ORIGINAL ARTICLE

Childhood Body Mass Index in Adolescent-Onset Anorexia Nervosa

Staci A. Berkowitz, MS[1]
Ashley A. Witt, PhD[1]
Christopher Gillberg, MD, PhD[2]
Maria Råstam, MD, PhD[3]
Elisabet Wentz, MD, PhD[2]
Michael R. Lowe, PhD[1,4]*

ABSTRACT

Objective: Although weight history is relevant in predicting eating disorder symptom severity, little is known about its role in the etiology of anorexia nervosa (AN). This study aimed to determine whether BMI or BMI trajectory differed between individuals who later developed adolescent-onset AN and a comparison group of HCs between school grades 1 through 6.

Method: This study was based on longitudinal data that identified 51 adolescents with AN and 51 matched HCs. Cases were identified through community screening in Sweden and included individuals born in 1969 through 1977. Measured weights and heights were retrieved and BMIs and weight trajectories of the AN and HC groups were compared using growth curve analysis. Main outcome measures included measured BMI and BMI trajectories from grades 1–6. Secondary outcomes examined included ponderal index at birth and maternal body weight.

Results: Individuals who later developed AN had higher BMIs than HCs between grades 1 and 6, by an average of 1.42 BMI-units. There was no difference in rate of weight gain between groups. Ponderal index at birth was higher for the AN as compared with HC group. Maternal weight did not differ significantly between groups.

Discussion: These findings, combined with those previously reported on the premorbid BMIs of those with bulimia nervosa, suggest that a predisposition toward elevated premorbid BMIs during childhood characterizes those who later develop anorexia or bulimia nervosa. These findings are consistent with a transdiagnostic perspective and suggest shared risk factors for AN and obesity. © 2016 Wiley Periodicals, Inc.

Keywords: anorexia nervosa; body mass index; development; BMI; weight; childhood; adolescent; eating disorders; adolescence

(Int J Eat Disord 2016; 49:1002–1009)

Introduction

Anorexia nervosa (AN) is a serious psychiatric illness characterized by a severe disturbance in the way body shape and weight are experienced and maintenance of a low body weight relative to that expected based on age and height. A growing body of research highlights the importance of the relation between an individual's historical highest and current body weights (weight suppression; WS) for a number of eating disorder outcomes. In the context of children and adolescents, WS has been conceptualized as a change in BMI units rather than in weight per se, given that these individuals may continue to grow in stature.[1] (Assessing WS in those who developed AN prior to reaching their adult height is challenging, a topic that is considered further in the Discussion.) Individuals higher in WS have displayed more severe eating disorder symptomatology prior to and following treatment and have taken longer to recover overall.[2–6] WS has also been associated with future weight gain, an outcome that individuals with eating disorders fear greatly.[1,2,4,5,7–9] In sum, historical body weight, in relation to current weight, is a robust predictor of current and longer-term eating disorder symptomatology and course.

Interestingly, although weight history seems relevant in predicting eating disorder symptoms and markedly low body weight is a striking feature of AN, little is known about the role of weight history in the etiology of AN. Only two published studies

Accepted 14 June 2016
*Correspondence to: M. R. Lowe; E-mail: ml42@drexel.edu
[1] Department of Psychology, Drexel University, Philadelphia, Pennsylvania
[2] Gillberg Neuropsychiatry Center, Institute of Neuroscience and Physiology, University of Göteborg, Sweden
[3] Department of Clinical Sciences, Lund University, Sweden
[4] The Renfrew Center for Eating Disorders, Philadelphia, Pennsylvania
Published online 27 July 2016 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/eat.22584

have reported on measured premorbid BMI among individuals with AN. A previous report from the current sample indicated that highest premorbid BMI for individuals with AN was approximately two BMI-units higher than BMI of an age-matched peer group, representing a large effect size.[10] A major limitation of this study was that there was no consideration of whether the elevated BMIs identified were transitory—occurring just prior to AN onset—or whether they reflected a longer-lasting developmental pattern that preceded AN onset. Thus, it is not clear whether individuals with AN reached BMIs that were on average higher than their peers at a young age, or just prior to AN onset. Additionally, Nielsen[11] evaluated weight status across development, measuring childhood weight status among individuals who later developed AN. Nielsen reported that childhood weight status was elevated among AN patients referred for treatment, as compared with published weight norms. These findings are intriguing and potentially important, as they identify childhood weight status as a putative risk factor for AN. However, the inclusion of only treatment-seeking AN individuals and the reliance on population weight norms—rather than a control group also assessed during childhood—limits the interpretability of the finding. Provided that body weight norms have been shown to differ by region, time period, and peer group,[12,13] this finding should be reexamined with a control group matched on these variables. This study addressed these limitations.

In addition, since these two past studies[10,11] were published over 20 years ago, no literature has further evaluated premorbid weight status in AN. Since then, elevated premorbid weights have been documented as a risk factor for BN.[14,15] Although premorbid weight status may also represent a risk factor for AN, conceptualizations of the etiology of AN published in recent years have ascribed little, if any, significance to this factor. Furthermore, the fact that there now exists substantial cross-sectional and prospective data indicating that WS predicts the maintenance or emergence of eating disorder characteristics in those with AN or BN, the field could benefit from research that highlights the relevance of past weight status in disordered eating.

The objective of this study was to determine whether BMI or weight trajectory during childhood differed between individuals who later developed adolescent-onset AN and a comparison group of healthy controls (HCs) matched on age, sex, grade-level, and school in a community sample; the AN sample included individuals who had never received eating disorder treatment (about 50%) or consultation (about 35%) at the time of enrollment. Secondarily, this study aimed to determine whether weight status at birth or maternal body weight differed between the groups. The data set examined provides a unique opportunity to evaluate longitudinal changes in body weight across development in an eating disordered and noneating disordered sample. To our knowledge, this data set represents the only case-controlled, prospective community-based sample of adolescent-onset AN.[16]

Methods

The Original Study

AN Group. In a prospective, longitudinal study, 51 individuals with adolescent-onset AN (48 females, 3 males) were recruited and followed over time. Half of this sample—the population group—consisted of all of the individuals living in Göteborg, Sweden in 1985 who were born in the year 1970 and met criteria for AN by the age of 18. In total, 4,291 individuals born in 1970 and living in Göteborg in 1985 were screened for AN (details on this procedure have been reported previously[17]) and the population Group (22 females, 2 males) is thought to constitute the entire Göteborg population of individuals with adolescent-onset AN born in that year (excluding one female individual who chose not to participate). The other half of the sample—the population-screening group—consisted of individuals with adolescent-onset AN attending the same Göteborg schools as the other participants, who were born in 1969 through 1977. Participants in the population-screening Group (26 females, 1 male) were identified to the research team at the same time as participants from the other group. Medical, psychological, and neuropsychological characteristics and outcomes in the total group of 51 participants have been reported in detail elsewhere.[18]

Participants were typically in the ninth or tenth grade at the time of assessment by research personnel, with a mean age of 16.1 years (SD = 1.4). Participant diagnosis included physical examination and review of growth charts kept by school nurses, with height and weight data documented at regular intervals from the first grade onward. Participant diagnosis was established following a thorough semi-structured clinical interview with the adolescent and with a parent to assess DSM-III-R criteria for AN. Interviews were administered by a psychiatrist (author MR) who gathered information on the participant's experience of symptoms and developmental history, lasting a minimum of 2 hours. Individuals in the AN group met DSM-III-R criteria for AN.

The two groups, the population group and population-screening group, were indistinguishable in terms of participant characteristics, except in regard to the number of

TABLE 1. AN sample characteristics

Variable	Minimum	Maximum	*M* (SD)
Age of AN onset	10.0	17.2	14.3 (1.6)
BMI at highest premorbid weight	15.5	30.9	21.1 (3.3)
Age at highest premorbid weight	9.8	16.6	13.9 (1.5)
BMI at lowest weight	9.5	18.7	15.0 (2.3)
Age at lowest weight	10.5	18.7	15.3 (1.7)

Notes: AN, anorexia nervosa; BMI, body mass index; *M*, mean; SD, standard deviation.

participants with a previously identified eating disorder, with 52% of the population group and 22% of the population-screening group having never received eating disorder consultation.[19] The two groups were pooled to form the current sample of 51 AN participants, all of whom met DSM-III-R (and, upon later review, DSM-IV) criteria for AN.

Sample characteristics of the AN group are presented in Table 1. Terms associated with this table are defined below:

Age of AN onset. This variable represents the best estimate of the age at which an individual experienced first symptoms of restrictive eating resulting in weight loss associated with AN. This age was determined based on information gathered during the assessment process.

BMI at highest premorbid weight. This value represents an individual's BMI at the time that highest body weight prior to AN onset was recorded. This value was determined by review of nurse charts and medical records of weight and height prior to illness onset.

Age at highest premorbid weight. This value represents the age at which the highest body weight prior to AN onset was recorded. This value is necessarily lower than age at AN onset.

BMI at lowest weight. This value represents an individual's BMI at the time that lowest body weight was recorded during the first episode of AN. This value was determined by review of nurse charts and medical records of weight and height following initial illness onset.

Age at lowest weight. This value represents the age at which lowest body weight during first episode of AN was reached. This value is necessarily higher than age of AN onset.

Matched HC Group. A comparison group of 51 healthy individuals (48 females, 3 males) who were age-, sex-, and school-matched with participants in the AN group were also included. Individuals in the HC group were selected to have birthdates closest to that of their matched HC counterpart, within the same school. In the case of one participant, the comparison case had to be selected from another school. Participants in the HC condition were physically and psychiatrically screened, and a parent for each individual was interviewed, in the same manner as individuals in the AN group (Note: participants participated in follow up studies 6, 10, and 18 years after initial assessment.[18,19,21] No individuals in the HC group were diagnosed with AN at any point during this follow-up period.)

TABLE 2. Weight status in AN and HC groups

	AN Group			HC Group		
Variable	*N*	*M* (SD)	*Z*-score	*N*	*M (SD)*	*Z-score*
Grade 1 BMI	46	16.87 (2.15)	0.74	50	15.39 (1.04)	−0.22
Grade 2 BMI	37	17.62 (2.21)	0.92	39	15.96 (1.37)	−0.04
Grade 4 BMI	48	18.08 (2.65)	0.50	49	16.45 (1.63)	−0.24
Grade 6 BMI[a]	34	19.63 (2.72)	0.57	46	18.03 (2.00)	−0.06
Birth ponderal index	47	27.72 (2.18)		48	26.74 (2.25)	
Maternal weight (kg)	43	59.40 (9.08)		46	56.92 (9.66)	

Notes: AN, anorexia nervosa group; HC, healthy control group; BMI, Body Mass Index; g, grams; kg, kilograms; *M*, mean; SD, standard deviation.

[a]Participants with AN onset prior to time point were not included. BMI *Z*-scores were calculated based on population reference values for children from Göteborg, Sweden.[20]

This Study. As part of this study, weight and height data from school nurse charts and medical records were assessed for all 102 (51 AN, 51 HC) participants. Weight and height data were available for each participant at school grades 1 through 6, though data was not available from each participant at all time points. We decided to calculate BMI only for school grades with data available from the majority (at least 75%) of participants, and to exclude school grades with data available from fewer than 20% of participants (school grades 3 and 5). Therefore, BMI was calculated at the following time points: school grades 1, 2, 4, and 6 (ages 7, 8, 10, and 12; see Table 2). BMI was calculated as kilograms per meters squared. BMI *z*-scores were also calculated based on Göteborg, Sweden population norms[20] (see Table 2). For main analyses, we chose to report BMI, rather than a standardized weight measure (e.g., BMI percentile; BMI *z*-scores) for ease of interpretation and because participants' weights were grouped by age for analytic purposes. However, main analyses were also carried out with *z*-scores.

We chose to include both males and females in this study, in spite of the small number of males, to be inclusive of all individuals with AN. Although AN more commonly affects females, both males and females are influenced by the disorder. Our sample is reasonably representative of AN within the community—with the "population group" reflective of the true proportions of males and females and a somewhat less comprehensive "population screening group," which includes an estimated 60% of individuals with AN born in years adjacent to 1970 in the same schools.[18] Because it is reasonable to expect that childhood weight trajectories to differ

between males and females, we re-ran main analyses with only females. The limited number of males in the study did not allow for male-only analysis.

For supplemental analyses, birth weight and length data from medical records was collected. We computed ponderal index (kg/m^3) at birth, a standard approach used for reporting body mass in newborns. Furthermore, maternal weights recorded in birth records were collected. Maternal weight referred to body weight just before the onset of pregnancy, based on self-report. Because maternal height data was not available for the majority of participants, maternal BMI was not calculated.

Data Analytic Strategy

Primary Analyses. Growth curve analysis,[22] a multilevel/hierarchical linear modeling method for longitudinal data, was used to compare BMI over time between first and sixth grade between the AN and HC groups. Curves were modeled with second-order orthogonal polynomials and fixed effects of diagnostic group (AN; HC) on all time terms. (We decided to model our data using second-order orthogonal polynomials (i.e., a quadratic model) because change in BMI during childhood is not expected to be linear, but rather standard medical growth charts suggest that a curvilinear function best represents such changes over time.) The fixed effects of diagnostic group were added individually and their effects on model fit were evaluated using model comparisons techniques for nested model. Improvements in model fit were evaluated by using -2 times the change in log-likelihood between the nested model, which is distributed as χ^2 with degrees of freedom representing number of parameters added to the more complex model of the two. Random effects at the matched pair level (i.e., based on each AN-HC pair matched on age, sex, and school) were also included in all time terms and diagnosis was treated as a within-matched pair variable. Because we were interested in modeling BMI over time prior to AN onset, BMI was not analyzed for participants in the AN group following illness onset. Thus, we excluded the grade 6 BMI value for four of the 51 individuals in the AN group with illness onset prior to grade 6 (but after grade 4); the data for these participants from earlier time points was still included in the model.

To confirm that the pattern of findings was not specific to the data analytic methods employed, several variations of our main statistical analysis were carried out. First, we ran analyses again only using data collected between grades 1 and 4; this allowed us to evaluate whether findings were driven by the exclusion of four individuals with AN at the grade 6 time point. Because the grade 1 through 4 time frame included only three time points, we were only able to look at a linear model and not a curvilinear function for this analysis. We also ran each of these models again including only females, and using BMI *z*-scores rather than BMI. Additionally, one-sample *t*-tests were use to compare BMI z-scores from the AN group to normative BMI data. These analyses were completed using R version 3.0.1 using the lme4 package.

TABLE 3. Correlations between weight status variables

Variable	1	2	3	4	5	6
1. Birth Ponderal Index	–					
2. Maternal weight (kg)	0.10	–				
3. Grade 1 BMI	0.24[b]	0.22[b]	–			
4. Grade 2 BMI	0.42[c]	0.23	0.92[c]	–		
5. Grade 4 BMI	0.30[c]	0.27[b]	0.83[c]	0.90[c]	–	
6. Grade 6 BMI[a]	0.39[c]	0.25[b]	0.78[c]	0.81[c]	0.86[c]	–

Notes: BMI, body mass index.
[a]Participants with AN onset prior to time point were not included.
[b]Denotes statistical significance at $p < .05$ threshold.
[c]Denotes statistical significance at $p < .01$ threshold.

Supplemental Analyses. Because group differences were found starting at grade 1, we conducted additional supplemental analyses to determine whether differences in weight status at birth (as measured by weight and ponderal index) or mothers' body weight were different between the AN and HC groups (see Table 2). This allowed us to determine whether differences in relative weight could be traced further back in time. Independent samples *t*-tests were used to compare group differences for ponderal index and maternal weight. A correlation matrix of weight status variables is presented in Table 3 to further explore relationships between variables. These analyses were completed using SPSS statistical software.

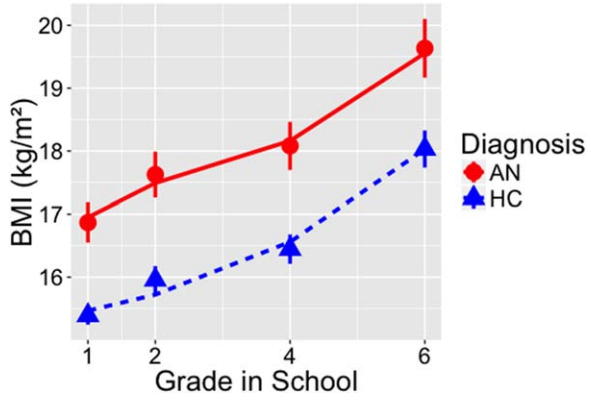
Results

Descriptive statistics characterizing the AN sample are shown in **Table 1**. Participants were typically in the ninth or tenth grade at the time of assessment by research staff, with a mean age of 16.1 years (SD = 1.4).

Primary Analyses

Both linear and quadratic functions of time significantly predicted the overall change in BMI between grades 1 and 6 for all participants (linear: estimate = 2.09, SE = 0.14, $p < 0.001$; quadratic: estimate = 0.41, SE = 0.09, $p < 0.001$) yielding a curvilinear profile of change over time with the unit of time representing one grade year. The effect of diagnosis (AN; HC) on the intercept significantly improved model fit (χ^2 (1) = 16.95, $p < 0.001$), suggesting individuals who later developed AN had higher BMIs than HCs by an average of 1.42 BMI-units (estimate = 1.42, SE = 0.33, $p < 0.001$). However, the effect of diagnosis on linear time (χ^2

FIGURE 1. Childhood Body Mass Index Over Time for Individuals who Later Develop AN and HCs: Observed Data and Model Fit. AN: anorexia nervosa; HC: healthy control; BMI: body mass index; for the purpose of illustration, observed data, and model fit are based on the main analysis evaluating BMI from grade 1 through grade 6. Individuals who later developed AN had higher BMIs than HCs by an average of 1.42 BMI-units (estimate = 1.42, SE = 0.33, $p < .001$). [Color figure can be viewed at wileyonlinelibrary.com]



(1) = 1.27, $p = 0.26$) and quadratic time (χ^2 (1) = 0.25, $p = 0.62$) were not significant, suggesting there was no difference in rate of weight gain between the groups between grades 1 and 6. This pattern was consistent when only grades 1–4 were considered [for the grade 1–4 analysis, a linear function of time appropriately modeled BMI over time for all participants (estimate: = 0.37, SE = 0.04, $p < 0.001$). The effect of diagnosis (AN; HC) on the intercept significantly improved model fit (χ2 (1) = 17.20, $p < 0.001$), suggesting individuals who later developed AN had higher BMIs than HCs by an average of 1.42 BMI-units (estimate = 1.42, SE = 0.33, $p < 0.001$). However, the effect of diagnosis on linear time (χ2 (1) = 0.40, $p = 0.53$) was not significant]. Both grade 1–6 and 1–4 results also held when only females were included in analysis. See **Fig. 1** for an illustration of observed BMI values over time and model fit. The pattern of findings (i.e., higher childhood BMI among the AN compared with HC group) was consistent when BMI *z*-scores were used, rather than BMI values. Furthermore, BMI values from the AN group were also significantly higher than BMI population norms for grades 1–6 (p's < 0.01); this difference was associated with a large effect size at each time point (see **Table 4**).

Supplemental Analyses

Birth and Maternal Weight Status. Ponderal index at birth was higher for the AN as compared with HC group, $t(93) = 2.15$, $p = 0.03$, Cohen's $d = 0.45$ (95% confidence interval: 0.03 − 0.85), suggesting a medium effect size in the difference between groups in ponderal index. There was also no statistically significant difference in maternal weight between AN and HC groups ($p = 0.22$).

TABLE 4. Body mass index in AN group compared with normative data

Variable	*t*	*df*	*p*	Cohen's *d*
Grade 1 BMI	3.54	45	0.001[b]	1.06
Grade 2 BMI	4.27	36	<0.001[b]	1.42
Grade 4 BMI	2.82	47	0.007[b]	0.82
Grade 6 BMI[a]	3.03	33	0.005[b]	1.05

Notes: BMI, body mass index.
[a]Participants with AN onset prior to time point were not included.
[b]Denotes statistical significance at $p < .01$ threshold.

Discussion

Longitudinal research is crucial for identifying factors associated with later development of AN; however, few prospective studies of this population have been conducted. The present study used a unique longitudinal dataset to investigate whether differences existed in early body weight or weight trajectories over time between individuals who later developed AN and HCs. These values were also compared to normative data. Results indicated that while BMI increased over time for all individuals, those who later developed AN were an average of 1.42 BMI units heavier than age-, sex-, and school-matched HCs across observation points; this BMI difference was detectable as early as the first grade (age 7). On average, those who developed AN also demonstrated elevated childhood BMI's relative to population norms, and these differences were associated with large effect sizes. Supplemental analyses indicated that ponderal index was significantly higher for the AN compared with HC group. This result may suggest that body mass differences observed during childhood may have existed earlier in life. Furthermore, maternal weight was not related to participant diagnostic status, although the lack of information on maternal height is a limitation of this analysis.

In addition to examining weight differences at each measurement point, we were also able to investigate differences in rate of weight gain over time. In our analyses from grades one through six, rate of weight gain did not differ between the groups. This pattern suggests that individuals in both groups showed similar rates of weight change throughout childhood, however individuals who later developed AN consistently showed higher BMIs than their peers, beginning in first grade.

For some individuals who went on to develop AN, higher weight status (in comparison to peers) was seen as early as birth, as measured by ponderal index (kg/m^3; a standard method for reporting body mass in newborns). Notably, the magnitude of the effect was smaller at birth than it was at the other time points. This suggest that some individuals who later developed AN experienced somewhat higher weight status as early as birth, though this difference stabilized and became more pronounced within this group by the age of 7. Relatedly, ponderal index at birth was correlated with later measures of childhood BMI, consistent with the notion that differences in BMI during childhood may, in some cases, be traceable to body mass differences detectable as early as birth.

The finding of elevated childhood BMI among individuals with AN is consistent with two other studies evaluating measured childhood weight status among eating disordered individuals. The first of these studies evaluated childhood weight status among AN individuals, similarly describing a tendency towards higher childhood body weight for height as compared with the population average.[11] The second study evaluated childhood weight status among a group of individuals with mixed eating disorder diagnoses, also reporting higher BMI as compared with the population average.[14] While these prior studies were completed in clinically referred samples, without a HC group, this study is the first to use a comprehensive community screening sample of individuals with AN and to employ a matched, HC group, as well as comparisons to population norms, strengthening the interpretation of findings.

Furthermore, elevated childhood BMI and ponderal index at birth among individuals with AN is intriguing in light of similar findings among those who later develop obesity or BN. Longitudinal research indicates that children at risk for later development of obesity show elevated BMI relative to peers by age 7 and elevated ponderal index at birth.[15] There is also evidence that overweight and obesity during childhood are associated with greater risk for BN.[23–25] As has been proposed for BN,[25] the present findings raise the possibility that disordered eating behaviors in AN may occur not just in response to distortions in body image, but also in response to actual elevations in BMI relative to peers starting at an early age. In fact, recent analyses of genome-wide association study data found a negative genetic correlation between obesity and AN, suggesting a shared genetic basis for factors influencing these two phenotypes.[26] It may be that a biological risk factor (elevated BMI) and a psychological risk factor (over-importance of weight and shape in one's self-concept[27]) combine to sharply elevate the risk for developing an eating disorder relative to peers with neither of these risk factors.

These finding of elevated childhood BMI among AN individuals in combination with recent reports on the predictive utility of WS on eating disorder symptomatology and recovery[4–6] is noteworthy. While BMI is typically used to describe an individual's body mass in absolute terms, WS is used ideographically to capture relative changes in body weight since achieving adult height. Within the context of a growing child or adolescent, the consideration of WS is complicated when a case of AN begins before adult height is reached. Furthermore, in such individuals, height may be stunted as a result of eating disorder onset during childhood or early adolescence. Some adjustments have been made to the calculation of WS in adolescent populations, such that WS has been evaluated as a change in age-adjusted BMI, rather than a change in weight,[1] addressing some (but not all) of these limitations. A key take away from the current paper is that, even when AN develops before adult height is reached, it may develop against a background of childhood BMIs that are above-average. This suggests that most AN individuals are at a low weight not only nomothetically (relative to population norms) but also idiographically (relative to their own elevated premorbid BMI). Thus, it seems that not only is weight status meaningful in predicting outcomes among individuals who have already developed eating disorders, but that measures of childhood absolute body weight may also help us understand who may be at increased risk for developing an eating and/or weight disorder.

Notably, although childhood overweight status has been associated with an increased risk for eating disorders, the prevalence of childhood overweight is much higher than the prevalence of AN, so most overweight children do not go on to develop AN. Thus, it is not childhood weight status in isolation, but perhaps childhood weight status in combination with other psychological variables that may contribute to eating disorder etiology and maintenance.

Furthermore, the timing of puberty has previously been identified as a risk factor for a range of psychopathology, with earlier compared to later pubertal onset among girls typically associated with greater risk.[28,29] Several studies have identified a relationship between early pubertal timing and eating disorder psychopathology more broadly;

however, there does not seem to be a link between early puberty and AN in particular.[30–33] Because higher body weight among girls is associated with earlier menarche,[20] it will be important for future studies to tease apart the potential role of each of these factors (body weight; pubertal timing) in conferred risk for AN. On one hand, it is possible that early puberty is a mechanism through which elevated childhood body weights are associated with risk for later eating disorder diagnosis. At the same time, because early puberty has not been found to be associated with AN in particular, it is likely there are other mechanisms at play.

Strengths of this study include the longitudinal design, use of contemporaneous and measured (rather than retrospective and self-reported) weights at multiple time points, use of an age-, sex-, and school-matched HC group, comparisons to normative data, and the use of a comprehensive community screening procedure to identify individuals with AN (which reduces selection bias commonly associated with clinical and research samples).

Nevertheless, the study has several limitations. Because this is a case-controlled study, with a relatively small sample size ($N = 51$ in each group), the selection of HCs necessarily impacts the study findings. Although comparisons to normative BMI data suggest a similar pattern of findings, it will be important to see these results replicate in another cohort setting with matched HCs. An additional limitation is that body weight and height measurements were not sufficiently frequent to allow examination of weight changes just prior to AN onset. Although we did not see a differential change in weight gain during childhood for the AN group, it is not possible to determine whether rate of weight gain or weight status just prior to AN onset differed from that of the HC group, as this would require more frequent (e.g., monthly) weight measurements. An additional limitation is that several individuals were excluded from the analyses of 6th grade BMI due to an onset of AN prior to this time point, which may complicate the interpretation of the findings; importantly, findings remained significant even when data were only considered through the grade 4 time point. As mentioned above, maternal height was not available for most participants and thus we were unable to compare the AN and HC groups on maternal BMI. Additionally, BMI was used as the primary outcome variable in this study, but we were unable to compare trajectories of other developmental indicators in the AN and HC groups. Furthermore, because the majority of the sample was female, it is not clear whether a male-only sample of individuals with AN would follow similar or different BMI trajectories.

These findings are consistent with the transdiagnostic theory of eating disorders put forth by Fairburn et al.,[34] which highlights the many shared features that exist across the range of eating disorder diagnoses. Our results, in combination with prior findings, suggest that elevated premorbid weights may be common across eating and weight disorders. It will be important for future studies to replicate these findings, evaluating body mass at birth and throughout childhood. Notably, childhood BMI likely represents only one of many important factors associated with general and specific risk for eating and weight disorders. In addition to study replication, further research is needed to consider potential mechanisms accounting for a relationship between elevated childhood BMI and the development of AN. Furthermore, future research is needed to identify environmental and psychological factors that differentially influence the development of over- as compared to under-eating behaviors in the presence of elevated childhood BMI. One obvious possibility is that some individuals who go on radical weight loss diets (a common point of entry to disordered eating) are responding in part to a BMI that is higher than most of their peers. Should these findings be replicated, it will be important that prevention programs take into account that children with elevated BMI relative to peers may be at risk for development of a range of eating and weight problems, including AN, BN, or obesity.

Data collection associated with the original study was supported by the Swedish Research Council (K2006-21X-20048–01-2), government grants under the ALF Agreement on Medical Education and Research, Knut and Alice Wallenberg's Foundation, the Söderström Königska Nursing Home Foundation, the Swedish Medical Society, and the Göteborg Freemasons. We want to thank Dr. Daniel Mirman for providing statistical consultation on this manuscript. There are no conflicts of interest to report.

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