



Risk factors for eating disorder symptoms at 15 years of age: a 9-year longitudinal cohort study

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ABSTRACT

Eating disorders (EDs) are typically diagnosed in the later stages of puberty, but risk factors for EDs are present in pre-pubertal children. This longitudinal, population-based birth cohort study aimed to examine prospective predictors of eating disorder symptoms in 15-year-olds. Specifically, we sought to test an adapted dual pathway model of disordered eating aetiology in this group. Participants in the Gateshead Millennium Study birth cohort ($n = 326$; 187 girls and 175 boys) completed self-report questionnaires assessing eating disorder symptoms and risk factors at ages 7, 9, 12, and 15 years. Measures included body image, depressive symptoms, and pubertal development; we also measured BMI at each age. The data were fitted to a model of eating disorder symptom development to help us understand the role of puberty, adiposity, body dissatisfaction, depressive symptoms, and previous eating disorder symptoms. We found that previous eating disorder symptoms were the strongest predictor of eating disorder symptoms at 15, and that depressive symptoms prospectively predicted eating disorder symptoms in girls. We furthermore found that depressive symptoms at 12 partially mediated the relationship between body dissatisfaction at 12 and eating disorder symptoms at 15 in girls. Pubertal development predicted concurrent eating disorder symptoms at 12 in girls but not boys. Overall, our findings support the roles of pubertal development, body dissatisfaction, and depressive symptoms in the pathogenesis of eating disorder symptoms across puberty. They add to growing evidence that there are different pathways to eating disorder symptoms in girls and boys.

1. Introduction

Eating disorders typically develop in adolescence, with incidence increasing markedly from age 10–12 to 13–16 (Cybulski et al., 2021). Research examining the causes of disordered eating predominantly includes participants from age 12. By this age, pubertal development is well-established (Bond et al., 2006) and disordered eating is common (Croll et al., 2002). Globally, around 20 % of children and adolescents screen positive for clinically disordered eating (López-Gil et al., 2023). Even more common are subclinical eating disorder symptoms in childhood: eating- and weight-related behaviours and cognitions which are found in eating disorders, but less severe, frequent, and/or impairing (Dias et al., 2023). Understanding the developmental precursors of eating disorder symptoms is vital, as they prospectively predict weight

gain, depression, and clinical eating disorders in later adolescence (Hahn et al., 2023; Landstedt et al., 2018), which in turn predict adult mental ill-health (Linardon et al., 2021; Wade et al., 2012).

Multiple factors contribute to the pathogenesis of disordered eating, including (epi)genetic, biological, sociocultural, and economic variables. Culbert et al. (2015) reviewed the biopsychosocial predictors of eating disorders, highlighting a lack of prospective studies beginning in pre-adolescence and spanning puberty. More recently, a review by Barakat et al. (2023) similarly concluded that prospective, developmental research into eating disorder risk is needed. The current study addressed this gap by tracking eating disorder symptoms and putative predictors across nine years in children aged 7 years at baseline, with follow up measurements every 2–3 years until age 15.

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1.1. Predictors of eating disorder symptoms from childhood to adolescence

Existing longitudinal evidence about predictors of eating disorder symptoms in non-clinical populations is scarce, and primarily involves US-based mid-to late-adolescent girls. Findings with adolescents indicate that higher adiposity leads to body dissatisfaction (Presnell et al., 2004), which subsequently predicts eating disorder symptoms through the mediating influence of negative affect (Ferreiro et al., 2014; Stice et al., 2011). Pubertal development is concurrent with higher adiposity (Silventoinen et al., 2022) but potentially also predicts girls' eating disorder symptoms through psychosocial, genetic, and endocrine mechanisms independent of BMI (Klump, 2013). These relationships constitute an adapted version of the Dual Pathway model of disordered eating (Lewis-Smith et al., 2020; Stice et al., 1996; see Supplementary Materials 1 Fig. S1). This framework originally focused on adult bulimic symptoms but has since been prospectively applied to other forms of adult/adolescent disordered eating (Holmes et al., 2015; Stice & Van Ryzin, 2019), and, cross-sectionally, to eating disorder symptoms in children (Evans et al., 2013; Vander Wal et al., 2008).

The dual pathway model proposes that elevated adiposity prompts subsequent body dissatisfaction because of the larger discrepancy between higher-weight bodies and the societally-promoted appearance ideal (Cruz-Sáez et al., 2020; Cybulski et al., 2021). This is less well-evidenced in children than adults, although both Blundell et al. (2024) and Lewis-Smith et al. (2020) found that increased BMI z-score predicted body dissatisfaction between ages 7–11. These findings underscore the importance of examining pre-pubertal developmental factors for disordered eating. The model next proposes that body dissatisfaction prompts global negative self-evaluations, underpinning the development of depressive symptoms. These then precipitate eating disorder symptoms via, for example, ruminative thinking (Nolen-Hoeksema et al., 2007; Skinner et al., 2012), eating- and body-related shame (Presnell et al., 2009), or to regulate negative emotions (Evans et al., 2013). In longitudinal data from children aged 7–14, Lewis-Smith et al. (2020) tested an adapted Dual Pathway model and found that pre-adolescent body dissatisfaction and depression mediated the relationship between adiposity and eating disorder symptoms. Blundell et al. (2024) also found that body dissatisfaction at 11 partially mediated the association between adiposity at 7 and depressive symptoms at 14, supporting the proposed causal pathways. The magnitude of associations in both studies was larger in girls than boys.

Peer-to-peer differences in pubertal development may additionally influence eating disorder symptoms, particularly in girls (Vo et al., 2021). Adolescents who are more or less pubertally advanced report heightened body dissatisfaction for both boys and girls (Dantas et al., 2017; Thériault et al., 2019), depression in girls (van Rijn et al., 2023), and disordered eating symptoms in boys and girls (Le Grange et al., 2014). Few existing longitudinal studies of eating disorder risk consider pubertal development's role in conjunction with other risk factors. Potentially, girls' and boys' different patterns of pubertal development alongside increasingly gender-differentiated sociocultural influences help explain higher eating disorder and depressive symptoms from age 13 in girls compared to boys (Klump, 2013; Papp et al., 2013). Thus, research should examine predictive pathways separately for boys and girls.

1.2. Persistence of eating disorder symptoms across time

Eating disorder symptoms persist across time in adolescents and adults, and growing evidence suggests that this applies to children (McClelland et al., 2020; Sanzari et al., 2023). A systematic review of longitudinal studies of eating disorder risk in childhood/adolescence suggests that early eating-related difficulties (e.g., dietary restriction, weight concerns) predict future onset of eating disorder symptoms

(McClelland et al., 2020). This across-time stability does not argue against the need to identify other early predictors of disordered eating – rather, it argues for the importance of detecting early symptoms of disordered eating (e.g., for use in prevention; Le et al., 2017).

Finally, it remains unclear at what age specific factors become prospectively predictive, and whether this differs for girls and boys. For example, body dissatisfaction did not prospectively predict eating disorder symptoms in several studies with younger children (Evans et al., 2017; Ferreiro et al., 2012; Rohde et al., 2015) but studies with older children (e.g., Sharpe et al., 2018) suggest it emerges as a predictor around 13. There is insufficient prior research to ascertain when either depressive symptoms or dietary restraint become prospective predictors, although they are prospectively predictive in adolescence (Kelly et al., 2015; Lewis-Smith et al., 2020; Murray et al., 2018; Stice & Van Ryzin, 2019). Research is needed over longer timespans, from younger ages, measuring multiple risk factors alongside eating disorder symptoms, to clarify if/when they cease being correlates and become prospective risk factors.

1.3. Previous studies with the current birth cohort (Gateshead Millennium Study Cohort)

This paper reports data on longitudinal risk factors for eating disorder symptoms at age 15 in a UK-based birth cohort, the Gateshead Millennium Study Cohort. Our previous papers reported earlier eating disorder symptom predictive data with this cohort at 9 and 12 respectively (Evans et al., 2017; Parkinson et al., 2012; see Parkinson et al., 2011 for cohort profile). These showed that 7-year-olds' dietary restraint longitudinally predicted eating disorder symptoms at 12, but body dissatisfaction and depressive symptoms were only correlates. Building on these findings, this paper integrated data from all timepoints to test the Dual Pathway model of eating disorder symptom development.

1.4. Current study

The aim of the current study was to establish predictors of eating disorder symptoms in adolescents at 15, using data from ages 7, 9, and 12. As a secondary aim, we also examined longitudinal predictors of 15-year body dissatisfaction. Fig. 1 shows the Dual Pathway model we tested separately for girls and boys.

We hypothesised (H1) that higher BMI would predict higher body dissatisfaction at the subsequent time point (as per Lewis-Smith et al., 2020). Based on existing research, we further hypothesised (H2) that body dissatisfaction would longitudinally predict eating disorder symptoms at age 15, mediated by depressive symptoms. As shown by Lewis-Smith et al. (2020) we predicted (H3) that depressive symptoms would also directly prospectively predict eating disorder symptoms. Finally, we hypothesised (H4) that puberty would be a significant covariate of eating disorder symptoms for girls but not boys (e.g., Klump, 2013; Lewis-Smith et al., 2020).

2. Methods

2.1. Participants

The study involved participants in the Gateshead Millennium Study (GMS) birth cohort. We invited mothers of all infants born in 35 pre-specified weeks (June 1999–May 2000) resident in Gateshead, an urban district in northeast England, to permit their infant(s) to join a longitudinal study of feeding and growth (Parkinson, Wright, & Drewett, 2007). 1029 infants (82 % of those invited) joined the study. Mothers were primarily from the white ethnic majority group (98 %) (Parkinson et al., 2010), which represented the ethnic composition of Gateshead at the time. Full cohort details are published elsewhere (Parkinson et al., 2007; Parkinson et al., 2010).

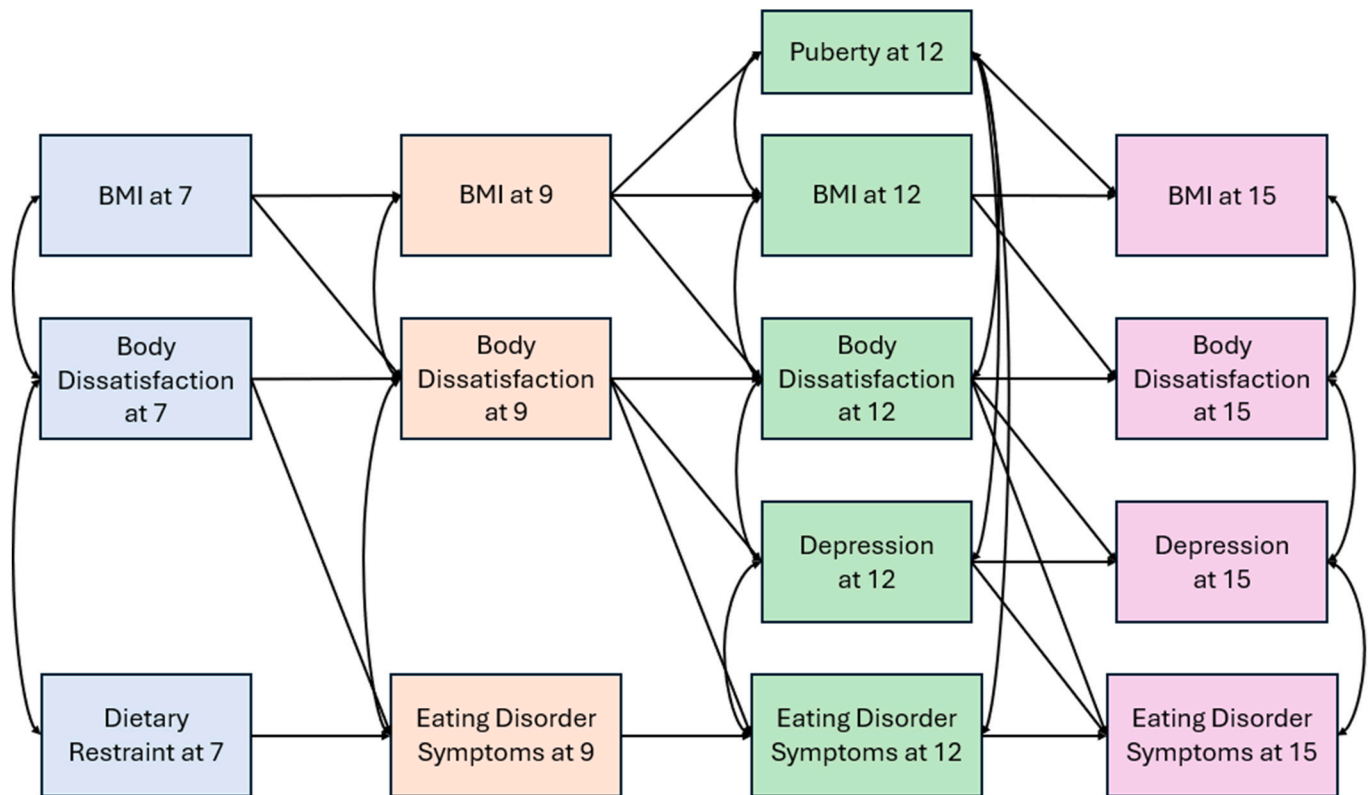


Fig. 1. Adapted dual pathway model of Eating Disorder Symptom development from age 7–15. Straight arrows indicate prospective effects while curved arrows indicate covariation. Blue boxes indicate variables at age 7, orange boxes indicate variables at age 9, green boxes indicate variables at age 12, and pink boxes indicate variables at age 15. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

We have followed up this cohort at intervals since recruitment. At each follow-up assessment, all children whose families have not previously asked to leave the study were eligible to participate. For the purposes of the present study, assessments of the children were taken at three follow-ups: 6–8 years referred to as 7 years in this paper (median 7.4, range 6.4–8.4; $n = 609$); 8–10 years referred to as 9 years (median 9.3, range 8.4–10.2; $n = 589$); 11–13 years referred to as 12 years (median 12.5 range 11.6–13.3; $n = 525$); and 15–16 years referred to as 15 years (median 15.2 range 14.5–16.2; $n = 362$). The mean interval between the 7- and 9-year assessments was 1.9 years ($SD = 0.2$ years), the mean interval between the 9- and 12-year assessments was 3.2 years ($SD = 0.3$ years), and the mean interval between the 12- and 15-year assessments was 2.7 years ($SD = 0.3$ years). Mothers gave written consent for their own participation and for their child to participate in the study, and children/adolescents were also asked to provide assent from age 9 onwards.

Ethical approval was granted by Gateshead and South Tyneside Local Research Ethics Committee (7-year follow up) and by Newcastle University Research Ethics Committee (9-year, 12-year, and 15-year follow ups).

2.2. Measures

2.2.1. Body image

Different, age-appropriate measures of body image were used at different measurement timepoints, described below. A pictorial figure-choice scale, designed for preadolescents, was used at 7 and 9 years. A questionnaire for adolescents was used at 12 and 15 years.

- i) The Children's Body Image Scale (CBIS) (Truby & Paxton, 2002, 2008) is a figure rating scale validated with children aged 7 to 12 as a measure of perceived and preferred body shape/size (Truby &

Paxton, 2002). Children completed this scale at 7 years and 9 years. It consists of seven photographic images of boys and seven of girls ranging from very low to very high BMI (representing the following BMI centiles: 3rd, 10th, 25th, 50th, 75th, 90th and 97th). Children view the images of either boys or girls depending on their gender. They select the image that looks most like them (perceived figure) and the image they would like to look like (preferred figure). A directional discrepancy score between the two images can be calculated as an index of body dissatisfaction: images are numbered 1–7, from lowest to highest adiposity, and the perceived figure score is subtracted from the preferred figure score to produce a directional discrepancy score. A negative score indicates a preference for a smaller figure, and a positive score indicates a preference for a larger figure. The CBIS has good test-retest reliability ($r = 0.7$) (Truby & Paxton, 2008) and construct validity in boys and girls aged 7–11-years (Truby & Paxton, 2002).

- ii) The Impact of Weight on Quality of Life-Kids (IWQOL-Kids) (Kolotkin et al., 2006) measures self-reported weight-specific quality of life for adolescents aged 11–19. We used the 9-item body esteem subscale at 12 and 15 years to assess body dissatisfaction. Participants indicate how frequently they experience an impact of weight on self-perceptions and appearance satisfaction using a Likert scale from 1 (always) to 5 (never). The subscale is a valid and reliable measure of body dissatisfaction (internal consistency: $\alpha = 0.95$) (Kolotkin et al., 2006). Lower scores on the IWQOL-Kids indicate higher body dissatisfaction. In the current sample, the internal consistency of this subscale was $\alpha = 0.93$ at 12-years and $\alpha = 0.96$ at 15-years.

2.2.2. Eating disorder symptoms and dietary restraint

At the age 7 data collection, dietary restraint was measured as a proxy for eating disorder symptoms because the ChEAT (used at ages 9

and 12) and the EAT-26 (used at age 15) are unsuitable for children aged 6 (or below), and some of the sample was not yet 7 at the point of measurement.

- i) The 7-item restraint subscale of the Dutch Eating Behaviour Questionnaire child version (DEBQ-C) (van Strien & Oosterveld, 2008) was used to assess dietary restraint at 7 years. It consists of seven items which ask the participant to rate how often they eat reduced amounts of food in order to lose or maintain weight, to which they respond 'no', 'sometimes', or 'yes'. Higher scores on this scale indicate higher dietary restraint. This scale has shown high reliability and validity in boys and girls aged 7–12 (van Strien & Oosterveld, 2008). In the current sample, the internal consistency of this subscale was $\alpha = 0.60$.
- ii) The 23-item Children's Eating Attitudes Test (ChEAT) (Maloney et al., 1988) was used to assess eating disorder symptoms (Garner et al., 1982; Garner & Garfinkel, 1979) at 9 and 12 years. Participants indicate how frequently they experience each symptom using a Likert scale from 1 (never) to 6 (always). Responses are scored 1, 2, or 3 for the three most symptomatic answers, with the three least symptomatic being scored 0. Scores are calculated by summing the answers, meaning that higher scores indicate more disordered eating symptomatology. The ChEAT demonstrates good test-retest reliability ($r = 0.8$) and internal consistency ($\alpha = 0.9$) (Maloney et al., 1988; Smolak & Levine, 1994) and is suitable for children from age 7. In the current sample, the ChEAT showed good internal consistency (α at 9 years = 0.83; 12 years = 0.85).
- iii) The 26 item Eating Attitudes Test (EAT-26) (Garner & Garfinkel, 1979) was used to assess eating disorder symptoms at 15-years. The EAT is virtually identical to the ChEAT, with only minor linguistic differences. It is scored in the same way as the ChEAT and demonstrates good reliability and internal consistency (Garner & Garfinkel, 1979; Ocker et al., 2007). In the current sample it showed good internal consistency ($\alpha = 0.88$).

2.2.3. Depressive symptoms

The 10-item Child Depression Inventory – Short Form (CDI-S) (Kovacs, 1992) was used to assess depressive symptoms at 12 and 15 years. Each item is made up of three statements regarding the participants' feelings over the preceding two weeks which are scored from 0 to 2. The total score is the sum of the ten items, with higher scores indicating greater depressive symptomatology. The CDI has been found to be a valid (girls internal consistency $\alpha = 0.87$; boys $\alpha = 0.84$) and reliable (girls $r = 0.74$; boys $r = 0.77$) measure of depressive symptoms with children aged 8–16 (Smucker et al., 1986). In the current study, the CDIs was completed at ages 12 and 15 with internal consistencies of $\alpha = 0.77$ at age 12, and $\alpha = 0.89$ at age 15.

2.2.4. Pubertal development

The 5-item Pubertal Development Scale (PDS) (Petersen et al., 1988) was used to measure pubertal development at age 12 and 15. Items ask about physical signs of pubertal maturation, and the last two are sex specific. Participants report whether the pubertal function has not started, barely begun, is underway, or is complete. Scores are calculated by averaging scores for all five questions with higher scores indicating the child is further through puberty. Scores can then be split into pre-, early-, middle-, and late-pubertal categories. The PDS has shown to have high internal consistency and reliability in both boys and girls across childhood and adolescence (Koopman-Verhoeff et al., 2020).

2.2.5. Adiposity (body measurements)

Weight and height were measured at each age using equipment bought from Chasmors, London. Weight was measured to 0.1 kg using Tanita scales TBF-300MA, and height was measured to 0.1 cm with the head in the Frankfurt plane using a Leicester portable height measure. Measurements were taken a minimum of twice, and until two

consecutive values within 0.1kg/0.1 cm were taken and the mean of these two values was used. Body mass index (BMI) was calculated as kg/m² as a proxy for adiposity. It constitutes an acceptable and pragmatic index of total body fat and percentage of body weight as fat in child and adolescent samples (Pietrobelli et al., 1998). BMI values were converted to z-scores for age and sex using the UK90 reference data set (Cole et al., 1995).

2.2.6. Socio-economic status

Socio-economic status was measured using family's postcode at birth, which was used to obtain the Townsend deprivation score (Townsend et al., 1989). Attrition was slightly higher in lower SES groups, but the overall distribution of SES remained broadly similar because children from lower SES families were purposefully over-recruited at baseline. Townsend score quintiles of the sample at age 15 compared to the cohort at birth can be found in the Supplementary Materials 1 (Table S1).

2.3. Procedure

The data were collected by researchers trained in anthropometry and the other study procedures. At each follow up the children were visited in schools, or at home, to collect anthropometric and questionnaire data. If necessary, the researchers helped the children with comprehension of the questionnaires, using a standardised study assessment protocol.

2.4. Statistical analysis

Analyses were conducted on all cases where eating disorder symptom score at 15 years was recorded. In total, 362 adolescents were measured in the 15-year follow-up, and eating disorder symptom scores were available for all of these. As participant numbers were fixed by cohort attrition, no power calculations were appropriate.

During analysis, anthropometric and scale data were not normally distributed, so the median and interquartile range was used to summarise the data, and Mann-Whitney U tests were used to compare scores between boys and girls. Correlations between values were calculated separately for boys and girls using Spearman's Rho. A significance threshold of $p < .005$ was used in the gender comparison and correlations analyses (see Supplementary Materials 1) to correct for multiple comparisons following analysis methods used in previous publications of this cohort (Evans et al., 2017). Path analysis was used to determine the relationship between across-time and within-time correlates of eating disorder symptoms at 15 for the boys and girls separately. Finally, we used mediation analysis to investigate the relationship between body dissatisfaction at 12 and eating disorder symptoms at 15, testing whether this was mediated by depressive symptoms at 12 as predicted by the Dual Pathway model. We used R Studio (RStudio Team, 2023) to analyse the data, using the Tidyverse (Wickham et al., 2019), rstatix (Kassambara, 2023), lavaan (Rosseel, 2012), and semPlots (Epskamp, 2022) packages. For clarity, throughout the inferential analyses, the body image variables (measured using different methods at 7 and 9 years, and 12 and 15 years) are referred to as body dissatisfaction such that higher scores indicate a more negative evaluation of one's body.

3. Results

3.1. Descriptive statistics

Disordered eating symptom data were collected for 362 adolescents (175 boys and 187 girls) at the 15-year follow up; 32.9 % were 14 years old, 64.4 % were 15 years old and 2.8 % were 16 years old. Sample descriptives are shown by sex assigned at birth at each follow up assessment in Table 1. Some variables had missing data; the proportion of complete data for each variable ranged from 81 to 86 % at age 7, 80–92 % at age 9, 94 % at age 12 and from 98 to 100 % at age 15.

Table 1
Median values for main variables and sex differences (n = 362).

	Boys (n = 175) Median (IQR)	Girls (n = 187) Median (IQR)	Z	p
7 Years:				
BMI	15.9 (2.5)	16.4 (2.7)	-1.9	0.060
BMI z-score	0.2 (0.5)	0.3 (0.8)	-0.9	0.355
Dietary Restraint	2.0 (0.6)	2.1 (0.6)	-0.1	0.941
Body Dissatisfaction	0.0 (1)	-1.0 (2)	-1.9	0.053
9 Years:				
BMI	16.9 (2.8)	17.8 (3.0)	-2.8	.005
BMI z-score	0.4 (0.8)	0.5 (1.0)	-1.0	0.339
Eating disorder symptoms	13.0 (12.0)	10.0 (11.0)	-2.7	0.008
Body dissatisfaction	0.0 (1)	-1.0 (1.0)	-3.1	.002
12 Years:				
BMI	19.2 (4.2)	20.3 (4.6)	-2.7	0.007
BMI z-score	0.6 (1.3)	0.7 (1.3)	-0.4	0.681
Eating disorder symptoms	6.5 (6.0)	6.0 (9.0)	-0.2	0.804
Depressive symptoms	1.0 (2.0)	2.0 (3.3)	-3.8	<.001
Body esteem	100.0 (5.6)	91.7 (25.7)	-5.5	<.001
Puberty	2.0 (0.8)	2.3 (0.9)	-4.3	<.001
15 Years:				
BMI	20.4 (4.5)	22.3 (5.1)	-4.1	<.001
BMI z-score	0.4 (1.0)	0.8 (1.5)	-2.3	0.021
Eating disorder symptoms	4.0 (4)	7.0 (9.0)	-5.2	<.001
Depressive symptoms	1.0 (2.0)	3.0 (6.0)	-7.2	<.001
Body esteem	100.0 (5.6)	83.3 (38.9)	-8.5	<.001
Puberty	3.0 (1.8)	3.9 (1.0)	-10.4	<.001

Boldface shows $p < .005$. IQR, interquartile range; BMI, Body Mass Index. Lower body esteem denotes higher body dissatisfaction.

Missing data was dealt with in the correlation and Mann-Whitney analyses using pairwise deletion, and in the path analysis using case-wise maximum likelihood estimation.

Girls had significantly higher depressive symptom scores than boys at ages 12 and 15, and significantly higher body dissatisfaction at 9, 12, and 15 years old. There was no significant difference in dietary restraint/eating disorder scores between boys and girls at 7, 9, and 12 years of age, but girls had significantly higher scores at age 15. Girls were significantly more advanced in pubertal development than boys at ages 12 and 15.

Zero-order non-parametric correlations (Spearman's rho) for each variable at ages 7, 9, 12, and 15 are reported in the Supplementary Materials 1 (Table S2). For boys, eating disorder symptoms at 15 were associated with higher body dissatisfaction at 15, and positively associated with depressive symptoms. For girls, eating disorder symptoms at 15 were correlated positively with BMI z-score at 7, disordered eating at 9, BMI z-score, depressive symptoms and disordered eating at 12, and depressive symptoms and puberty at 15, and with greater body dissatisfaction (more negative scores) at both 12 and 15.

3.2. Path analysis for eating disorder symptoms at 15 years old in girls

3.2.1. Fit indices

Goodness-of-fit indices were reported for each model. As suggested by Hu and Bentler (1999) and Iacobucci (2010) we included: the χ^2 and its corresponding df ; the standardised root mean square residual (SRMR); the comparative fit index (CFI); the Tucker-Lewis Index (TLI); and the root mean squared error of approximation (RMSEA). For χ^2 , a non-significant χ^2 value ($p > .05$), for CFI and TLI a value of >0.95 , for SRMR a value of <0.08 , and for RMSEA a value of <0.06 would indicate good model fit.

The full models were intended to test theoretically-derived hypothesised paths in the longitudinal development of eating disorder symptoms from childhood to middle adolescence. We were interested in both significant and non-significant paths. As such, the models were overfitted and goodness of fit measures were not expected to be ideal. Best fit models can be found in Supplementary Materials 1 (Table S3 and Table S4).

3.2.2. Model comparison

Table 2 shows the fit indices for each model. Model 1 shows the full model for girls using data from ages 7–15. The fit indices for Model 1 (Fig. 2 Panel A) indicated that the model was not an optimal fit for the data as the χ^2 was significant, and the SRMR, TLI, CFI, and RMSEA are not within acceptable ranges. Model 2 (Fig. 2 Panel B) consisted of Model 1 with paths where p was >0.100 removed. This remained a suboptimal fit for the data. Path estimates and standard deviations can be found in the Supplementary Materials 2.

Therefore, in the final model (Model 2), we found that higher BMI z-score at 7 significantly predicted increased BMI z-score and higher body dissatisfaction at 9, and covaried negatively with body dissatisfaction at 7 (indicating more desire for a thinner body). At 9 years, BMI z-score predicted more advanced puberty, increased BMI z-score, and increased body dissatisfaction at 12 and covaried with body dissatisfaction at 9 (indicating more desire for a thinner body). Furthermore, eating disorder symptoms at 9 significantly predicted increased eating disorder symptoms at 12. At 12 years, higher BMI z-score predicted higher BMI z-score and increased body dissatisfaction at 15; body dissatisfaction scores at 12 predicted higher body dissatisfaction scores and higher depression scores at 15; higher depression scores at 12 predicted higher depression scores and more eating disorder symptoms at 15; and higher eating disorder symptom scores at 12 predicted higher disordered eating symptom scores at 15. Furthermore at 12, puberty covaried positively with BMI z-score, which covaried negatively with body esteem, which in turn covaried negatively with depressive symptoms. Depressive symptoms positively covaried with eating disorder symptoms although not significantly so. At 15 years, only body dissatisfaction covaried with depressive symptoms.

3.3. Depressive symptoms as a mediator of the relationship between body dissatisfaction and disordered eating symptoms in girls

To test the hypothesised mediation effects of depressive symptoms at 12 between body dissatisfaction at 12 and disordered eating at 15 in girls, we conducted a mediation analysis with depressive symptoms at 12 as the mediator. A Sobel test indicated a significant mediation effect ($z = -3.22$, $p = .001$), suggesting that depressive symptoms at 12 significantly, but partially, mediated the relationship between body dissatisfaction at 12 years and eating disorder symptoms at 15 years in girls (see Fig. 3).

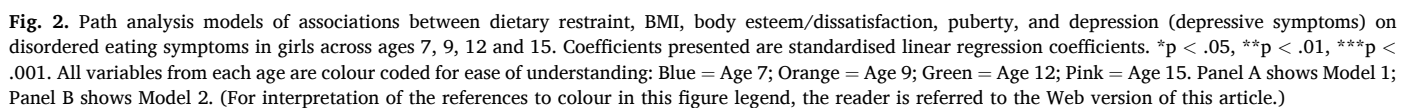
3.4. Path analysis for eating disorder symptoms at 15 years old in boys

Goodness-of-fit indices were calculated in the same way as for the

Table 2
Fit indices for the two models in girls.

	χ^2	df	p-value (Chi-Squared)	SRMR	CFI	TLI	RMSEA
Model 1	287.28	70	<0.001	0.131	0.855	0.787	0.129
Model 2	284.79	69	<0.001	0.148	0.857	0.811	0.129

Notes. SRMR = standardised root mean square residual; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation. Model 1 = original model; Model 2 = Model 1 with paths where $p > .100$ removed.



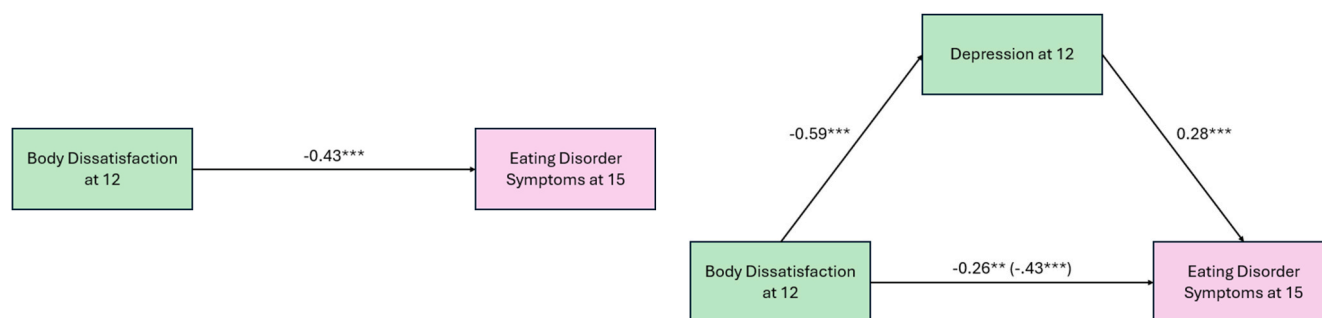


Fig. 3. Mediation model showing the relationship between body esteem at 12 and eating disorder symptoms at 15 with the depressive symptoms at 12 as the mediator. Coefficients presented are standardised linear regression coefficients. Coefficients in parentheses represent the direct, unmediated effect. * $p < .05$, ** $p < .01$, *** $p < .001$.

girls.

Table 3 shows the fit indices for each model. Model 3 (Fig. 4 Panel A) shows the full model for boys using data from ages 7–15. The fit indices for Model 3 indicated that the model was not an optimal fit for the data as the X^2 was significant, and the SRMR, TLI, CFI, and RMSEA are not within acceptable ranges. Model 4 (Fig. 4 Panel B) consisted of Model 3 with the paths where p was above 0.100 were removed and was a better though not perfect fit for the data as CFI was within the acceptable range ($CFI > 0.95$), and TLI and RMSEA are very close to optimal range (TLI > 0.95 ; RMSEA < 0.06). Path estimates and standard deviations can be found in the Supplementary Materials 2.

Therefore, in the final model (Model 4), we found that higher BMI z-score at 7 significantly predicted increased BMI z-score and higher body dissatisfaction at 9, and covaried negatively with body dissatisfaction at 7 (indicating more desire for a thinner body). At 9 years, BMI z-score predicted more advanced puberty and increased BMI z-score, and covaried negatively with body dissatisfaction at 9 (indicating more desire for a thinner body). High body dissatisfaction at 9 was non-significantly predictive of higher depressive symptoms at 12, and significantly covaried with higher eating disorder symptoms at 9. Furthermore, more eating disorder symptoms at 9 significantly predicted increased eating disorder symptoms at 12. At 12 years, higher BMI z-score predicted higher BMI z-score at 15; body dissatisfaction scores at 12 predicted higher body dissatisfaction scores at 15; higher depressive symptom scores at 12 predicted higher depressive symptoms scores and non-significantly predicted more eating disorder symptoms at 15; and higher eating disorder symptoms at 12 non-significantly predicted higher disordered eating symptoms at 15. Furthermore at 12, body dissatisfaction covaried with depression. At 15 years, BMI z-score covaried with body dissatisfaction, which covaried positively with depressive symptoms, which in turn covaried positively with disordered eating symptoms.

4. Discussion

The aim of this study was to examine longitudinal predictors of eating disorder symptoms in a cohort of 15-year-olds using concurrent

Table 3
Fit indices for the two models in boys.

	X^2	df	p-value (Chi-Squared)	SRMR	CFI	TLI	RMSEA
Model 3	165.75	70	<0.001	0.092	0.910	0.867	0.088
Model 4	148.74	87	<0.001	0.091	0.935	0.921	0.070

Notes. SRMR = standardised root mean square residual; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation. Model 3 = original model; Model 4 = Model 3 with paths where $p > .100$ removed.

data and that gathered previously at ages 7, 9, and 12. We examined whether a theoretical model of eating disorder symptom development (the Dual Pathway model) reflected the prospective and within-time relationships observed in the data.

We hypothesised (H1) that higher BMI would predict higher body dissatisfaction at the subsequent timepoint. This hypothesis was fully supported at every age in girls, but only for 7-year BMI z-score and 9-year body dissatisfaction in boys. This finding fits with previous research showing that adiposity more strongly predicts body dissatisfaction in girls than boys (Blundell et al., 2024; Lewis-Smith et al., 2020), potentially due to diverging body size ideals of low adiposity for girls and high muscularity for boys (Murray et al., 2017) and greater sociocultural pressures towards thinness in girls. Gender differences with age likely also reflect both a more deleterious effect of weight stigma and teasing in older girls (Jendryca & Warschburger, 2016) and, potentially, a curvilinear relationship between BMI and body dissatisfaction in boys rather than the linear relationship seen in girls (Calzo et al., 2012).

Based on the Dual Pathway model, we hypothesised (H2) that higher body dissatisfaction would predict higher eating disorder symptoms at the subsequent timepoint, mediated by depressive symptoms. In girls, we indeed found that 12-year depressive symptoms partially mediated the prospective relationship between 12-year body dissatisfaction and 15-year eating disorder symptoms. This was not found at 9- or 12 years, or with boys, when body dissatisfaction instead covaried with eating disorder symptoms. This partially replicates what has been found previously: body dissatisfaction covaries with disordered eating in pre-adolescence (Allen et al., 2008; Forrester-Knauss et al., 2012) but becomes a longitudinal predictor only in middle-adolescence (Ferreiro et al., 2012; Martini et al., 2023; Rohde et al., 2015). Furthermore, had we measured dietary restraint past age 7, we may have found earlier predictive effects of body dissatisfaction as evidence suggests that pre-adolescent body dissatisfaction predicts future dietary restraint but not eating disorder symptoms (Allen et al., 2008; Forrester-Knauss et al., 2012). Alternatively, previous research notes fluctuations in body dissatisfaction throughout adolescence (Lacroix et al., 2023; Nelson et al., 2018; Wang et al., 2019), stabilising in late adolescence to early adulthood (Lacroix et al., 2022; Wang et al., 2019). Its variability may feasibly explain why it is not a consistent longitudinal predictor until adolescence.

Besides acting as a mediator, we further hypothesised (H3) that higher depressive symptoms would directly prospectively predict disordered eating symptoms. Our findings supported this: girls' 12-year depressive symptoms significantly predicted 15-year eating disorder symptoms. In boys, 12-year depressive symptoms were not associated with concurrent disordered eating and marginally non-significantly predicted 15-year disordered eating. These findings are in keeping with some previous research in this age group showing a significant relationship in girls but not boys (Ferreiro et al., 2012), but not with

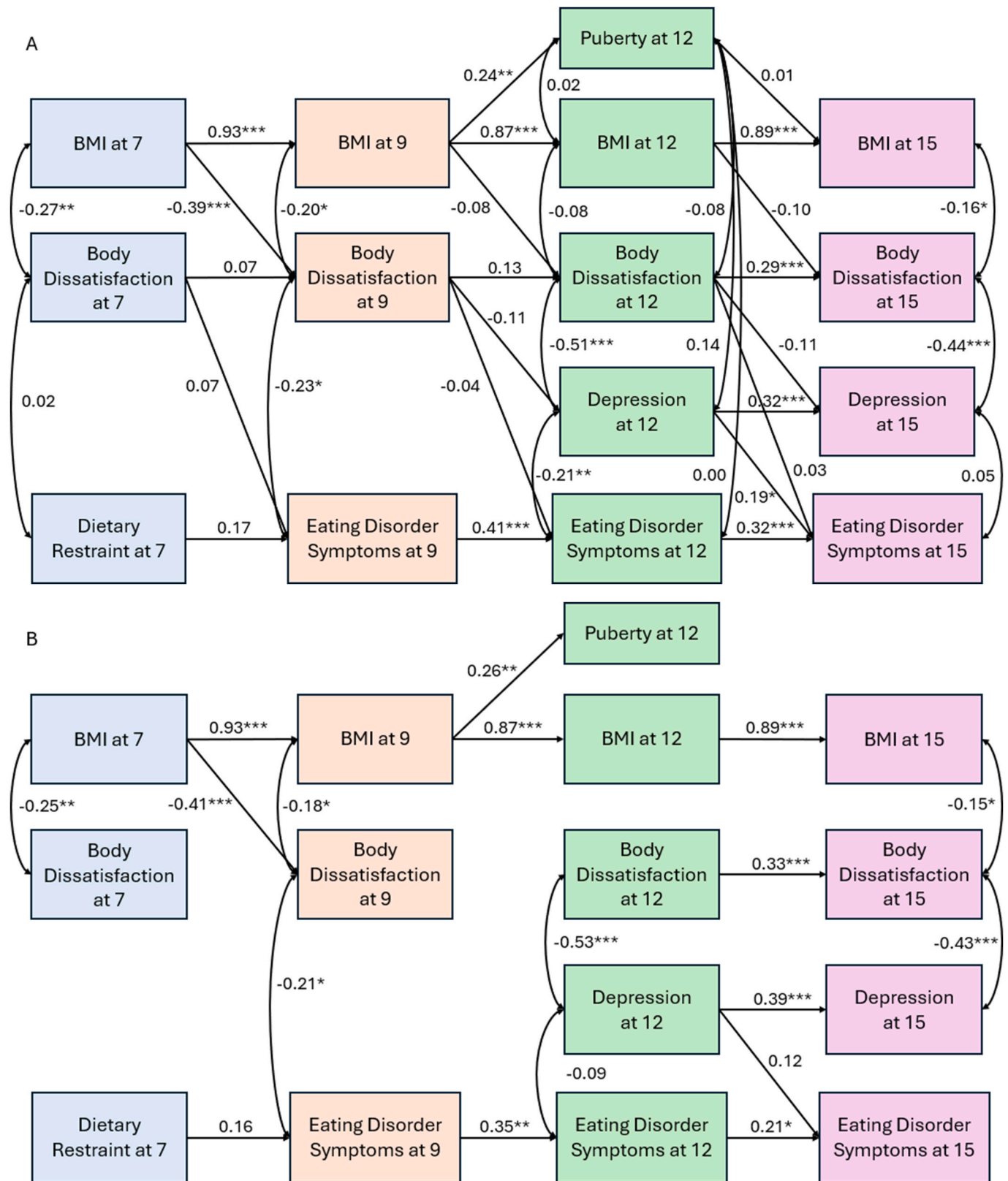


Fig. 4. Path analysis models of associations between dietary restraint, BMI, body esteem/dissatisfaction, puberty, and depressive symptoms on disordered eating symptoms in boys across ages 7, 9, 12 and 15. Coefficients presented are standardised linear regression coefficients. * $p < .05$, ** $p < .01$, *** $p < .001$. Panel A shows Model 3; Panel B shows Model 4.

Lewis-Smith et al. (2020) which showed the relationship in both groups albeit less strongly for boys. Our results also fit with literature with older adolescents demonstrating longitudinal relationships between depressive symptoms and eating difficulties in girls but not boys (e.g., Allen et al., 2013).

Finally, we hypothesised (H4) that pubertal development would covary with eating disorder symptoms for girls, but not boys. The results supported this - girls with more advanced pubertal development at 12 had higher concurrent eating disorder symptoms - and showed coherence with the existing literature (Le Grange et al., 2014; Vo et al., 2021). Although we did not hypothesise this, previous research shows associations between pubertal development and both body dissatisfaction and depressive symptoms (Dantas et al., 2017; Thériault et al., 2019; van Rijn et al., 2023), which we did not find. However, our models included BMI z-score which appears to account for these relationships (Dantas et al., 2017).

Considering the model overall, the strongest predictor of 15-year disordered eating symptoms was 12-year eating disorder symptoms. This fits with previous publications with this cohort (Evans et al., 2017) and other research (Abebe et al., 2014; Neumark-Sztainer et al., 2007), and strongly suggests that early prevention of eating disorder symptoms is critical to long-term reductions. We did not directly test sex differences because our hypotheses were already partially sex-differentiated based on theory, so we conducted separate analyses. As expected, our results suggest key differences in the pathways that predict 15-year outcomes. We furthermore found that each variable at 12 years old autocorrelated with itself at 15 for both girls and boys, but only prospectively predicted another variable (BMI → body esteem; body esteem → depressive symptoms; depressive symptoms → eating disorder symptoms) in girls. This suggests that eating disorder symptoms in boys may be driven in part by variables not included in the current study. Compared to previous studies using the GMS cohort, we found that dietary restraint at 7 was *not* a significant prospective predictor of eating disorder symptoms at 9 or 12, as reported previously (Evans et al., 2017; Parkinson et al., 2012), although there was a trend towards significance in boys. This may be a consequence of the reduced sample size over time. All other previously reported relationships which were tested in the current model remained significant.

In the longitudinal models we tested, the 7- and 9-year-old data was not particularly longitudinally salient, with only BMI z-score at 9 in girls predicting variables other than itself. This may indicate that variables at 7- or 9-years-old may be more relevant to maintaining symptoms and/or maintaining those other variables which then become risk factors for disordered eating around age 12. That is to say, predictors of eating disorders in pre-pubertal periods may be important because they predict concurrent symptoms rather than future risk.

4.1. Study strengths, limitations and directions for future research

The current study presents a rare and useful dataset in our understanding of the development of disordered eating through from mid-childhood and adolescence. Longitudinal research is vital in identifying potential causal pathways across time and the current study tracked a group of children across a critical period for the emergence of eating disorders. This allowed us to see that longitudinal and within-time relationships between different components of the dual pathway model may come 'online' at different ages. Of course, by spanning this age range, it was not possible to use identical measures at each time point; body dissatisfaction was measured using a figure choice scale at age 7 and 9 and a questionnaire at 12 and 15. Disordered eating was only measured from age 9 onwards, with dietary restraint measured at the 7-year time point. Depressive symptoms and puberty were not measured before the 12-year time point; measuring puberty at 9 would be unlikely to reveal sufficient variance for analyses so a recommendation for future research would be to collect annual data across the 10–13 year age range to examine more closely the impacts of puberty during the key years for

its onset. Similarly, including depressive symptoms from an earlier age would allow us to assess when depressive symptoms become a longitudinally salient predictor of future disordered eating. Future longitudinal research should consider how sociocultural factors in the earlier steps of the dual-pathway model, such as sociocultural pressures, or internalisation of body ideals predict disordered eating across childhood and adolescence.

Finally we acknowledge that by 15-years-old, we had 36 % of the original sample remaining in the study – an attrition rate that is not unreasonable over 15 years, but does mean that we did not have analytic power to look at models with multiple interaction effects (for instance, examining moderating effects of gender on each pathway) (Devlieger & Rosseel, 2017). With a relatively simple model such as ours, and almost 200 participants per sample, the models should be robust enough for the purposes of the current study (Boomsma, 1985). Moreover, many of our (pre-15-year) findings are consistent with those reported in previous papers, for which a greater proportion of the original sample remained. However, reduced samples size should be considered a key limitation of this study, and any non-significant findings should be interpreted with caution.

4.2. Conclusions

In conclusion, we investigated pathways to disordered eating at 15 separately in boys and girls. Our results allowed us to consider multiple putative predictors, measured at multiple timepoints, in children from ages 7 to 15. This enabled the testing of a theoretically-driven prospective model of eating disorder symptom development and showed differing pathways by gender for the development and maintenance of disordered eating. Our study adds to the growing body of literature investigating how eating disorder symptoms develop throughout childhood and adolescence, and what factors may increase risk of eating disorder development.

CRedit authorship contribution statement

Louise N. Hanson: Writing – original draft, Formal analysis. **Ashley J. Adamson:** Writing – review & editing, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **Laura Basterfield:** Writing – review & editing, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **John J. Reilly:** Writing – review & editing, Methodology, Funding acquisition, Conceptualization. **Xanne Janssen:** Writing – review & editing, Methodology. **Mark S. Pearce:** Writing – review & editing, Methodology, Funding acquisition, Conceptualization. **Lynda G. Boothroyd:** Writing – review & editing, Supervision. **Elizabeth H. Evans:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization.

Ethical statement

Ethical approval was granted by Gateshead and South Tyneside Local Research Ethics Committee (7 year follow up) and by Newcastle University Research Ethics Committee (9-year, 12-year, and 15-year follow ups).

This work was carried out in accordance with the World Medical Association Declaration of Helsinki. Individuals' privacy rights have been observed and informed consent was obtained from parents of participants prior to study participation.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.appet.2025.108149>.

Data availability

Data will be made available on request.

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