Longitudinal Analyses Suggest Genomic and Psychological Origins of Disordered Eating and Comorbidities

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Twenty million people in the European Union are estimated to have an eating disorder, costing approximately €1 trillion (>1 trillion US dollars) per year.1 Eating disorders are severe mental health illnesses that commonly emerge in adolescence. They are debilitating for the patients and their loved ones, with symptoms often becoming more embedded and complex over time. Anorexia nervosa is the eating disorder with the highest mortality of all psychiatric disorders. After decades of research, the underlying risk factors for eating disorders remain poorly understood.

More recently, large international collaboration efforts have aimed to elucidate the genetic underpinnings of eating disorders, in particular anorexia nervosa. Findings support the notion that, on a genomic level, anorexia nervosa shares a complex etiology with a wide array of phenotypes, ranging from metabolic functions to personality constructs such as neuroticism.2 In light of these findings, emerging research has examined the associations between genetic risk scores and symptoms of eating disorders. For example, previous research has reported associations between genetic risk scores for body mass index and binge eating, fasting, and other weight-loss behaviors, supporting the idea of a shared genetic etiology, which had previously only been explored on a phenotypic level.3,4

The study by Robinson et al5 replicates previous findings but also moves forward by considering a wide range of genetic, behavioral, and psychological risk factors across adolescence. The authors analyzed data from the IMAGEN study, a population cohort that includes longitudinal measures of eating disorder symptoms, as well as a plethora of other psychiatric and psychological constructs, in addition to genomics.

The main findings by Robinson et al5 suggested that an earlier higher body mass index was associated with later fasting, whereas neuroticism, alcohol and drug use, and self-harm was associated with later binge eating. Purging in adolescence was found to be associated with earlier low agreeableness as well as conduct problems and alcohol and drug use. On a genetic level, their results mirrored the phenotypic findings. Greater polygenic risk scores associated with higher body mass index were also associated with increased fasting, whereas the polygenic risk score for neuroticism was associated with higher risk of binge eating, and the polygenic score for attention-deficit/hyperactivity disorder was associated with greater purging behavior. These findings stress the complex and distinct etiologic overlaps between the different eating disorder symptoms. However, this study went a step further by considering the association between those eating disorder symptoms in early adolescence and later health outcomes, proposing that dieting and purging behaviors at 14 years of age were associated with depression and anxiety symptoms as well as with increased emotional problems and smoking at 19 years. This study highlights the value of analyzing data from longitudinal cohort studies with measures across psychological and biological domains. This approach enables researchers to hypothesize and test complex models, which are more likely to approximate the true origins of onset of common mental health illnesses, such as eating disorders.

Overall, these findings have important implications for clinical practice. Given the interwoven nature of both childhood psychiatric disorders, namely attention-deficit/hyperactivity disorder, and adolescent affective disorders with eating disorder symptoms, greater clinical awareness and prompter recognition of psychiatric comorbidities by primary care teams are essential. Identifying individuals who are at risk of developing eating disorders will create opportunities for earlier
intervention, potentially preventing the onset of eating disorders or improving their clinical course. This can only be achieved by effective multidisciplinary efforts between primary care and psychiatric specialisms, in close collaboration with patients and their loved ones.6 

Adolescence is a transitional stage in development with complex psychosocial, neurobiological, and brain circuitry changes. Eating disorders often interrupt normative adolescent developmental processes and, together with the potential adverse effects of starvation on the brain, may contribute to young people missing important opportunities to develop the skills necessary to become autonomous and self-reliant individuals.7 Failure to recognize and treat eating disorders can therefore have devastating consequences. Such failures contribute to the chronicity and severity of the illness as well as to the development of comorbidities, such as anxiety and depression.

REFERENCES