CPEU ARTICLE

Nature and Nurture: A contemporary view of eating disorders

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Reprinted with permission from SCAN’s Pulse, Fall 2009, Vol 28, No 4, pp 5-7, official publication of Sports, Cardiovascular, and Wellness Nutrition (SCAN), American Dietetic Association, Chicago, IL.

Learning Objectives—After you have read this article, you will be able to:

1. Describe and recognize gene-environment correlations as they relate to the development of eating disorders.
2. Discuss and give examples of the four groups of factors that influence eating disorders.
3. Develop clinical strategies that incorporate current research on genetic and sociocultural risk factors for eating disorders.

For decades, eating disorders were viewed as disorders of choice—the belief prevailed that patients somehow chose emaciation, dehydration, osteoporosis, and ultimately death. Research in the past decade has ignited an appreciation of the role genetic factors play in determining risk for eating disorders. Patients and families are aware of this research and often seek answers from providers. Even if we are not geneticists, a basic understanding of the genetic research on eating disorders can help us translate this knowledge for our patients and inform our clinical practice. Accurate interpretations of research on environmental and genetic risk factors empower patients and families.

The significant role of genetic factors in the development of eating disorders is becoming increasingly clear (1,2). Eating disorders and disordered eating run in families, although sporadic cases clearly occur (3-5). Family studies are unable to unravel the extent to which this familial pattern is due to genes or environment. However, twin studies can answer that question and reveal that liability to anorexia nervosa, bulimia nervosa, and binge eating disorders are significantly influenced by genetic factors (6). A number of molecular genetic investigations designed to identify and study specific genes that influence eating disorders risk—including association studies, linkage studies, and emerging genome-wide association studies—have been conducted or are under way (7,8).

Genes and environment: Not just one or the other

Genetic research does not discount the role of environment in development of eating disorders (9-11). Sociocultural factors, such as the pursuit of an unrealistic thin ideal, promote the disordered eating and body dissatisfaction that presage eating disorders (12). Given the fact that virtually all women are exposed to these sociocultural influences yet only a small proportion develop clinical eating disorders, it is clear that we need elucidation of the factors that make individuals vulnerable to the lure of the “thin is ideal” message. Genetic research might indeed be the key to helping us unlock such mysteries as why some individuals are more likely than others to fall prey to environmental triggers such as restrictive dieting.

As an important first step in understanding what makes an individual prone to developing an eating disorder, we need to move away from explanations that are either genetic or environmental. Instead, we must appreciate the various ways in which genes and environment can interact to influence risk of eating disorders.

Passive, active, and other gene-environment correlations

One way in which genes and environment can interact is reflected in passive gene-environment correlations. This refers to the fact that the same people who give us our genes (our biological parents) also create our environments (unless we are adopted). For example, we know that a high intelligence quotient (IQ) is heritable, but intelligent parents do more than simply pass down their smart genes—they also create enriched environments for their children. This is called the “double advantage.”

Unfortunately, double disadvantages also can occur. In disordered eating, this might refer to a parents who not only pass down genes that influence liability to eating disorders but also model eating disordered behaviors and attitudes to their children if they themselves are struggling with disordered eating (e.g., restriction, compulsive exercise, body dissatisfaction, drive for thinness). Children in these circumstances may receive a “double-dose” of eating disorder risk as a result of both genetic and environmental exposures.

Active gene-environment correlation refers to situations in which an individual with a genetic vulnerability to an eating disorder actively seeks out environments that emphasize appearance and thinness, such as gymnastics or ballet (2). The prevalence of disordered eating among ballet companies (13) may not only be due to the influence of ballet on disordered eating, but also may reflect selection of that high-risk environment by individuals prone to eating disorders. Our genes influence the choice of environments we make in life.

We also need to consider gene x environment (G x E) interactions that occur when an individual’s genotype influences his/her vulnerability (i.e., response to environmental risk factors) (14). For example, the influence of a weight-preoccupied coach on the eating behaviors of the athletes he or she coaches is likely to vary as a function of each athlete’s genotype. Ultimately, genetic research may prove to be the key to elucidating environmental risk factors for eating disorders and helping us understand more about specific risks factors and populations—i.e., which factors place which individuals at risk for developing which eating disordered behaviors.

A plausible scenario: How genes might influence eating disorders

A question commonly posed by clinicians, families, and patients is “How do genes work in influencing risk for eating disorders?” People may mistakenly believe there is one gene for anorexia nervosa or one gene for bulimia nervosa and if you have it, you get the illness. Mothers sometimes ask if their babies can be tested in utero for eating disorders. In reality, eating disorders are complex traits—influenced by many vulnerability genes and environmental factors. There is no genetic test for eating disorders. The genetics of eating disorders are probabilistic, not deterministic.
Factors influencing eating disorders can be grouped into four types: genetic risk factors, genetic protective factors, environmental risk factors, and environmental protective factors. An individual’s actual risk for developing eating disorders depends on her or his “dose” of all of these factors. In all likelihood, timing also is an influencer—the probability are developmental windows of risk and protection that researchers have not yet clarified.

To illustrate the influence of these various types of risks factors, consider the hypothetical case of “Anna,” aged 16. Her aunt had anorexia nervosa and presumably Anna inherited some genetic vulnerability factors for anorexia nervosa (genetic risk factors). Her father was a tall, naturally thin jumper and it appeared from birth and throughout childhood and adolescence that Anna inherited his tendency towards constitutional thinness (protective genetic factors). At the time when her peers were going on diets, Anna seemed to be buffered against this behavior because she never had to limit calories to maintain her natural slim physique.

In high school, Anna started running track and had a coach who encouraged everyone to lose weight to improve performance—in fact, at one point he tried to put the whole team on a diet (environmental risk factor). Anna tried the diet for one day but got so hungry she stopped following the coach’s instructions. Her family regularly ate breakfast and dinner together and provided an open environment to discuss the unrealistic pressures that the coach put on the athletes (protective environmental factors). In Anna’s case, although she may have had some elevated genetic risk, she was also fortunate to have both genetic and environmental buffers. For a different athlete who had a less buffering risk profile, the team diet might have become the trigger for a descent into anorexia nervosa.

Clinical implications
Clinicians can put current information about risk factors to use in multiple ways. First, we can educate parents about genetic and environmental factors that influence eating disorders. A clear and sensitive explanation can educate parents about genetic and risk factors to use in multiple ways. First, we can put current information about genetic and environmental factors to use in multiple ways. At the time when Anna’s peers were going on diets, Anna seemed to be buffered against this behavior because she never had to limit calories to maintain her natural slim physique.

In summary, although genetic research has not yet progressed to a point where it can be used directly to develop treatments or prevention interventions, we can continue to develop interventions aimed at promoting buffering rather than predisposing environments. Given the current state of knowledge in the fields of genetics and disordered eating, such interventions remain the best approach to reducing risks for these pernicious and potentially lethal disorders.

REFERENCES:

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