Genetics of Eating Disorders

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Abstract

Eating disorders are serious psychiatric illnesses influenced by interacting genetic, biological, psychological, behavioural, and social factors. Genetic variation can increase susceptibility, but it does not determine that a person will develop anorexia nervosa, bulimia nervosa, binge-eating disorder, avoidant/restrictive food intake disorder, or another eating disorder. No single “eating-disorder gene” explains these conditions.

Family and twin studies demonstrate substantial heritability, while genome-wide association studies identify many common variants with very small individual effects. Anorexia nervosa has genetic relationships with psychiatric, metabolic, and anthropometric traits. Emerging genomic research also demonstrates that binge-eating behaviour and anorexia nervosa have both shared and distinct biological patterns. These findings support a biopsychosocial model rather than explanations based on vanity, weak willpower, family blame, or a simple desire to be thin (National Institute of Mental Health [NIMH], 2026).

Introduction

Eating disorders affect people of all ages, sexes, genders, racial and ethnic groups, body sizes, sexual orientations, and socioeconomic backgrounds. They can cause malnutrition, cardiovascular complications, gastrointestinal problems, endocrine and bone changes, metabolic disturbance, suicide risk, and death. People may be underweight, average weight, or higher weight; appearance alone cannot establish whether someone has an eating disorder.

The exact causes are not fully understood. Current research examines how inherited genetic variation interacts with brain development, appetite and reward systems, metabolism, temperament, stress, puberty, dieting, trauma, social environment, weight stigma, and cultural pressures. Genes influence probabilities, not destiny. A person with elevated genetic liability may never develop an eating disorder, while someone without a known family history may become ill (NIMH, 2026).

Eating Disorders as Complex Traits

Eating disorders are complex traits. Unlike disorders caused primarily by a single rare mutation, common eating disorders generally reflect the combined effects of many variants and many non-genetic factors. Each common genetic variant normally contributes only a small amount of risk.

Researchers therefore use large samples to compare patterns across the genome. A genome-wide association study does not identify a direct cause in an individual. It detects statistical associations between variants and a diagnosis or behaviour. Findings must be replicated, studied in diverse populations, and connected with functional biological evidence before they can influence clinical practice.

A 2025 review reported heritability estimates broadly ranging from approximately 48% to 74% across eating-disorder phenotypes, while emphasising that estimates vary by disorder, population, design, diagnostic method, and environmental context (Suresh Kumar et al., 2025). Heritability describes variation within a population; it does not mean that a stated percentage of one person's illness was "caused by genes."

Family and Twin Research

Relatives of people with eating disorders have higher average rates of eating disorders and related traits than the general population. This clustering may reflect shared genes, shared environment, modelling, family responses to illness, or combinations of these factors.

Twin studies compare similarity between identical twins, who share nearly all inherited DNA sequence, and fraternal twins, who share on average about half of segregating genetic variation. Greater similarity among identical twins supports genetic influence, but identical twins also may experience more similar environments. Twin research estimates population-level genetic and environmental contributions; it cannot identify one person's cause.

Older statements that a relative is exactly seven to twelve times more likely to develop any eating disorder should be interpreted cautiously because risk differs by diagnosis and study. Family history is clinically relevant, but it is not a prediction that illness will occur.

Genome-Wide Findings in Anorexia Nervosa

Anorexia nervosa has received the greatest genomic research attention. Genome-wide association studies have identified replicated risk loci and genetic correlations with conditions and traits including obsessive-compulsive disorder, depression, anxiety-related phenotypes, educational attainment, physical activity, body mass index, insulin-related measures, and lipid metabolism.

These findings led researchers to describe anorexia nervosa as a “metabo-psychiatric” illness: psychiatric and metabolic biology may both contribute to vulnerability and persistence. This does not mean that metabolism alone causes restrictive eating or that genomic findings are ready for individual diagnosis. The identified variants explain only part of liability, and the pathways between genetic association, physiology, behaviour, and illness remain under investigation.

A 2023 genome-wide analysis found additional anorexia nervosa risk loci and extensive genetic overlap with psychiatric and related traits, reinforcing the view that the disorder cannot be understood through body-image pressure alone (Bang et al., 2023).

Genetics of Binge Eating, Bulimia Nervosa, and Other Disorders

Genetic research historically focused too narrowly on anorexia nervosa and participants of European ancestry. This limited understanding of bulimia nervosa, binge-eating disorder, ARFID, purging disorder, and eating disorders affecting men, gender-diverse people, racialised populations, and people at higher body weights.

A large genomic analysis published in 2025 examined binge-eating behaviour alongside anorexia nervosa. It identified six loci associated with binge-eating behaviour and eight with anorexia nervosa. Both phenotypes showed positive genetic correlations with several psychiatric disorders, but they had opposite genetic relationships with some anthropometric traits. Most genetic signal was not simply explained by body mass index (Hübel et al., 2025).

This work shows that “eating-disorder genetics” is not one uniform pathway. Restriction, binge eating, purging, sensory avoidance, fear of adverse consequences, appetite, reward, impulse control, and metabolic regulation may involve partly overlapping and partly distinct biology.

Candidate Genes and the Risk of Overstatement

Early studies frequently tested individual “candidate genes” selected because they were thought to influence serotonin, dopamine, oestrogen, appetite, stress, or personality. Many reported associations failed to replicate because samples were small, statistical power was limited, populations differed, and publication bias favoured positive results.

Claims that mutations in ESRRA or HDAC4 give an individual an 85% or 90% chance of developing an eating disorder are not appropriate for general clinical use. Those findings came from rare variants studied in a small number of families and do not establish common predictive tests. Current research favours adequately powered genome-wide approaches, sequencing, functional genomics, and replication.

Genes and pathways involving appetite, energy balance, reward, neurodevelopment, and stress remain important research targets. However, listing molecules such as BDNF, ghrelin, or AGRP does not prove that one pathway causes a patient's disorder. Molecular findings should be presented as hypotheses and population associations, not deterministic explanations (Suresh Kumar et al., 2025).

Psychiatric and Metabolic Biology

Eating disorders involve systems that regulate hunger, satiety, reward, habit, emotion, cognition, interoception, and energy balance. These systems change during starvation, binge eating, purging, stress, and recovery. Researchers must distinguish biological vulnerabilities that existed before illness from consequences of malnutrition or disordered behaviour.

For example, starvation can alter hormones, reward processing, attention, anxiety, gastrointestinal function, and brain structure. These effects can reinforce restriction and make recovery difficult even when the original trigger is no longer present. Binge eating and compensatory behaviours may likewise alter reward and control circuits. Biology is therefore both a risk factor and a consequence of illness.

A 2024 review concluded that anorexia nervosa, bulimia nervosa, binge-eating disorder, and ARFID are heritable and likely differ in their relationships with metabolic and anthropometric traits. It also emphasised the need to translate genomic variants into specific genes, pathways, tissues, and mechanisms before findings become medically actionable (Bulik et al., 2024).

Temperament and Personality

Traits such as perfectionism, harm avoidance, anxiety, cognitive rigidity, compulsivity, impulsivity, reward sensitivity, emotional dysregulation, or negative urgency may be associated with particular eating-disorder presentations. Some are partly heritable and may appear before illness. Others may intensify during malnutrition or chronic stress.

These traits are neither necessary nor sufficient for diagnosis. Many perfectionistic, anxious, impulsive, or emotionally sensitive people never develop eating disorders. The same trait may create risk in one environment and protection in another. For example, persistence may support rigid restriction during illness but can also support engagement with recovery.

Gene–Environment Interplay

Genes and environment are not separate competing explanations. Genetic differences may influence sensitivity to dieting, puberty, stress, trauma, exercise, food insecurity, medication, illness, social comparison, or weight-related teasing. Environmental exposure can also differ partly because

individuals select or evoke environments associated with their traits.

Potential environmental and developmental contributors include:

- dieting and negative energy balance;
- puberty and hormonal change;
- weight stigma and bullying;
- trauma, abuse, and chronic stress;
- food insecurity or disrupted feeding;
- sports or occupations that emphasise weight, leanness, or appearance;
- social-media and peer comparison;
- family and cultural messages concerning food and bodies;
- co-occurring anxiety, depression, obsessive-compulsive symptoms, autism, or ADHD; and
- medical conditions affecting appetite, swallowing, gastrointestinal symptoms, or sensory processing.

None should be used to blame patients or families. A trigger is not the same as the complete cause.

Epigenetics

Epigenetics concerns molecular processes that influence gene activity without changing the underlying DNA sequence. DNA methylation, histone modification, and other regulatory mechanisms can respond to nutrition, hormones, stress, medication, age, and illness.

Studies have reported epigenetic differences in people with eating disorders, especially anorexia nervosa. Interpretation is difficult because many studies are cross-sectional and use blood rather than disease-relevant tissues. It may be impossible to determine whether a difference existed before illness, resulted from starvation or treatment, reflected smoking or medication, or arose from another factor.

Epigenetic findings do not mean that an eating disorder permanently “switches genes on or off,” nor do they establish that trauma or parental experience is biologically inherited in a simple manner. Longitudinal, adequately powered, and replicated studies are needed.

Genetic Correlations and Co-Occurring Conditions

Eating disorders commonly co-occur with anxiety, depression, obsessive-compulsive disorder, substance-use disorders, post-traumatic stress, autism, and attention-deficit/hyperactivity disorder. Genetic correlation can indicate that some inherited variants influence liability to more than one trait.

A genetic correlation does not prove that one disorder causes another. It also does not mean that

every person with an eating disorder will have the correlated condition. Shared biology may operate through emotion regulation, compulsivity, reward, metabolism, neurodevelopment, or other pathways.

Anorexia nervosa and binge-eating phenotypes may share psychiatric genetic liability while differing in their relationships with body mass and metabolic traits. This helps explain why one biological model cannot be applied to all eating disorders (Hübel et al., 2025).

Diversity and Limitations of Genetic Research

Many genomic studies have overrepresented white women of European ancestry. This creates several problems:

- risk loci may not transfer equally across ancestral populations;
- polygenic scores are less accurate in underrepresented groups;
- male and gender-diverse presentations may be missed;
- diagnostic bias may affect who enters studies;
- people with atypical anorexia or higher body weights may be excluded; and
- cultural and socioeconomic exposures may be inadequately measured.

Researchers have called for broader recruitment, community partnership, harmonised phenotyping, better measurement of social environment, and studies that include diagnoses beyond anorexia nervosa. Replacing stereotypes with inclusive science is necessary for findings to benefit all patients (Munn-Chernoff et al., 2022).

Can Genetic Testing Predict an Eating Disorder?

At present, routine clinical genetic testing cannot predict who will develop a common eating disorder or select a proven personalised treatment. Polygenic scores aggregate effects across many variants, but current scores explain only a limited proportion of liability and are sensitive to ancestry and study design.

Direct-to-consumer tests should not be used to diagnose eating disorders, estimate a child's future illness with confidence, or guide treatment without clinical evidence. A family history can alert clinicians to elevated vulnerability, but assessment must focus on current symptoms, medical stability, behaviours, psychological state, and functioning.

Implications for Treatment and Prevention

Genetic research has important implications even before a genetic test is clinically useful:

- Eating disorders are not choices or moral failures.
- Parents and patients should not be blamed for causing the illness.
- Symptoms deserve early, evidence-based treatment regardless of body size.
- Restoring adequate nutrition is biologically important, not merely behavioural.
- Co-occurring psychiatric and medical conditions should be assessed.
- Treatment may need to address anxiety, compulsivity, sensory sensitivity, reward, impulsivity, trauma, or metabolism.
- Families can be valuable allies in care.

Treatment may include medical monitoring, nutritional rehabilitation, psychotherapy, family-based approaches, and medication for selected symptoms or co-occurring conditions. No current treatment should be withheld because a person lacks a family history or a genetic marker. Early detection and treatment improve the possibility of recovery (NIMH, 2026).

Protective Factors

Genetic liability does not make prevention irrelevant. Protective environments can reduce exposure to triggers, support early recognition, and improve recovery. Helpful approaches include:

- adequate and regular nutrition;
- reducing weight stigma and appearance-based bullying;
- avoiding unnecessary restrictive dieting;
- supportive responses to puberty and body change;
- responsible coaching in sport and dance;
- media literacy;
- access to culturally competent healthcare;
- family and peer support;
- early assessment of emerging symptoms; and
- treatment that is accessible regardless of weight, gender, or background.

Conclusion

Genetic evidence has transformed understanding of eating disorders. Family and twin studies show substantial heritability, and genomic studies now identify risk loci, genetic correlations, and biological differences among anorexia nervosa, binge-eating behaviour, and other phenotypes. The evidence rejects simplistic explanations based on appearance, attention seeking, or lack of self-control.

At the same time, genetics is not destiny. Eating disorders arise through complex interaction among many variants and biological, psychological, developmental, and social conditions. Most identified variants have very small effects, current polygenic scores are not diagnostic, and research samples remain insufficiently diverse.

The most responsible conclusion is therefore biopsychosocial: eating disorders are biologically influenced, environmentally responsive, serious, and treatable. Genetic research should reduce stigma, improve mechanisms and classification, and eventually contribute to prevention and personalised care—but it should not create fatalism, blame, or premature testing (Bulik et al., 2024; NIMH, 2026).

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