiles utilizing the Western Australian Pregnancy Cohort, which included pregnancy, birth, and childhood data with eating disorder outcome assessed at age 14.

These studies advance the field of risk factor research in eating disorders, representing two of the first longitudinal prospective cohort studies to examine putative risk variables for eating disorders in individuals followed from birth. While the methodology, cohorts, and outcomes of interest differ, the study findings are complementary and consistent with prior risk factor research in this area.^{5–6} Nicholls and Viner³ identified 101 cases of AN and found that female gender, early feeding or undereating problems, and maternal depressive symptoms predicted

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AN onset, while higher self-esteem and higher BMI conferred protection. Allen and colleagues⁴ similarly reported that female gender was a strong predictor of eating disorder pathology, and also found that being perceived as overweight by one's parent increased risk for eating disorder pathology.

This research is important in that it begins to elucidate variables that confer risk for AN specifically, and for eating disorder pathology more broadly. Additionally, the research is important because it affirms the difficulty of doing prospective epidemiological research for low prevalence disorders. Strengths of Nicholls and Viner's study include the large sample size, which resulted in detection of the largest number of AN cases in this type of research to date, and examination of specific candidate risk factors identified by previous meta-analysis and review.⁵⁻⁶ Allen and colleagues utilized psychometrically sound self-report measures of eating disorder outcome, considered eating disorder pathology of all types at varying degrees of severity, and further, considered risk for eating disorder development relative to other types of psychopathology.

Yet these studies highlight the challenges of risk factor research in eating disorders. While longitudinal study designs are requisite to determine causality, in spite of the large sample sizes in these studies, rates of the predicted outcome(s) were low. In the second study in particular, all eating disorders were combined into a single predicted outcome, which may miss nuances of differential risk for the range of eating disorders. Although these samples are carefully tracked and well characterized, classes of risk variables that may be important were not considered (e.g., genetic, neurobiological). Certain previously established risk variables including premorbid dieting, body dissatisfaction and internalization of a thin body ideal were not included in either study. Additionally, psychiatric comorbidity, in particular anxiety disorders, and its temporal relation to the eating disorder, was not examined. Further, potential gene-environment correlation or interaction effects were not considered.⁷ Indeed, while the collective body of risk factor research in the field of eating disorders indicates that female gender increases risk for eating disorder psychopathology, AN and BN specifically, and to a lesser degree, BED, questions remain about *why* females, *which* females in particular may be most at risk, and *when* (e.g., during which developmental windows).

The next wave of risk factor research in eating disorders should build on and learn from these studies. Longitudinal epidemiological studies *may* be of use in considering shared risk for the varying eating disorder groups, particularly in light of Fairburn and colleagues' proposed transdiagnostic conceptualization of eating disorders.⁸ In addition to confirming well-established risk factors, measurable variables that have been posited as candidate endophenotypes for eating disorders (e.g., set-shifting deficits, local processing biases) could be explored. Risk factor research should also utilize methodologies other than longitudinal epidemiological studies to elucidate relationships between environmental and biological variables that may increase risk for *certain* individuals.⁷ Longitudinal studies of high-risk groups, or family studies (e.g., discordant twin designs) may be viable. Indeed, some of the risk variables identified in these two studies suggest an interplay between individual or genetic variables and environment, particularly family. For example, parents who themselves have difficulties with eating, or eating disorders, model these attitudes and behaviors, in addition to passing on a biological vulnerability. Early feeding problems may result from an interaction between child temperament and parental overconcern with eating and therefore, be a proxy for a latent risk. Similarly, parents' perception that their child is overweight is likely influenced by parents' own eating/weight attitudes and behaviors; collectively, these may differentially increase youth eating disorder risk as they interact with individual child variables (e.g., self-esteem, negative affect). Youth who have a biological vulnerability toward the development of eating disorder pathology may elicit weight/shape concern because of their own focus on these, or seek out environments (e.g., activities) that reinforce a focus on eating/weight/shape.

Concomitant with advancements in risk factor research are large-scale genetic studies that bring us closer to understanding biological underpinnings of these disorders. Ideally, multi-disciplinary investigations that draw on the findings from these and other risk factor studies as well as genetics research will be forthcoming in the next decade. In the interim, studies such as these in this issue of the *Journal* can be useful in impelling programs that may help to reduce eating disorder risk. Other investigators have focused on developing prevention programs that target high-risk groups using internet-based programs or media literacy and have demonstrated promising findings.^{9–10} Based on these two *Journal* papers, prevention programs geared towards improving early feeding or eating interactions, and parent psychoeducation around healthy child weight ranges would also be useful first steps.

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