WHAT IS THE LINK BETWEEN DISORDERED EATING AND BORDERLINE PERSONALITY DISORDER ACROSS ADOLESCENCE?

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Introduction. Eating disorders are serious mental illnesses that afflict approximately 2% of the population. Approximately half of individuals with an eating disorder present with a comorbid personality disorder, most commonly, borderline personality disorder (BPD), another serious mental illness. Both disorders increase the risk of further morbidity and early mortality, but there is a lack of robust and longitudinal research examining the link between these symptoms. The aims of this study were to examine trajectories of disordered eating behaviour and BPD features over time, identify patterns of comorbidity, temporal precedence, unique and shared childhood risk factors (sociodemographic, interpersonal, clinical), and the population attributable fractions (PAFs) associated with each modifiable risk factor.

Method. We used data across five-years of adolescence (ages 14-18) from the Canadian McMaster Teen Study (N=544; 56% girls; 74% white). Clinically significant disordered eating behaviour was measured using the Short Screen for Eating Disorders (12-items rated on a 5-point scale). BPD was measured using The Borderline Personality Features Scale for Children (24-items measured on a 5-point scale). We assessed a range of risk factors across sociodemographic (sex, race/ethnicity, household income, parent education, sexual orientation), interpersonal (physical abuse, sexual abuse, bullying victimization, bullying perpetration), and clinical (anxiety, depression, somatization, hyperactivity, impulsivity) domains. All risk factors were transformed into dichotomous exposure variables. We used a semi-parametric group-based modelling approach to identify the number and shape of trajectories and estimate conditional probabilities. Unique and shared risk factors were examined using multinomial logistic regressions.

Results. Most adolescents followed a trajectory of low disordered eating (*n*=418, 77%), followed by a moderate symptom trajectory (*n*=100, 18%), and a high symptom trajectory (*n*=26, 5%). Regarding BPD symptoms, most followed a trajectory of moderate symptoms (*n*=225, 41%), followed by a low symptom trajectory (*n*=204, 38%), and a high symptom trajectory (*n*=114, 21%). Approximately one quarter of the sample had comorbid disordered eating and BPD symptoms. Belonging to the high disordered eating trajectory was a stronger indicator of belonging to the high BPD features trajectory than was the reverse. Girls were at the highest risk of being in the high disordered eating symptom group (OR=21.9), while LGBTQ youth were at the highest risk of being in the high BPD features group (OR=20.2). Although there were no unique risk factors for disordered eating, bullying perpetration and hyperactivity were unique risk factors for BPD features. The PAFs indicated that eliminating bullying victimization would reduce clinically significant cases of disordered eating by 66% and cases of borderline personality by 37%. Bullying victimization contributed the largest PAFs of all the modifiable risk factors.

Conclusion. This is the first study to unravel the links between disordered eating behaviour and BPD features. Determining that disordered eating most often comes first in the temporal ordering of symptoms has important implications for treatment in comorbid cases. As the current results indicate that disordered eating increases the chance of developing further psychiatric problems, treatments that target disordered eating behaviour may inhibit the development of personality psychopathology among some youth. As bullying victimization had the largest PAFs (larger than sexual and physical maltreatment), while bullying perpetration was a unique risk factor for high levels of BPD symptoms, reducing violence, especially childhood bullying, remains a public health priority.