



## Regular Article

# Trajectories, comorbidity, and risk factors for adolescent disordered eating and borderline personality disorder features

Kirsty S. Lee<sup>1</sup>  and Tracy Vaillancourt<sup>2,3</sup> 

<sup>1</sup>Department of Psychology, Faculty of Science, University of Warwick, Coventry, UK, <sup>2</sup>Counselling Psychology, Faculty of Education, University of Ottawa, Ottawa, ON, Canada and <sup>3</sup>School of Psychology, Faculty of Social Sciences, University of Ottawa, Ottawa, ON, Canada

### Abstract

**Purpose:** Borderline personality disorder (BPD) and eating disorders are highly comorbid, but the shared course of symptoms and associated risks remain poorly understood. The aim of this study was to examine joint symptom trajectories, temporal precedence, risk factors, and population attributable fractions (PAFs) in a community sample of adolescents, using a developmental psychopathology and psychosocial framework. **Methods:** Across five years (age 14–18 years), adolescents ( $n = 544$ , 56% girls) reported on BPD features and disordered eating behavior. Sociodemographic, interpersonal, and clinical risks were assessed in childhood (age 10–13 years). We used a person-centered approach to examine latent class growth analyses, joint trajectory models, and calculated PAFs. **Results:** Three-class solutions were found for both disordered eating and BPD features (low, moderate, high), creating nine joint trajectories. High levels of disordered eating were a stronger indicator of high levels of BPD features than was the reverse. Girls and LGBTQ+ youth were most likely to be in a high symptom trajectory. Bullying perpetration and clinical hyperactivity were unique risks for BPD features. Bullying victimization contributed the largest PAF to disordered eating and BPD features. **Conclusion:** We identified several novel and clinically relevant findings related to temporality, risks, screening, and the treatment of adolescent eating problems and BPD.

**Keywords:** Adolescents; borderline personality; disordered eating; longitudinal; risk factors

(Received 24 January 2023; revised 21 April 2023; accepted 7 June 2023)

### Introduction

Eating disorders (disturbances in eating, weight, and body image) are serious psychiatric disorders with a lifetime prevalence of 2% globally (Swanson et al., 2011). Clinical and subclinical symptoms of eating disorders can cause significant distress to individuals and their families, substantially impair interpersonal relationships, and increase the risk of medical morbidities, psychiatric morbidities, and early mortality, due to physical health complications or suicide (Demmler et al., 2019; Swanson et al., 2011). Eating disorders are often studied as discreet conditions, such as anorexia nervosa, bulimia nervosa, and binge eating disorder, but as diagnostic instability, non-specific eating disorders (Other Specified Feeding and Eating Disorders), and subclinical symptoms are common (Micali et al., 2015; Schaumberg et al., 2018; Zanarini et al., 2010), studies using symptoms-based rather than diagnostic-based approaches are needed. Although most disordered eating onsets and peaks during adolescence (Demmler et al., 2019; Micali et al., 2015), the course of comorbidity and associated risk factors remain poorly understood (van Furth et al., 2016).

Approximately half of individuals with an eating disorder present with a comorbid personality disorder, most commonly (22%), borderline personality disorder (BPD) (Martinussen et al.,

2017). BPD is another serious psychiatric disorder, demarcated by emotional, behavioral, and interpersonal instability, and like eating disorders, is associated with premature mortality (Temes et al., 2019). Prior studies that investigated the overlap between eating disorders and BPD were primarily based on adult women, clinical samples, and cross-sectional or short-term prospective data (Martinussen et al., 2017). To date, no study has investigated co-occurring trajectories of disordered eating and BPD or delineated temporality at the time of symptom onset. Unraveling the joint trajectories of disordered eating and BPD symptoms will have implications for understanding the developmental and temporal patterns of these psychopathologies at the intraindividual level. Determining temporal precedence has been identified as a clinical priority (van Furth et al., 2016), largely because knowing whether symptoms of eating disorder or personality disturbance come first has consequences for the timing and ordering of treatment. Intuitively, it may seem reasonable to predict that personality disturbance precedes other psychiatric problems, but there is evidence that poor diet quality and disordered eating often precede other mental health problems (Lee & Vaillancourt, 2018; Marx et al., 2017; Micali et al., 2015).

Considering the increased risk of early mortality and morbidity associated with eating disorders and BPD and that a high-risk dual trajectory would be found in the data, we were interested in identifying the comparative contribution of antecedent risk factors. Presently, a major limitation of research on risk factors is the lack of disorder-specific etiologies. This is because most risk factors and

**Corresponding author:** Tracy Vaillancourt; Email: [tracy.vaillancourt@uottawa.ca](mailto:tracy.vaillancourt@uottawa.ca)

**Cite this article:** Lee, K. S., & Vaillancourt, T. (2023). Trajectories, comorbidity, and risk factors for adolescent disordered eating and borderline personality disorder features. *Development and Psychopathology*, 1–12, <https://doi.org/10.1017/S0954579423000792>



outcomes follow the principles of equifinality and multifinality, in that many risk factors can lead to the same outcome, and one risk factor can lead to many outcomes (Cicchetti & Rogosch, 1996). Much research has examined contemporaneous risk factors for disordered eating and BPD features, and the selected risk factors in the current study build on this prior work and fit within a psychosocial framework. We focused on risk factors available in the data set that were non-age-overlapping and have shown the largest effects previously. For each modifiable risk factor, small reductions in their occurrence could significantly reduce the incidence of eating disorders and BPD. Therefore, we examined population attributable fractions (PAF), an epidemiological tool that quantifies the impact of risk exposure and clinical burden in populations (Mansournia & Altman, 2018; Williamson, 2010).

Our selected risk factors were broadly characterized as sociodemographic (i.e., sex, race/ethnicity, sexual orientation, household income, parent education), interpersonal (i.e., physical abuse, sexual abuse, bullying victimization, bullying perpetration), and clinical (i.e., anxiety, depression, somatization, hyperactivity, impulsivity). In previous umbrella reviews and meta-analyses, interpersonal factors, especially childhood physical abuse, sexual abuse, and peer bullying victimization have contributed the largest risks to eating disorders and BPD (Lereya, Copeland, Costello, et al., 2015; Solmi et al., 2021; Winsper et al., 2016). Clinical factors, such as comorbid mood and anxiety disorders, and neurodevelopmental conditions, such as attention deficit hyperactivity disorder (ADHD) have also been strongly implicated in the development of disordered eating and BPD features (2019a; Haltigan & Vaillancourt, 2016a; Jacobi et al., 2004, Lee & Vaillancourt, 2018; Stepp et al., 2016; Vaillancourt et al., 2014; Winsper et al., 2016, 2017).

In terms of sociodemographic risks, girls are more likely than boys to be diagnosed with an ED or BPD. Girls typically feel more pressure to be thin than boys, and attempt to lose weight by engaging in restrained eating (Buchanan et al., 2013; McCabe et al., 2002) and extreme weight loss behavior (McCabe & Ricciardelli, 2005), while boys typically feel more pressure to be muscular than girls (McCabe et al., 2002). Sociocultural influences (e.g., media images, family and peer pressure, bullying victimization and perpetration) may drive expectations about body shape or size differently for boys and girls (Helfert & Warschburger, 2011; Lee et al., 2017; Knauss et al., 2007). However, some research suggests that adolescent girls and boys may be more similar in their disordered eating behavior than previously thought (Latzer et al., 2012), as there appear to be few sex differences in meal skipping (Brown et al., 2016) and laxative abuse (Buchanan et al., 2013). Similarly, research has indicated that girls score higher on measures of BPD features than boys (Haltigan & Vaillancourt, 2016a; Vaillancourt et al., 2014), but in adulthood, the clinical presentation of BPD among women and men is more similar than different (Johnson et al., 2003). Sex assigned at birth may thus be a key predictor of trajectory membership among adolescents. Other demographics, such as race/ethnicity and socioeconomic factors, have been inconsistently associated with ED and BPD (Meaney et al., 2016; Stepp et al., 2016; Wildes et al., 2001), while research suggests that individuals with an ED or BPD features are less likely to be heterosexual (Castellini et al., 2016; Reich & Zanarini, 2008; Reuter et al., 2016).

The aims of the present study were to examine: (a) the course of disordered eating and BPD features, (b) joint trajectories and patterns of co-occurrence, (c) temporal precedence, (d) unique and shared childhood risk factors, and (e) the burden of probable

clinical cases attributable to each risk factor, using data from a community-based cohort study that spanned five years of adolescence (age 14–18 years). Based on prior research, we expected that there would be a high degree of co-occurrence between disordered eating and BPD symptoms (Martinussen et al., 2017), that disordered eating would be a stronger precursor to BPD features than the reverse (Lee & Vaillancourt, 2018), and that all interpersonal and clinical risk factors would be significant predictors of disordered eating and BPD features (Lee & Vaillancourt, 2019b; Lereya, Copeland, Costello, et al., 2015), but that more girls than boys would be in the high symptom trajectories (Buchanan et al., 2013; McCabe et al., 2002; Vaillancourt et al., 2014).

## Method

### Participants

Data were drawn from the Canadian *McMaster Teen Study*, a longitudinal study examining bullying, mental health, and academic achievement. In 2008, a random sample of children ( $N = 875$ ) were recruited from Grade 5 classrooms ( $M_{\text{age}} = 10.91$ ,  $SD = 0.36$ ) across 51 schools in southern Ontario. Participants have been followed up annually until age 26, with 80% ( $n = 703$ ) of children participating in the longitudinal arm of the study (Vaillancourt et al., 2013). The present study included data from 544 adolescents (77%) who completed measures of disordered eating and BPD features between the ages 14–18 years. The analytic sample was restricted to participants with data on disordered eating and BPD features at one or more time point.<sup>1</sup> The sample consisted of 56% girls, were majority white (76.3%; African/West Indian = 4%, South Asian = 3.5%, Indigenous = 2.1%, Middle Eastern = 1.6%, Asian = 1.4%, South/Latin American = 1%, other = 1.9%, and missing = 8.2%), with a medium income (\$70,000–\$80,000). Almost two-thirds of the sample had parents with a Bachelor's degree. Most adolescents identified as heterosexual (86.4%; Lesbian/Gay = 1.6%, bisexual = 9%, other = 3.6%).

### Procedure

This study complies with the Helsinki Declaration of 1975, as revised in 2008. All procedures involving human participants were approved by the McMaster University Research Ethics Board (grades 5–8) and the University of Ottawa Research Ethics Board (grades 9–12). Each year, written parental consent and child assent were obtained. In Grade 5, data were collected during school hours using a paper/pencil survey. In subsequent years, adolescents completed a paper/pencil or electronic version of the questionnaire at home. Participation was compensated with a gift card that incrementally increased in value.

### Measures

#### Disordered eating behavior

The Short Screen for Eating Disorders (SSED) was used to assess disordered eating behavior (Miller & Boyle, 2009). SSED items reflect behavior identified in the DSM-IV for eating disorders and items were verified by a team of ED specialists, psychiatrists, psychologists, dietitians, pediatricians, and social workers (Miller & Boyle, 2009). The SSED exhibit 83–97% sensitivity-specificity in predicting cases versus non-cases among girls and boys. The scale

<sup>1</sup>Trajectories for participants with data at two or more time points were examined and the solutions were equivalent.

consists of 12 items rated on a 5-point scale (0 = never, 1 = a few times a month, 2 = once a week, 3 = 2–4 times every week, 4 = almost every day) and examines clinically significant behavior (e.g., “How often did you take diet pills?”; “How often did you vomit on purpose after eating?”). Items were averaged to create a composite score, with higher scores reflecting higher severity of disordered eating behavior. The measure had good reliability at every time point (min–max  $\alpha = 0.75\text{--}0.82$ ).

#### *Borderline personality features*

The Borderline Personality Features Scale for Children was used to examine self-reported BPD features from age 14 to 18 years (Crick et al., 2005). The scale consists of 24 items rated on a 5-point scale (0 = not at all true, 1 = hardly ever true, 2 = sometimes true, 3 = often true, and 4 = always true). A composite score was calculated at each time point by averaging the items, with higher scores indicating higher symptoms severity. Internal consistencies across time were excellent (min–max  $\alpha = 0.90\text{--}0.91$ ). Factorial invariance of the Borderline Personality Features Scale for Children has previously been established (Haltigan & Vaillancourt, 2016b).

#### *Risk factors*

All modifiable risk factors were transformed into dichotomous variables to calculate odds ratios, relative risks, and population attributable fractions.

**Sociodemographic factors.** Sex assigned at birth was self-reported (boy = 0, girl = 1) and verified with parent reports. Race/ethnicity was based on a combination of self and parent reports and was dichotomized into White and non-White due to the low prevalence of most non-White racial/ethnic backgrounds. Household income was assessed using an 8-point scale using increments of \$10,000. The median income for the province (Ontario) from which the students were recruited was \$70,910 and for the city was \$76,222 (<http://www.statscan.gc.ca>). The median income of our sample was \$70,000–\$80,000, which maps onto the city and province levels and represents a middle socioeconomic stratum in Canada, thus income was recoded as average ( $\geq \$70,000 = 0$ ) or below average ( $< \$70,000 = 1$ ). When children were aged 11, parents reported their own highest level of education using a five-point scale (e.g., did not complete high school = 1; graduate degree = 5), which was dichotomized into <Bachelor’s degree = 0 or  $\geq$ Bachelor’s degree = 1 as above average incomes ( $\geq \$70,000$ ) were clustered among those with university degrees. Adolescents were contacted at age 19–20 and reported on their sexual orientation. As there were low prevalence rates for each LGBTQ+ orientation, sexual orientation was dichotomized into heterosexual or LGBTQ+.

**Interpersonal factors.** Childhood bullying victimization and perpetration was measured from age 11 to 13 using an adapted version of the Olweus Bully/Victim questionnaire (Vaillancourt et al., 2010), which consists of one general item “Since the start of the school year, how often have you been bullied at school?” and four specific items (i.e., physical, verbal, social, cyber victimization). Responses were on a 5-point scale (not at all = 0; many times a week = 4). The same items, with slight wording adaptations, were used to measure bullying perpetration. Children were classified as being exposed to bullying victimization or bullying perpetration if they responded that they had been victimized or had bullied others at least once a month at any time between ages 11 and 13 years. Child maltreatment was measured using the Childhood Experiences of Violence Questionnaire Short-Form (Walsh et al., 2008). Adolescents were contacted at age 19–20 and

retrospectively reported on childhood physical and sexual abuse using the stem “How many times before the age of 16 did an adult . . .” followed by 3 items related to physical abuse and 1-item on sexual abuse. Adolescents who endorsed any of the physical abuse items (no = 0, yes = 1) were classified as being exposed to physical abuse and adolescents who endorsed the sexual abuse item (no = 0, yes = 1) were classified as being exposed to sexual abuse.

**Clinical factors.** Most clinical symptoms were examined using the Behavior Assessment System for Children-Second Edition (BASC-2; Reynolds & Kamphaus, 2004). The BASC-2 is the gold standard measure for this age group and contains subscales on depression (12 items), generalized anxiety (10 items), hyperactivity (8 items), and somatization (7 items). Items were rated using a 5-point scale (never = 0; very often = 4) or a dichotomous response (false = 0, true = 2). Data on anxiety, depression, and hyperactivity were collected from age 11 and data on somatization were collected from age 13. Item raw scores were summed and transformed into standardized T-scores, with scores of 60 or above being clinically significant (clinical and subclinical symptom levels; Reynolds & Kamphaus, 2004). Children with T-scores  $\geq 60$  at any time between ages 11 and 13 years were classified as exhibiting clinically significant symptoms of anxiety, depression, somatization, or hyperactivity. Impulsivity was measured by parent-report from age 11 to 13 using the three impulsivity items from the Brief Child and Family Phone Interview (Cunningham et al., 2009). The Brief Child and Family Phone Interview has strong reliability and validity and been normed for parent samples. Items were measured on a 3-point scale (never = 0; often = 2), which were summed and transformed into z-scores. Children with z-scores  $\geq 1$  (equivalent to a T-score of 60) at any time between ages 11 and 13 years were classified as exhibiting clinically significant symptoms of impulsivity (Cunningham et al., 2009).

At age 19–20, the M.I.N.I. International Neuropsychiatric Interview (Sheehan et al., 1997) was administered to participants by a trained postgraduate interviewer to examine diagnosable eating disorders. Adolescents were included in the ED clinical proxy group if they met criteria for any eating disorder (Anorexia Nervosa, Bulimia Nervosa, or Binge Eating Disorder). As part of the clinical interview, the Borderline Symptom List (BSL) was also administered (Bohus et al., 2009). Mean score cut-points on the BSL can be transformed into symptom severity classifications, whereby scores  $> 1.5$  (high, very high, or extremely high symptoms) separate treatment-seeking BPD patients from clinical and healthy controls (Kleindienst et al., 2020). Adolescents were included in the BPD clinical proxy group if they scored  $> 1.5$  on the BSL.

#### *Analysis*

Missing data patterns between participants included and excluded from the core analytic sample, as well as descriptive data, were examined using a series of independent sample *t*-tests, chi-square tests ( $\chi^2$ ), and Pearson’s correlations in SPSS (IBM version 26). We compared adolescents retained in the sample with those who dropped on variables available at Time 1 (i.e., sex assigned at birth, race/ethnicity, household income, parent education, anxiety, depression, hyperactivity, impulsivity, bullying victimization, and bullying perpetration).

The analyses were performed in several steps that aligned with the research aims. To examine the independent trajectories of disordered eating and BPD features, semiparametric group-based

models were estimated in Mplus 8 (Muthén & Muthén, 2017) using latent class growth analysis with full information maximum likelihood estimation to handle missing data. Latent class growth analysis identifies heterogeneity within a sample and classifies individuals based on the probability that they belong to a given class (Nagin, 2005). We tested up to four-class solutions for disordered eating behavior and BPD features separately to identify the number and shape (e.g., linear, quadratic) of distinct trajectories. Evaluation of the best fitting models was based on the Bayesian information criterion (BIC), the Lo-Mendell-Rubin likelihood ratio test, the bootstrapped likelihood ratio test, entropy, and the identification of a conceptually meaningful set of classes (Nylund et al., 2007). We then used the growth parameters from the final trajectory models as the starting values for the joint trajectory model (Haltigan & Vaillancourt, 2014; Vaillancourt & Haltigan, 2018). Conditional probability analyses were run to examine the joint patterning of co-occurring symptoms and the temporal precedence of disordered eating and BPD trajectories. Conditional probability analyses are a standard method of examining temporal precedence in these models (Haltigan & Vaillancourt, 2014; Vaillancourt & Haltigan, 2018).

To examine unique and shared childhood risk factors, we used multinomial logistic regressions on joint class membership. Model 1 included each unadjusted (crude) risk factor. Models 2–4 were partially adjusted for sociodemographic, interpersonal, and clinical risk factors, respectively. To examine the burden of probable clinical cases, we calculated the adjusted population attributable fraction (aPAF) for each modifiable risk factor that remained statistically significant in the adjusted models (Williamson, 2010). Adjusted PAFs are calculated using the following formula:

$$\text{aPAF} = \text{PD}((\text{RR}_A - 1)/\text{RR}_A) \quad (1)$$

where PD is the prevalence of exposure among cases (i.e., exposed vs. not exposed) and  $\text{RR}_A$  is relative risk adjusted for confounders. Risk factors were therefore dichotomized into the same metric to compare  $\text{RR}_A$  and calculate PAFs. The highest symptom trajectory classes of disordered eating and BPD features were transformed into binary outcome variables and used as proxies of clinical cases. As a post hoc analysis, we examined whether the adolescent high-risk trajectory classes were adequate proxies for adult clinical cases using binary logistic regressions with 95% confidence intervals (CIs) to examine whether the high-risk trajectory classes predicted clinically diagnosable ED or BPD in the future.

## Results

### Missing and descriptive data

Little's test indicated that data were not Missing Completely at Random,  $\chi^2(375) = 506.709$ ,  $p < .001$ . Adolescents who dropped out had lower household income ( $t = -6.44$ ,  $df = 615$ ,  $p < .001$ ), lower parent education ( $t = 7.70$ ,  $df = 805$ ,  $p < .001$ ), were more likely to be non-White ( $\chi^2 = 7.23$ ,  $df = 1$ ,  $p = .007$ ), and had higher levels of anxiety ( $t = 2.705$ ,  $df = 794$ ,  $p = .007$ ), depression ( $t = 4.06$ ,  $df = 694$ ,  $p < .001$ ), hyperactivity ( $t = 2.94$ ,  $df = 718$ ,  $p = .003$ ), impulsivity ( $t = 4.33$ ,  $df = 636$ ,  $p < .001$ ), bullying victimization ( $t = 2.26$ ,  $df = 782$ ,  $p = .024$ ) and bullying perpetration ( $t = 2.83$ ,  $df = 621$ ,  $p = .005$ ) at Time 1. There were no sex differences in dropout ( $\chi^2 = 0.16$ ,  $df = 1$ ,  $p = 9.46$ ). Variables had an average of 9.3% missing data (min = 0, max = 20.2).

Mean rates of disordered eating behavior for the total sample gradually increased over time (Table 1), while mean rates of BPD features were stable over time. There were large within-time correlations between disordered eating behavior and BPD features across adolescence (age 14  $r = .57$ , age 15  $r = .56$ , age 16  $r = .60$ , age 17  $r = .55$ , and age 18  $r = .51$ , all  $ps < .001$ ). Of the total sample, 13.6% identified as LGBTQ+, 15.6% reported physical abuse, 8.4% reported sexual abuse, and 48% reported being bullied. Approximately one in five adolescents reported clinically significant anxiety, depression, or hyperactivity (Table 1).

### Course of disordered eating behavior

A three-class solution was selected (Figure 1) as it had the lowest BIC value and all other model fit statistics were good (Table 2). Most adolescents followed a stable and *low* trajectory of disordered eating ( $n = 418$ , 77%). The next largest group followed a *moderate* trajectory ( $n = 100$ , 18%), which began low in early adolescence and increased over time (slope = 0.33). The smallest group followed a *high* trajectory ( $n = 26$ , 5%), which began low in early adolescence and rose rapidly in subsequent years (slope = 1.02) peaking at age 17. The posterior probabilities indicated that adolescents were well matched to their trajectory group (.97 for the low group, .90 for the moderate group, and .97 for the high group).

### Course of BPD features

A three-class solution was selected (Figure 2) because although the BIC value was slightly higher than the four-class solution, the other fit indices suggested the three-class solution was more parsimonious (Table 2). The largest group of adolescents followed a stable and *moderate* trajectory of BPD features ( $n = 225$ , 41%). The next largest group followed a stable and *low* trajectory ( $n = 204$ , 38%). The smallest group followed a moderately stable and *high* trajectory ( $n = 114$ , 21%), in which there was a slight increase in symptoms across early adolescence (slope = 0.18) that peaked at age 16. The posterior probabilities indicated that adolescents were well matched to their trajectory group (.94 for the low group, .89 for the moderate group, and .92 for the high group).

### Joint trajectories and patterns of comorbidity

The proportions of adolescents within each joint trajectory group are displayed in Table 3. Although there were nine possible joint trajectories (3 × 3 classes), there were no adolescents in the high disordered eating/low BPD features group. The largest proportion of adolescents reported low disordered eating/low BPD features (healthy group). Almost a quarter of the total sample (23%) had co-occurring symptoms.

### Temporal precedence of comorbidity

Conditional probability analyses (Table 3) indicated that belonging to the high disordered eating trajectory was a better indicator of belonging to the high BPD features trajectory than was the reverse. That is, there was a high probability (0.83) that adolescents in the high disordered eating trajectory would also be in the high BPD features trajectory, while there was a low probability (0.17) that adolescents in the high BPD features trajectory would also be in the high disordered eating trajectory. The probability that adolescents in the moderate disordered eating trajectory would be in the high

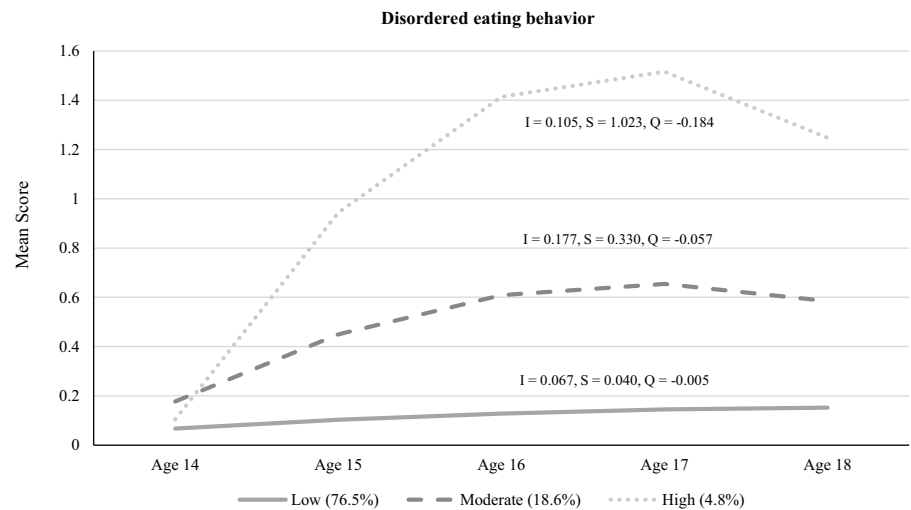
**Table 1.** Descriptive statistics

RISK FACTORS	Total		Boys		Girls		
	N	%	N	%	N	%	
<i>Demographic risks</i>							
<b>Race/ethnicity</b>	498		214		284		
White	413	82.9	173	80.8	240	84.5	
Non-white	85	17.1	41	19.2	44	15.5	
<b>Income</b>	508		228		280		
<70,000	189	37.2	82	36	107	38.2	
≥70,000	319	62.8	146	64	173	61.8	
<b>Parent education</b>	525		234		291		
< Bachelor's degree	199	37.9	92	39.3	107	36.8	
≥ Bachelor's degree	326	62.1	142	60.7	184	63.2	
<b>Sexual orientation</b>	324		128		196		
Heterosexual	280	86.4	119	93	161	82.1	
LGBTQ+	44	13.6	9	7	35	17.9	
<i>Interpersonal risks</i>							
<b>Physical abuse</b>	403		163		240		
No	340	84.4	134	82.2	206	85.8	
Yes	63	15.6	29	17.8	34	14.2	
<b>Sexual abuse</b>	407		167		240		
No	373	91.6	163	97.6	210	87.5	
Yes	34	8.4	4	2.4	30	12.5	
<b>Bullying (vic)</b>	542		240		302		
No	282	52	147	61.3	135	44.7	
Yes	260	48	93	38.7	167	55.3	
<b>Bullying (perp)</b>	541		240		301		
No	475	87.8	210	87.5	265	88	
Yes	66	12.2	30	12.5	36	12.	
<i>Clinical risks</i>							
<b>Anxiety</b>	542		240		302		
No	421	77.7	195	81.3	226	74.8	
Yes	121	22.3	45	18.8	76	25.2	
<b>Depression</b>	542		240		302		
No	421	77.7	205	85.4	216	71.5	
Yes	121	22.3	35	14.6	86	28.5	
<b>Somatization</b>	481		213		268		
No	428	89	198	93	230	85.8	
Yes	53	11	15	7	38	14.2	
<b>Hyperactivity</b>	542		240		302		
No	435	80.3	198	82.5	237	78.5	
Yes	107	19.7	42	17.5	65	21.5	
<b>Impulsivity (PR)</b>	534		239		295		
No	402	75.3	174	72.8	228	77.3	
Yes	132	24.7	65	27.2	67	22.7	
OUTCOMES	N	Mean	SD	Mean	SD	Mean	SD
<i>Disordered eating</i>							
Age 14	481	0.22	0.32	0.16	0.22	0.27	0.38

(Continued)

**Table 1.** (Continued)

OUTCOMES	N	Mean	SD	Mean	SD	Mean	SD
Age 15	486	0.27	0.37	0.17	0.25	0.36	0.42
Age 16	449	0.30	0.41	0.17	0.24	0.40	0.48
Age 17	434	0.29	0.37	0.19	0.25	0.37	0.42
Age 18	447	0.30	0.35	0.21	0.28	0.37	0.38
<b>BPD features</b>							
Age 14	480	1.26	0.60	1.16	0.55	1.34	0.63
Age 15	484	1.28	0.63	1.12	0.57	1.42	0.64
Age 16	448	1.33	0.64	1.15	0.57	1.48	0.65
Age 17	434	1.31	0.63	1.12	0.59	1.46	0.62
Age 18	448	1.33	0.65	1.18	0.65	1.45	0.62

**Figure 1.** Three-class solution for trajectories of disordered eating behavior.

BPD features trajectory (0.57) was similar to the probability that adolescents in the moderate BPD features trajectory would be in the high disordered eating trajectory (0.61).

### Unique and shared childhood risk factors

Most risk factors were statistically significant in the unadjusted (crude) models (Table 4, Model 1), except for race/ethnicity in the disordered eating and BPD features models, and parent education, bullying perpetration, hyperactivity, and impulsivity in the disordered eating models (see Table 4 for high vs low symptom trajectory groups; all other comparisons are in the supplementary material). Sex (being a girl) was the largest risk factor for belonging to the high disordered eating group (OR = 21.9, 95% CI = 2.9–164.5), while sexual orientation (LGBTQ+) was the largest risk factor for belonging to the high BPD features group (OR = 21.5, 95% CI = 7.0–66.3). In the adjusted models for disordered eating, sex could not be computed because there was only one boy in the high disordered eating trajectory. Most risk factors remained significant and of similar magnitude in the partially adjusted models (Models 2–4) and most risk factors were shared among disordered eating and BPD features. There were no unique risks for disordered eating, but

bullying perpetration and hyperactivity were unique risk factors for BPD features.

### Population attributable fractions (PAFs)

Adjusted PAFs were estimated for each *modifiable* risk factor that remained significant in the adjusted models (sex and sexual orientation were not deemed to be modifiable). Under the assumption that adolescents in the high symptom trajectory groups were at risk of becoming clinical cases, the results indicated that eliminating bullying victimization would have the largest impact on incidence reduction; keeping all other exposures constant and eradicating bullying victimization would prevent new cases of eating disorders by 66% and BPD by 37% (see supplementary file for all adjusted PAFs).

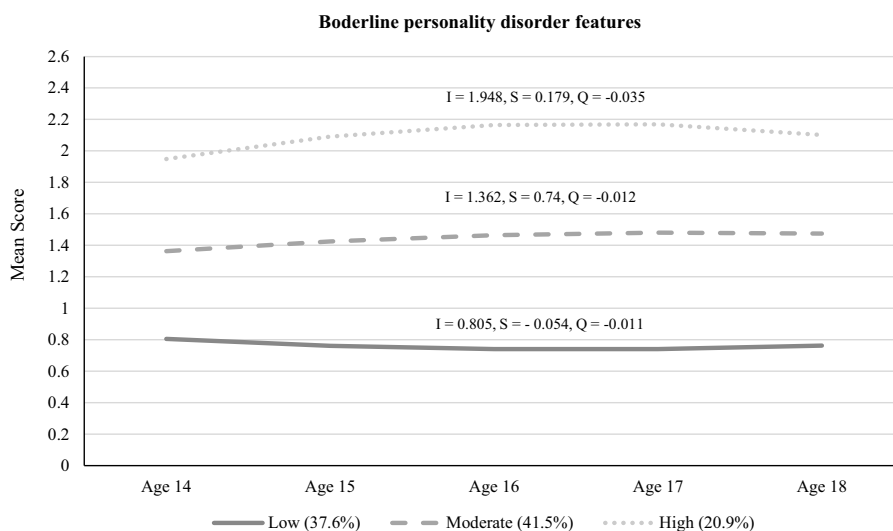
### Post hoc tests for clinical proxies

The prevalence of clinical eating disorders by age 19–20 was 41.7% among adolescent in the high disordered eating trajectory (vs. 17.8% in the moderate trajectory and 4.8% in the low trajectory). The odds that adolescents in the high disordered eating trajectory would develop a clinical eating disorder was 14 times higher than

**Table 2.** Model fit statistics

Classes	BIC	LMR-LRT	BLRT	Entropy
Disordered eating behavior				
One	1699.794	NA	NA	NA
Two	901.146	0.04	<0.001	0.952
Three	620.65	0.03	<0.001	0.907
Four	645.846	0.50	1	0.561
BPD features				
One	4426.656	NA	NA	NA
Two	3424.069	<0.001	<0.001	0.844
Three	3143.058	<0.001	<0.001	0.816
Four	3011.505	0.06	<0.001	0.785

Note. BIC = Bayesian Information Criteria; LMR-LTR = Lo-Mendell-Rubin likelihood ratio test; BLRT = bootstrapped likelihood ratio test.

**Figure 2.** Three-class solution for trajectories of features of borderline personality disorder.

adolescents in the low disordered eating trajectory (OR = 14.18, 95% CI = 3.96–50.77).

The prevalence of clinical BPD by age 19–20 was 36.8% among adolescent in the high BPD features trajectory (vs. 15.2% in the moderate trajectory and 1.5% in the low trajectory). The odds that adolescents in the high BPD features trajectory would develop clinical BPD was 37 times higher than adolescents in the low BPD features trajectory (OR = 37.21, 95% CI = 8.46–163.64).

## Discussion

We examined the course of disordered eating and BPD features, patterns of co-occurrence, temporal precedence, and risk factors across a developmentally sensitive period for the onset of these psychiatric problems. The main findings were that: (a) five percent of adolescents followed a high symptom trajectory of disordered eating, while 21% of adolescents followed a high symptom trajectory for BPD features; (b) almost a quarter of symptomatic youth reported co-occurring disordered eating and BPD features; (c) high levels of disordered eating were a stronger indicator of high levels of BPD features than was the reverse; (d) non-modifiable risk

factors were the strongest predictors of belonging to a high trajectory group (girls and LGBTQ+ youth). Most risk factors were shared, but bullying perpetration and hyperactivity were unique to BPD features; and (e) bullying victimization contributed the largest burden to expected clinical cases of eating disorders and BPD.

As the individual trajectories and rates of comorbidity found in this study are in line with previous work (Aimé et al., 2008; Haltigan & Vaillancourt, 2016a; Martinussen et al., 2017), we focus the discussion on the novel findings related to our other aims. First, although prior research has suggested substantial comorbidity between eating disorders and BPD, as replicated in this study with 23% of symptomatic youth reporting co-occurring symptom, there has been a lack of research investigating temporality. For the first time, we found that adolescents can experience high levels of BPD features without engaging in disordered eating behavior, but moderate or high levels of disordered eating were associated with moderate or high levels of BPD features. This finding mirrors research showing that disordered eating precedes depression (Lee & Vaillancourt, 2018) and fits broadly with the field of nutritional psychiatry (Marx et al., 2017). Disordered eating may also be an

**Table 3.** Joint and conditional probabilities

BPD features	Disordered eating behavior		
	Low	Moderate	High
N			
Low	198	6	0
Moderate	169	51	5
High	43	54	16
Probabilities of joint group membership			
Low	0.36	0.01	0.00
Moderate	0.31	0.09	0.01
High	0.08	0.10	0.03
Probabilities of disordered eating behavior conditional on BPD features			
Low	0.99	0.01	0.00
Moderate	0.75	0.23	0.02
High	0.23	0.61	0.17
Probabilities of BPD features conditional on disordered eating behavior			
Low	0.53	0.01	0.00
Moderate	0.41	0.42	0.17
High	0.06	0.57	0.83

indicator of self-harm or self-neglect that precipitates other symptoms of psychopathology. Compromised eating behavior thus plays a nonignorable role in mental health. The clinical implications are that interventions targeting disordered eating may inhibit the development of comorbid psychopathology. This warrants further investigation.

Second, girls were at the largest risk for high levels of disordered eating behavior, while LGBTQ+ youth were at the largest risk for high levels of BPD features. Regarding disordered eating, our results corroborate prior research showing that girls engage in more pathological eating behavior than boys (Croll et al., 2002; Micali et al., 2014), but we are cognizant that we were likely underpowered to detect effects among boys. Likewise, our results align with cross-sectional research showing that LGBTQ+ status confers risk for BPD, above and beyond sex assigned at birth, anxiety, and depression (Reuter et al., 2016). To our knowledge, this is the first prospective study to show that LGBTQ+ adolescents are at risk of fitting a profile of high BPD features longitudinally. Adolescence is a time of identity formation, which may explain why we found a higher proportion of adolescents with BPD features in this sample (21%) than has been found among university samples (9.7%; Meaney et al., 2016). LGBTQ+ youth may experience unique difficulties in identity formation in the face of invalidating environments and the enduring stigmatization of sexual orientations that deviate from heterosexual. Because we used a blunt characterization of sex (boy vs. girls) and sexual orientation (heterosexual vs. LGBTQ+), further research is needed to identify patterns for specific sexual and gender orientations and to examine the social and neurobiological mechanisms that link gender and sexuality to disordered eating and BPD features. Further research is also needed to examine risk factors for intersectional identities, as we were underpowered to test for these potential interactions.

Third, most risk factors were shared, but there were two unique childhood risk factors for high levels of BPD features: clinically significant hyperactivity and bullying perpetration. Although the overlap between ADHD and BPD is well documented (Vaillancourt et al., 2014; Winsper et al., 2017), questions remain as to whether ADHD and BPD are the same disorder in different stages of development (Gomes Cano et al., 2017) or whether they are different disorders with shared etiology, with early regulatory problems being one potential pathway (Vaillancourt et al., 2014; Winsper et al., 2017). Although bullying perpetration was a unique risk factor, bullying perpetration and victimization often co-occur (Haltigan & Vaillancourt, 2014; Lereya et al., 2015), and prior research has shown that childhood bullying victimization is a risk factor for probable BPD (Wolke et al., 2012). Developmentally sensitive research on the precise mechanisms that result in divergent pathways to BPD and eating disorders is therefore needed. The non-specific effects of the other sociodemographic, interpersonal, and clinical risk factors align with the principles of equifinality and multifinality (Cicchetti & Rogosch, 1996) and may explain the high levels of co-occurring symptoms.

Fourth, this is the first study to examine population attributable fractions in relation to disordered eating. We included a broad range of the most common and largest risk factors, but we were unable to examine all risks common to ED and BPD, such as genetic predisposition, gene-environment interactions, neglect, emotional abuse, or sleep problems (Amad et al., 2014; Morales-Muñoz et al., 2021; Solmi et al., 2021; Winsper et al., 2016; Yilmaz et al., 2015). In this study, childhood physical and sexual abuse were the largest interpersonal risk factors for BPD and bullying victimization was the largest interpersonal risk factor for disordered eating. The higher prevalence of bullying victimization in comparison to child maltreatment meant that expected clinical cases of eating disorder and BPD could be reduced the most by eradicating bullying. A study of large population-based cohorts from the United States and United Kingdom has similarly shown that being bullied in childhood is a stronger risk factor for internalizing problems than child maltreatment (Lereya et al., 2015), while other research has shown that bullying and physical abuse have similar effects on mental health (Lee & Vaillancourt, 2019b). The bullying measure used in this study examined general bullying, however, prior work has shown that bullying victims do receive more negative feedback about their appearance, which likely increases the risk of disordered eating (Lee et al., 2018). The strikingly high PAFs observed in this study mean that coordinated efforts to intervene early in childhood bullying remain a priority.

Despite the merits of our study, including the prospective design and the person-centered approach, it is not without limitations. Notably, we were unable to compare all levels of symptom co-occurrence because of relatively small numbers and thus low power within some of the trajectory groups. Further research is needed to determine whether risk factors are differentially associated with varying levels of comorbidity. As is typical in longitudinal research, there was participant attrition (9.3%). We accounted for missing data using the full information maximum likelihood estimation estimator, which has equivalent performance to multiple imputation (Lee & Shi, 2021). However, as missing data were systematic, we cannot conclude that the results are representative of our randomly sampled population. The analytic sample consisted of adolescents with higher household income and parental education, lower levels of mental health morbidities, and lower levels of bullying victimization and perpetration. As highly vulnerable adolescents were therefore most likely to drop out, the



**Table 4.** Crude and adjusted odds ratios (95% confidence intervals) for high vs low symptom trajectory groups

		Model 1 Unadjusted OR (95% CI)	Model 2 Adjusted AOR (95% CI)	Model 3 Adjusted AOR (95% CI)	Model 4 Adjusted AOR (95% CI)
Disordered eating behavior (High symptom group)	Sociodemographic risks				
	Sex	21.9 (2.9–164.5)	^	–	–
	Race/ethnicity	1.0 (0.3–3.4)	1.2 (0.1–10.6)	–	–
	Low income	2.8 (1.1–6.9)	1.5 (0.4–6.2)	–	–
	Parent education	0.5 (0.2–1.3)	0.3 (0.1–1.6)	–	–
	Sexual orientation	4.4 (1.3–15.4)	2.3 (0.5–10.5)		
	Interpersonal risks				
	Physical abuse	5.9 (2.0–17.6)	–	6.6 (1.8–23.8)	–
	Sexual abuse	9.0 (2.7–29.7)	–	7.3 (2.0–26.3)	–
	Bullying (vic)	4.7 (1.7–13.0)	–	7.4 (1.5–36.3)	–
	Bullying (perp)	1.9 (0.6–5.9)	–	2.3 (0.5–9.7)	–
	Clinical risks				
	Anxiety	3.1 (1.3–7.6)	–	–	0.9 (0.3–3.1)
	Depression	4.7 (1.9–11.2)	–	–	6.3 (2.0–19.8)
	Somatization	8.0 (2.9–22.4)	–	–	6.5 (2.0–21.0)
	Hyperactivity	2.1 (0.8–5.3)	–	–	1.6 (0.5–5.0)
Impulsivity (PR)	1.0 (0.3–2.7)			0.4 (0.1–1.5)	
BPD features (High symptom group)	Sociodemographic risks				
	Sex	3.7 (2.3–6.2)	2.8 (1.2–6.3)	–	–
	Race/ethnicity	0.7 (0.3–1.4)	1.1 (0.3–3.3)	–	–
	Low income	1.9 (1.1–3.1)	1.2 (0.6–2.8)	–	–
	Parent education	0.4 (0.2–0.7)	0.6 (0.3–1.4)	–	–
	Sexual orientation	21.5 (7.0–66.3)	20.2 (5.5–74.7)		
	Interpersonal risks				
	Physical abuse	5.2 (2.4–11.4)	–	5.1 (2.2–11.7)	–
	Sexual abuse	10.2 (3.3–31.8)	–	8.6 (2.6–27.7)	–
	Bullying (vic)	3.1 (1.9–5.1)	–	2.3 (1.2–4.3)	–
	Bullying (perp)	2.9 (1.4–6.0)	–	3.0 (1.2–7.2)	–
	Clinical risks				
	Anxiety	7.2 (4.0–12.9)	–	–	4.3 (2.2–8.5)
	Depression	6.3 (3.5–11.3)	–	–	3.1 (1.5–6.1)
	Somatization	7.0 (3.0–16.5)	–	–	3.1 (1.2–8.0)
	Hyperactivity	4.8 (2.7–8.8)	–	–	3.4 (1.7–6.7)
Impulsivity (PR)	2.9 (1.7–5.1)			1.8 (0.9–3.5)	

Note. Model 1 values are the crude estimates for each univariate risk factor; Model 2 estimates are adjusted for sociodemographic risk factors; Model 3 estimates are adjusted for interpersonal risk factors; Model 4 estimates are adjusted for clinical risks factors. AOR = adjusted odds ratio; CI = confidence interval; Vic=victimization; Perp=perpetration; PR = parent-reported. In all models the low symptom trajectory group was used as the reference category.

effect sizes may be larger than reported here. Our sample was also mostly white, and although this is reflective of the Canadian population (<http://www.statscan.gc.ca>), further large-scale and well-powered studies are needed to examine intersectionality (e.g., race/ethnicity and LGBTQ+). We used a composite measure of disordered eating, which did not allow us to test associations between BPD features and disorder-specific eating pathologies such as anorexia nervosa, bulimia nervosa, or binge eating disorder. However, the composite allowed us to overcome issues associated with female-centric and thinness-orientated (anorexia,

bulimia) classifications (Murray et al., 2017) and to account for diagnostic instability, Other Specified Feeding and Eating Disorders, and subclinical symptomology. Our measure is also unique because it focuses on eating disorder *behavior* and not *thoughts*, which are normative and overrepresented in community samples of girls (Miller et al., 2009). Most of our measures were self-reports, which can inflate effect sizes because of shared method variance, although our inclusion of parent-reported data and the longitudinal design helped to mitigate some of this bias. Last, the implications and generalizations that can be drawn from the data

are limited by the equivocal link between symptoms and disorders. However, in our post hoc analyses, we found that almost half of adolescents in the high disordered eating trajectory and over a third of adolescents in the high BPD features trajectory met the criteria for a clinical disorder by age 19–20. As subclinical symptoms are common and debilitating (Micali et al., 2015; Schaumberg et al., 2018; Zanarini et al., 2010), and most mental health problems remain undiagnosed and untreated (Wang et al., 2007), the measurement of symptoms in community samples remains valuable and important.

Notwithstanding the above limitations, this is the first study to systematically examine the joint trajectories, comorbidity, and unique risk factors for adolescent eating disorders and BPD – two of the most serious and enduring psychiatric illnesses – and we make several new contributions to knowledge. Although girls and LGBTQ+ youth fit profiles of high vulnerability, bullying prevention efforts could notably reduce eating and BPD pathology. Research into the early treatment of eating problems warrants further investigation, and the assessment of sexual orientation may be valuable when screening for BPD in young people.

**Supplementary material.** The supplementary material for this article can be found at <https://doi.org/10.1017/S0954579423000792>.

**Availability of data and material.** Data available on request.

**Code availability.** Code available on request.

**Funding statement.** This research was supported by Grants 833-2004-1019 and 435-2016-1251 from the Social Sciences and Humanities Research Council of Canada, Grants 201009MOP-232632-CHI-CECA-136591 and 201603PJT-365626-PJT-CECA-136591 from the Canadian Institute of Health Research, and the Grant PA-13-303 from the Ontario Mental Health Foundation.

**Competing interests.** Not applicable.

**Ethical standard.** This study was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All persons gave their informed consent prior to their inclusion in the study.

## References

- Aimé, A., Craig, W. M., Pepler, D., Jiang, D., & Connolly, J. (2008). Developmental pathways of eating problems in adolescents. *International Journal of Eating Disorders*, 41(8), 686–696. <https://doi.org/10.1002/eat.20561>
- Amad, A., Ramoz, N., Thomas, P., Jardri, R., & Gorwood, P. (2014). Genetics of borderline personality disorder: Systematic review and proposal of an integrative model. *Neuroscience & Biobehavioral Reviews*, 40, 6–19. <https://doi.org/10.1016/j.neubiorev.2014.01.003>
- Bohus, M., Kleindienst, N., Limberger, M. F., Stieglitz, R.-D., Domsalla, M., Chapman, A. L., Steil, R., Philipsen, A., & Wolf, M. (2009). The short version of the borderline symptom list (BSL-23): Development and initial data on psychometric properties. *Psychopathology*, 42(1), 32–39. <https://doi.org/10.1159/000173701>
- Brown, C. L., Skelton, J. A., Perrin, E. M., & Skinner, A. C. (2016). Behaviors and motivations for weight loss in children and adolescents. *Obesity*, 24(2), 446–452. <https://doi.org/10.1002/oby.21370>
- Buchanan, N. T., Bluestein, B. M., Nappa, A. C., Woods, K. C., & Depatie, M. M. (2013). Exploring gender differences in body image, eating pathology, and sexual harassment. *Body Image*, 10(3), 352–360. <https://doi.org/10.1016/j.bodyim.2013.03.004>
- Castellini, G., Lelli, L., Ricca, V., & Maggi, M. (2016). Sexuality in eating disorders patients: Etiological factors, sexual dysfunction and identity issues. A systematic review. *Hormone Molecular Biology and Clinical Investigation*, 25(2), 71–90. <https://doi.org/10.1515/hmbci-2015-0055>
- Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, 8(4), 597–600. <https://doi.org/10.1017/S0954579400007318>
- Crick, N. R., Murray-Close, D., & Woods, K. (2005). Borderline personality features in childhood: A short-term longitudinal study. *Development and Psychopathology*, 17(4), 1051–1070. <https://doi.org/10.1017/S0954579405050492>
- Croll, J., Neumark-Sztainer, D., Story, M., & Ireland, M. (2002). Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: Relationship to gender and ethnicity. *Journal of Adolescent Health*, 31(2), 166–175. [https://doi.org/10.1016/S1054-139X\(02\)00368-3](https://doi.org/10.1016/S1054-139X(02)00368-3)
- Cunningham, C. E., Boyle, M. H., Hong, S., Pettingill, P., & Bohaychuk, D. (2009). The brief child and family phone interview (BCFPI): 1. Rationale, development, and description of a computerized children's mental health intake and outcome assessment tool. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 50(4), 416–423. <https://doi.org/10.1111/j.1469-7610.2008.01970.x>
- Demmler, J. C., Brophy, S. T., Marchant, A., John, A., & Tan, J. O. A. (2019). Shining the light on eating disorders, incidence, prognosis and profiling of patients in primary and secondary care: National data linkage study. *The British Journal of Psychiatry: The Journal of Mental Science*, 1-8(2), 105–112. <https://doi.org/10.1192/bjp.2019.153>
- Gomes Cano, C., Pires, S., & Serrano, A. C. (2017). Attention deficit hyperactivity disorder (ADHD) and borderline personality disorder (BPD) – intersections systematic review. *European Psychiatry*, 41(S1), S439–S439. <https://doi.org/10.1016/j.eurpsy.2017.01.440>
- Haltigan, J. D., & Vaillancourt, T. (2014). Joint trajectories of bullying and peer victimization across elementary and middle school and associations with symptoms of psychopathology. *Developmental Psychology*, 50(11), 2426–2436. <https://doi.org/10.1037/a0038030>
- Haltigan, J. D., & Vaillancourt, T. (2016a). Identifying trajectories of borderline personality features in adolescence: Antecedent and interactive risk factors. *Canadian Journal of Psychiatry*, 61(3), 166–175. <https://doi.org/10.1177/0706743715625953>
- Haltigan, J. D., & Vaillancourt, T. (2016b). The borderline personality features scale for children (BPFS-C): Factor structure and measurement invariance across time and sex in a community-based sample. *Journal of Psychopathology and Behavioral Assessment*, 38(4), 600–614. <https://doi.org/10.1007/s10862-016-9550-1>
- Helfert, S., & Warschburger, P. (2011). A prospective study on the impact of peer and parental pressure on body dissatisfaction in adolescent girls and boys. *Body Image*, 8(2), 101–109. <https://doi.org/10.1016/j.bodyim.2011.01.004>
- Jacobi, C., Hayward, C., de Zwaan, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin*, 130(1), 19–65. <https://doi.org/10.1037/0033-2909.130.1.19>
- Johnson, D. M., Gunderson, J. G., Stout, R. L., McGlashan, T. H., Shea, M. T., Morey, L. C., Grilo, C. M., Zanarini, M. C., Yen, S., Markowitz, J. C., Sanislow, C., Ansell, E., Pinto, A., Skodol, A. E. (2003). Gender differences in borderline personality disorder: Findings from the Collaborative Longitudinal Personality Disorders Study. *Comprehensive Psychiatry*, 68(4), 827–837. [https://doi.org/10.1016/S0010-440X\(03\)0090-7](https://doi.org/10.1016/S0010-440X(03)0090-7)
- Kleindienst, N., Jungkunz, M., & Bohus, M. (2020). A proposed severity classification of borderline symptoms using the borderline symptom list (BSL-23). *Borderline Personality Disorder and Emotion Dysregulation*, 7(1), 1–11. <https://doi.org/10.1186/s40479-020-00126-6>
- Knauss, C., Paxton, S. J., & Alsaker, F. D. (2007). Relationships amongst body dissatisfaction, internalisation of the media body ideal and perceived pressure from media in adolescent girls and boys. *Body Image*, 4(4), 353–360. <https://doi.org/10.1016/j.bodyim.2007.06.007>
- Latzer, Y., Azaiza, F., & Tzischinsky, O. (2012). Not just a western girls' problem: Eating attitudes among Israeli-Arab adolescent boys and girls. *International Journal of Adolescence and Youth*, 19(3), 382–394. <https://doi.org/10.1080/02673843.2012.747973>
- Lee, K. S., Dale, J., Guy, A., & Wolke, D. (2018). Bullying and negative appearance feedback among adolescents: Is it objective or misperceived

- weight that matters? *Journal of Adolescence*, 63(1), 118–128. <https://doi.org/10.1016/j.adolescence.2017.12.008>
- Lee, K. S., Guy, A., Dale, J., & Wolke, D. (2017). Does psychological functioning mediate the relationship between bullying involvement and weight loss preoccupation in adolescents? A two-stage cross-sectional study. *International Journal of Behavioral Nutrition and Physical Activity*, 14(1), 1–11. <https://doi.org/10.1186/s12966-017-0491-1>
- Lee, K. S., & Vaillancourt, T. (2018). Longitudinal associations among bullying by peers, disordered eating behavior, and symptoms of depression during adolescence. *JAMA Psychiatry*, 75(6), 1–9. <https://doi.org/10.1001/jamapsychiatry.2018.0284>
- Lee, K. S., & Vaillancourt, T. (2019a). A four-year prospective study of bullying, anxiety, and disordered eating behavior across early adolescence. *Child Psychiatry & Human Development*, 50(5), 815–825. <https://doi.org/10.1007/s10578-019-00884-7>
- Lee, K. S., & Vaillancourt, T. (2019b). The role of childhood generalized anxiety in the internalizing cluster. *Psychological Medicine*, 50(13), 2272–2282. <https://doi.org/10.1017/S0033291719002484>
- Lee, T., & Shi, D. (2021). A comparison of full information maximum likelihood and multiple imputation in structural equation modeling with missing data. *Psychological Methods*, 26(4), 466–485. <https://doi.org/10.1037/met0000381>
- Lereya, S. T., Copeland, W. E., Costello, E. J., & Wolke, D. (2015). Adult mental health consequences of peer bullying and maltreatment in childhood: Two cohorts in two countries. *The Lancet Psychiatry*, 2(6), 524–531. [https://doi.org/10.1016/S2215-0366\(15\)00165-0](https://doi.org/10.1016/S2215-0366(15)00165-0)
- Lereya, S. T., Copeland, W. E., Zammit, S., & Wolke, D. (2015). Bully/victims: A longitudinal, population-based cohort study of their mental health. *European Child and Adolescent Psychiatry*, 24(12), 1–11. <https://doi.org/10.1007/s00787-015-0705-5>
- Mansournia, M. A., & Altman, D. G. (2018). Population attributable fraction. *BMJ*, 360(February), 2–3. <https://doi.org/10.1136/bmj.k757>
- Martinussen, M., Friberg, O., Schmierer, P., Kaiser, S., Øvergård, K. T., Neunhoeffer, A. L., Martinsen, E. W., & Rosenvinge, J. H. (2017). The comorbidity of personality disorders in eating disorders: A meta-analysis. *Eating and Weight Disorders*, 22(2), 201–209. <https://doi.org/10.1007/s40519-016-0345-x>
- Marx, W., Moseley, G., Berk, M., & Jacka, F. (2017). Nutritional psychiatry: The present state of the evidence. *Proceedings of the Nutrition Society*, 76(4), 427–436. <https://doi.org/10.1017/S0029665117002026>
- McCabe, M. P., & Ricciardelli, L. A. (2005). A prospective study of pressures from parents, peers, and the media on extreme weight change behaviors among adolescent boys and girls. *Behaviour Research and Therapy*, 43(5), 653–668. <https://doi.org/10.1016/j.brat.2004.05.004>
- McCabe, M. P., Ricciardelli, L. A., & Finemore, J. (2002). The role of puberty, media and popularity with peers on strategies to increase weight, decrease weight and increase muscle tone among adolescent boys and girls. *Journal of Psychosomatic Research*, 52(3), 145–153. [https://doi.org/10.1016/S0022-3999\(01\)00272-0](https://doi.org/10.1016/S0022-3999(01)00272-0)
- Meaney, R., Hasking, P., & Reupert, A. (2016). Prevalence of borderline personality disorder in university samples: Systematic review, meta-analysis and meta-regression. *PLoS ONE*, 11(5), 1–15. <https://doi.org/10.1371/journal.pone.0155439>
- Micali, N., Ploubidis, G., De Stavola, B., Simonoff, E., & Treasure, J. (2014). Frequency and patterns of eating disorder symptoms in early adolescence. *Journal of Adolescent Health*, 54(5), 574–581. <https://doi.org/10.1016/j.jadohealth.2013.10.200>
- Micali, N., Solmi, F., Horton, N. J., Crosby, R. D., Eddy, K. T., Calzo, J. P., Sonnevile, K. R., Swanson, S. A., & Field, A. E. (2015). Adolescent eating disorders predict psychiatric, high-risk behaviors and weight outcomes in young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(8), 652–659. <https://doi.org/10.1016/j.jaac.2015.05.009>
- Miller, J. L., & Boyle, M. (2009). *Short screen for eating disorders (SSED)*. McMaster University.
- Miller, J. L., Vaillancourt, T., & Hanna, S. E. (2009). The measurement of, eating-disorder-thoughts, and “eating-disorder-behaviors”: Implications for assessment and detection of eating disorders in epidemiological studies. *Eating Behaviors*, 10(2), 89–96. <https://doi.org/10.1016/j.eatbeh.2009.02.002>
- Morales-Muñoz, I., Durdurak, B. B., Bilgin, A., Marwaha, S., & Winsper, C. (2021). Understanding the relationship between sleep problems in early childhood and borderline personality disorder: A narrative review. *Nature and Science of Sleep*, 13, 2175–2202. <https://doi.org/10.2147/NSS.S311672>
- Murray, S. B., Nagata, J. M., Griffiths, S., Calzo, J. P., Brown, T. A., Mitchison, D., Blashill, A. J., & Mond, J. M. (2017). The enigma of male eating disorders: A critical review and synthesis. *Clinical Psychology Review*, 57(July), 1–11. <https://doi.org/10.1016/j.cpr.2017.08.001>
- Muthén, L. K., & Muthén, B. O. (2017). *Mplus user's guide* (8th ed.). Muthén & Muthén.
- Nagin, D. (2005). *Group-based modeling of development*. Harvard University Press. <https://doi.org/10.4159/9780674041318>
- Nylund, K. L., Asparouhov, T., & Muthén, B. O. (2007). Deciding on the number of classes in latent class analysis and growth mixture modeling: A Monte Carlo simulation study. *Structural Equation Modeling*, 14(4), 535–569. <https://doi.org/10.1080/1070510701575396>
- Reich, D. B., & Zanarini, M. C. (2008). Sexual orientation and relationship choice in borderline personality disorder over ten years of prospective follow-up. *Journal of Personality Disorders*, 22(6), 564–572. <https://doi.org/10.1521/pedi.2008.22.6.564>
- Reuter, T. R., Sharp, C., Kalpakci, A. H., Choi, H. J., & Temple, J. R. (2016). Sexual orientation and borderline personality disorder features in a community sample of adolescents. *Journal of Personality Disorders*, 30(5), 694–707. <https://doi.org/10.1521/pedi.2015.29.224>
- Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior assessment system for children* (2nd ed.). Pearson.
- Schaumberg, K., Jangmo, A., Thornton, L. M., Birgegård, A., Almqvist, C., Norring, C., Larsson, H., & Bulik, C. M. (2018). Patterns of diagnostic transition in eating disorders: A longitudinal population study in Sweden. *Psychological Medicine*, 49(5), 1–9. <https://doi.org/10.1017/S0033291718001472>
- Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Janavs, J., Weiller, E., Keskiner, A., Schinka, J., Knapp, E., Sheehan, M. F., Dunbar, G. C. (1997). The validity of the Mini International Neuropsychiatric Interview (MINI) according to the SCID-P and its reliability. *European Psychiatry*, 12(5), 232–241. [https://doi.org/10.1016/S0924-9338\(97\)83297-X](https://doi.org/10.1016/S0924-9338(97)83297-X)
- Solmi, M., Radua, J., Stubbs, B., Ricca, V., Moretti, D., Busatta, D., Carvalho, A. F., Dragioti, E., Favaro, A., Monteleone, A. M., Shin, J. II, Fusar-Poli, P., Castellini, G. (2021). Risk factors for eating disorders: An umbrella review of published meta-analyses. *Brazilian Journal of Psychiatry*, 43(3), 314–323. <https://doi.org/10.1590/1516-4446-2020-1099>
- Stepp, S., Lazarus, S., & Byrd, A. (2016). A systematic review of risk factors prospectively associated with borderline personality disorder: Taking stock and moving forward. *Personality Disorders: Theory, Research, and Treatment*, 7(4), 316–323. <https://doi.org/10.1037/per0000186.A>
- Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents. *Archives of General Psychiatry*, 68(7), 714–723. <https://doi.org/10.1001/archgenpsychiatry.2011.22>
- Temes, C. M., Frankenburg, F. R., Fitzmaurice, G. M., & Zanarini, M. C. (2019). Deaths by suicide and other causes among patients with borderline personality disorder and personality-disordered comparison subjects over 24 years of prospective follow-up. *Journal of Clinical Psychiatry*, 80(1), 30–36. <https://doi.org/10.4088/JCP.18m12436>
- Vaillancourt, T., Brittain, H. L., McDougall, P., & Duku, E. (2013). Longitudinal links between childhood peer victimization, internalizing and externalizing problems, and academic functioning: Developmental cascades. *Journal of Abnormal Child Psychology*, 41(8), 1203–1215. <https://doi.org/10.1007/s10802-013-9781-5>
- Vaillancourt, T., Brittain, H. L., McDougall, P., Krygman, A., Boylan, K., Duku, E., & Hymel, S. (2014). Predicting borderline personality disorder symptoms in adolescents from childhood physical and relational aggression, depression, and attention-deficit/hyperactivity disorder. *Development and Psychopathology*, 26(3), 817–830. <https://doi.org/10.1017/S0954579414000418>

- Vaillancourt, T., & Haltigan, J. D. (2018). Joint trajectories of depression and perfectionism across adolescence and childhood risk factors. *Development and Psychopathology*, 30(2), 461–477. <https://doi.org/10.1017/S0954579417000979>
- Vaillancourt, T., Trinh, V., McDougall, P., Duku, E., Cunningham, L., Cunningham, C., Hymel, S., & Short, K. (2010). Optimizing population screening of bullying in school-aged children. *Journal of School Violence*, 9(3), 233–250. <https://doi.org/10.1080/15388220.2010.483182>
- van Furth, E. F., van der Meer, A., & Cowan, K. (2016). Top 10 research priorities for eating disorders. *The Lancet Psychiatry*, 3(8), 706–707. [https://doi.org/10.1016/s2215-0366\(16\)30147-x](https://doi.org/10.1016/s2215-0366(16)30147-x)
- Walsh, C. A., MacMillan, H. L., Trocmé, N., Jamieson, E., & Boyle, M. H. (2008). Measurement of victimization in adolescence: Development and validation of the Childhood Experiences of Violence Questionnaire. *Child Abuse and Neglect*, 32(11), 1037–1057. <https://doi.org/10.1016/j.chiabu.2008.05.003>
- Wang, P. S., Aguilar-Gaxiola, S., Alonso, J., Angermeyer, M. C., Borges, G., Bromet, E. J., Bruffaerts, R., de Girolamo, G., de Graaf, R., Gureje, O., Haro, J. M., Karam, E. G., Kessler, R. C., Kovess, V., Lane, M. C., Lee, S., Levinson, D., Ono, Y., Petukhova, M., . . . , J. E. Wells (2007). Use of mental health services for anxiety, mood, and substance disorders in 17 countries in the WHO world mental health surveys. *Lancet*, 370(9590), 841–850. [https://doi.org/10.1016/S0140-6736\(07\)61414-7](https://doi.org/10.1016/S0140-6736(07)61414-7)
- Wildes, J. E., Emery, R. E., & Simons, A. D. (2001). The roles of ethnicity and culture in the development of eating disturbance and body dissatisfaction: A meta-analytic review. *Clinical Psychology Review*, 21(4), 521–551. [https://doi.org/10.1016/S0272-7358\(99\)00071-9](https://doi.org/10.1016/S0272-7358(99)00071-9)
- Williamson, D. F. (2010). The population attributable fraction and confounding: Buyer beware. *International Journal of Clinical Practice*, 64(8), 1019–1023. <https://doi.org/10.1111/j.1742-1241.2010.02443.x>
- Winsper, C., Hall, J., Strauss, V. Y., & Wolke, D. (2017). Aetiological pathways to Borderline Personality Disorder symptoms in early adolescence: Childhood dysregulated behaviour, maladaptive parenting and bully victimisation. *Borderline Personality Disorder and Emotion Dysregulation*, 4(1), 1–10. <https://doi.org/10.1186/s40479-017-0060-x>
- Winsper, C., Lereya, S. T., Marwaha, S., Thompson, A., Eyden, J., & Singh, S. P. (2016). The aetiological and psychopathological validity of borderline personality disorder in youth: A systematic review and meta-analysis. *Clinical Psychology Review*, 44, 13–24. <https://doi.org/10.1016/j.cpr.2015.12.001>
- Wolke, D., Schreier, A., Zanarini, M. C., & Winsper, C. (2012). Bullied by peers in childhood and borderline personality symptoms at 11 years of age: A prospective study. *Journal of Child Psychology and Psychiatry*, 53(8), 846–855.
- Yilmaz, Z., Hardaway, J. A., & Bulik, C. M. (2015). Genetics and epigenetics of eating disorders. *Advances in Genomics and Genetics*, 5, 131. <https://doi.org/10.2147/AGG.S55776>
- Zanarini, M. C., Reichman, C. A., Frankenburg, F. R., Reich, D. B., & Fitzmaurice, G. (2010). The course of eating disorders in patients with borderline personality disorder: A 10-year follow-up study. *International Journal of Eating Disorders*, 43(3), 226–232. <https://doi.org/10.1002/eat.20689>