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Assessing Eating Disorder Symptoms in Adolescence: Is There a Role for Multiple Informants?

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Abstract

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Objectives—Epidemiologic studies of adolescent psychiatric disorders often collect information from adolescents and parents, yet most eating disorder epidemiologic studies rely only on adolescent report.

Methods—We studied the eating disorder symptom reports provided by 7,968 adolescents from the Avon Longitudinal Study of Parents and Children (ALSPAC), and their parents, who were sent questionnaires at participants' ages 14 and 16 years. Both adolescents and parents were asked questions about the adolescent's eating disorder symptoms, including binge eating, vomiting, laxative use, fasting, and thinness. We assessed the concordance of parent and adolescent report cross-sectionally using kappa coefficients, and further looked at how the symptom reports were predictive of adolescent body mass and composition measured at a clinical assessment at 17.5 years. Generalized estimating equations were used to model the symptom reports' associations with risk factors and clinical outcomes.

Results—Parents and adolescents were largely discordant on symptom reports cross-sectionally ($\text{kappa} < 0.3$), with the parent generally less likely to report bulimic symptoms than the adolescent but more likely to report thinness. Female adolescents were more likely to report bulimic symptoms than males (e.g., 2-4 times more likely to report binge eating), while prevalence estimates according to parent reports of female vs. male adolescents were similar. Both parent and adolescent symptom reports at ages 14 and 16 years were predictive of age-17.5 body mass and composition measures; parentally-reported binge eating was more strongly predictive of higher body mass and composition.

Discussion—Parent report of eating disorder symptoms seemed to measure different, but potentially important, aspects of these symptoms during adolescence. Epidemiologic eating disorder studies should consider the potential value added from incorporating parental reports. In particular, studies of male eating disorder presentations may be improved by using multiple sources of information.

Keywords

eating disorder; adolescence; multiple informants; ALSPAC

Assessments using multiple informants have been increasingly incorporated into studies of childhood psychiatric disorders, with many epidemiologic studies including interviews or questionnaires completed by the child, a parent, and even a teacher¹⁻³. These methods have been used in studies of many different psychiatric disorders, including major depressive, bipolar, generalized anxiety, attention deficit/hyperactivity, and conduct disorders^{2, 4}. For disorders that involve symptom denial or non-normative behavioral manifestations in particular, incorporating an adult informant is often integral to measuring a valid diagnosis⁵.

Prior epidemiologic studies of eating disorders during adolescence have relied solely on information provided by the adolescent. Clinical studies, however, highlight the importance of a parental perspective and parental involvement^{6, 7}. Indeed, best practice for obtaining a clinical diagnosis of an adolescent's eating disorder should include information from the parents⁸. Clinical studies have shown relatively low concordance between parent and adolescent symptom reports, with prior investigators postulating this may be due to minimization of symptoms and hiding of behaviors⁹⁻¹¹. More specifically, anorexia nervosa

is defined in part by a denial of the illness, so parents may be particularly helpful in accurately identifying and describing these symptoms; alternatively, bulimic behaviors frequently involve secrecy and shame, which may imply parent report is indicative of severity and treatment-seeking.

For studies that gather information from multiple sources, four general analytic approaches have been implemented: (1) using data only from one informant (even if data from another informant is collected); (2) using data from each informant separately; (3) pooling information into a single construct (e.g., “OR rules”); or (4) jointly modeling the information. Although the first three approaches are most commonly employed, these methods have notable limitations, including inefficiency (Approach 1), limited interpretability (Approach 2), missing data issues (Approaches 2, 3), and inability to directly assess the relevance of source of information (Approaches 1, 2, 3). Alternatively, models developed by Fitzmaurice and colleagues (Approach 4) overcome many of these limitations^{12, 13}. Specifically, this approach allows information from multiple informants to contribute jointly to the same model, which allows for efficient use of all available data, and provides an opportunity to directly assess whether the source of information changes the relationship between the construct of interest (e.g., eating disorder symptoms) and both risk factors (e.g., sex) and outcomes (e.g., health outcomes).

In the Avon Longitudinal Study of Parents and their Children (ALSPAC), a population-based cohort study following children from birth through adulthood, parents and adolescents were asked about the adolescent's eating disorder symptoms at two assessments in adolescence¹⁴. We used this information to: (1) estimate the prevalence of eating disorder symptoms according to different methods for utilizing the available information; (2) model the prevalence of eating disorder symptoms within levels of covariates; and (3) assess the predictive validity of parent and adolescent report of eating disorder symptoms in models predicting subsequent measurements of body mass and composition.

Methods

Study Design

ALSPAC is an on-going longitudinal study of women and their children¹⁴. All pregnant women living in the geographical area of Avon, UK, who were expected to deliver between 1st April 1991 and 31st December 1992 were invited to join the study. Recruitment was aimed to recruit women as early in pregnancy as possible, via contact through media, community recruitment visits, and through advertisements of the study at routine antenatal and maternity health services. Further information on study procedures have been described elsewhere¹⁴. An estimated 85-90% of the eligible population enrolled in the study, including children from 14,541 pregnancies. At one year, there were 12,388 singleton children alive with complete information on child's sex and maternal age. For the present study, we restricted to singletons to avoid potential correlation between siblings. Children and their parents have been followed at regular intervals through young adulthood to investigate a range of psychological, physical and social outcomes. The present study focuses on responses to questionnaires sent to the parents and adolescents when the adolescents were 14 and 16 years, on clinical assessments when the adolescents were 17.5 years. We present

results for the 7,968 (64%) participants who had at least one of the questionnaires returned (i.e., by either informant at either assessment age). The differences between those who were followed through adolescence and those who were lost to follow-up have been previously reported: e.g., adolescents who remained in the study were more likely to be female, white, and have parents with higher incomes¹⁴. Demographic characteristics are described in **Table 1**.

Ethical approval for the study was obtained from the ALSPAC Laws and Ethics Committee and the Local Research Ethics Committees.

Measures

Adolescents completed questions on eating disorder symptoms adapted from the purging behavior assessments in the McKnight Risk Factor Survey and the Youth Risk Behavior Surveillance System Questionnaires¹⁵. Adolescents were asked whether they had engaged in eating disorder behaviors in the past year, including binge eating (overeating with loss of control; two questions), vomiting, laxative use, or fasting to control weight. Response options included: never; less than once per month; 1-3 times per month; once per week; 2-6 times per week; daily. At 14 years, adolescents were asked to describe their weight, with options including: very underweight; slightly underweight; about the right weight; slightly overweight; very overweight.

Parents completed questions from the Development and Well-Being Assessment (DAWBA), a semi-structured interview measuring psychiatric diagnoses in children and adolescents. The eating disorder section measures symptomatology for DSM-IV and ICD-10 diagnoses^{2, 16}. A questionnaire version of the ED-DAWBA, with no skip rules, was given to parents. Details on validation of this questionnaire are provided elsewhere¹⁷. Parents were asked whether their adolescent in the study had engaged in eating disorder behaviors in the past three months, including binge eating (overeating with loss of control; one question), vomiting, laxative use, or fasting. Response options included: no; a little; a lot; tried but not allowed; don't know. When the adolescent was 14 years, parents also reported on their adolescent's body type, with options including: very thin; thin; average; plump; fat.

Due to inconsistency of possible answer options across informants, parent and adolescent report of each of the above-described eating disorder behaviors (binge eating, vomiting, laxative use, and fasting) were dichotomized as any vs. no endorsement. For age 14 report of thinness, we also dichotomized responses, defining thinness as a parent report of "very thin" or an adolescent report of "very underweight."

At age 17.5 years, 4,264 participants had face-to-face clinical assessments at the ALSPAC study base. This visit included measurements of height and weight (which we used to calculate body mass index [BMI]), and Dual-energy x-ray absorptiometry scans from which absolute fat mass and lean mass were estimated. At age 18.5 years, participants were sent another questionnaire, which included a question whether they had ever been treated for an eating disorder.

Finally, we use three measures ascertained before the child's first birthday, including maternal education, maternal parity, and sex of the child. Maternal education was dichotomized as A level and above versus up through the general certification of secondary education. Note a general certificate of secondary education is most often taken when students are 14-16 years. Maternal parity was dichotomized as multiparity vs. primiparity, indicating whether the child was a first/only child or has older siblings.

Analyses

We first utilized more traditional approaches for estimating symptom prevalences using multiple informants, including: using only adolescent report, only parent report, and an "OR" rule method (i.e., yes if either the adolescent or parent or both endorsed the symptom, no if neither endorsed the symptom). These methods are presented using a complete case analysis.

We modeled prevalence of symptoms as well as informant effects^{12, 13, 18}. Specifically, for prevalence models of eating disorder symptoms as reported by either informant, we used generalized estimating equations (GEE) with an independence working correlation structure, empirical variance and logit-link to incorporate information provided by both respondents while accounting for correlation between measurements. Models included the child's sex, maternal education, and maternal parity, as well as the informant (adolescent vs. parent) as predictors for each symptom at each age. Significant covariate-informant product terms ($\alpha=0.05$) were included in final models.

We further incorporated the multiple informant reports as predictors using GEE to model BMI, absolute fat mass, and lean mass at age 17.5 years. Predictors included parent and adolescent reports of eating disorder symptoms, along with child's sex, maternal education, and parity. Significant informant -symptom product terms ($\alpha=0.05$) were included in the final models. All analyses were carried out in Stata 12.

One disadvantage of the standard GEE is that it requires that missingness be missing completely at random. While inverse probability weighting approaches have been proposed to loosen this restriction^{18, 19}, these are not feasible for complex non-monotone patterns. We imputed data on missing symptom reports and covariates using a chained equation approach with $m=25$ chains run for 25 iterations, with a linear regression model for continuous predictors and predictive mean matching for categorical variables²⁰. All variables in **Table 1** and all symptom reports were included in imputation models²¹.

Results

Prevalence estimates of binge eating, vomiting, laxative use, fasting, and thinness using complete case analysis are presented in **Table 2**. Using only adolescent report, estimates are comparable to what has been found in other population-based Western studies of eating disorder symptoms, or would be expected from such studies of full eating disorders, in youth^{22, 23}. Over 5% of adolescents reported binge eating, while reports of purging behaviors were slightly more rare; behaviors were more common at age 16 than at age 14. Relying only on parent report, prevalence estimates for each symptom were lower, with the

largest discrepancies seen for purging behaviors (e.g., vomiting at age 16: 4.9% according to adolescents vs. 0.3% according to parents). The estimated prevalence for each symptom was highest when taking an “OR rule” approach, with estimates often greater than the sum of parent-only and adolescent-only estimates due to missing data patterns.

For study participants for whom we had both the adolescent and parent report, we estimated the concordance between the two informants with kappa coefficients, overall and stratified by sex. Kappa estimates were all below 0.3, indicating at most slight to fair agreement. Given the universally low concordance, we have included these kappa estimates as supplemental material only, along with two specific examples to illustrate the patterns of discordance (**eTable 1-3**). For vomiting and laxative use, adolescents tended to endorse the behavior more often than parents, and parents only rarely indicated the presence of the behavior when the adolescent denied it. In contrast, for binge eating, fasting, and thinness, parents and adolescents reported more similar magnitudes of prevalence, but were in disagreement about their presence.

We modeled prevalence of each symptom at each age by covariates and informant in **Table 3**. For binge eating, vomiting, laxative use, and fasting, these models indicate that prevalence estimates for each symptom are higher when reported by the adolescent relative to their parent; for thinness, the inverse was observed. An informant-sex product term was significant for vomiting, fasting, and binge eating measured at both ages, indicating the magnitude of any differences in prevalence of these symptoms between females and males depends on the source of information. An example of model-based prevalence estimates of each symptom by informant, age group, and sex are presented in **eFigure 1**.

In **Tables 4**, we present models predicting BMI at age 17.5 years, with sex, parity, maternal education, each eating disorder symptom, and informant as predictors. Models indicate that within an informant, symptom reports were predictive of BMI (e.g., using these models, we would expect the child of a parent reporting binge eating at 14 years would have a BMI 2.5 kg/m² higher at 17.5 years than the child of a parent reporting no binge eating at age 14). Generally, BMI at 17.5 years were nearly identical whether the parent reported a lack of symptom or the adolescent reported a lack of symptom. The symptom-informant product terms indicate parents' report of binge eating was more predictive of BMI than the adolescents' report of binge eating; this was less consistently seen for other symptoms. In **eTables 4 and 5**, we present similar findings for predicting absolute fat and lean mass at 17.5 years using parent and adolescent eating disorder symptom reports; we further provide example model-based estimates of expected BMI, fat mass, and lean mass by informant and symptom endorsement in **eFigures 2 and 3**. We ran similar models for predicting eating disorder treatment, but the rarity of this outcome limited interpretability (results not shown). Of note, even though treatment status was reported by the adolescent, the relationship between symptom reports and the adolescents' reported treatment status was similar regardless of the informant reporting the symptom status.

Conclusions

Epidemiologic studies of eating disorders in youth have traditionally relied on only self-report, while the potential usefulness of parent report was unknown. In the present study, parents and adolescents generally were discordant when reporting on eating disorder symptoms, with purging behaviors generally reported more frequently by the adolescents, while discordance for binge eating, fasting, and thinness was perhaps due more to disagreement than possibly over- or under-reporting by one informant over another. Thus, prevalence estimates based adolescent only, parent only, or “OR rule” measures varied considerably. Our prevalence models highlight the importance of the source of information, particularly by sex: adolescent reports aligned with other epidemiologic studies finding an increased risk in females^{22,25}, while parent reports had a more muted sex difference. Finally, eating disorder symptom reports by either the parent or adolescent were generally predictive of subsequent body mass and composition measures, although parent report of binge eating may be considered more predictive.

The patterns of discordance between these reports may be unsurprising given clinical knowledge of how these symptoms manifest in eating disorder patients. Adolescents reported purging behaviors (vomiting and laxative use) more frequently than their parents, a pattern that aligns with the often hidden nature of bulimic behaviors: similar to other psychiatric constructs that often involve secrecy (e.g., deliberate self-harm, substance use), we expected some adolescents to report these symptoms on an anonymous questionnaire even though the behavior may be hidden from their parents. Indeed, when adolescents were asked to explain why they thought their parents would fail to report the adolescent used drugs or alcohol when the adolescent reported they did use one of these substances, most expected that the parents were simply unaware of the behavior⁵. Information on purging behaviors provided by parents, then, may aid investigators in interpreting frequency of, severity of, or treatment-seeking for the behaviors rather than affirming presence or absence. For binge eating and thinness, parents and adolescents may be offering different perspectives or interpretations of these constructs. In particular, parents and adolescents may simply disagree on what eating “objectively large” amounts of food looks like (part of the definition of binge eating), or use different anchors to assess bodyweight. Finally, fasting exhibited both styles of discordance: at age 14 years, prevalence of fasting as reported by parent and adolescent was not greatly dissimilar (yet still discordant), while adolescents endorsed fasting at 16 years at a much greater frequency than parents. Like purging behaviors, perhaps fasting is a behavior the adolescent would largely try to hide. However, at younger ages, parents may be more involved in adolescents’ eating habits (e.g., family meals) and thus the behavior is not as easily hidden. Further research may explore the importance of family meals for the early identification of disordered eating.

Our models of symptom prevalence indicate that measuring sex differences may be complicated by the source of information. Largely based on results from treatment-seeking samples, it has been argued that eating disorders are much more common in females than males, with gender ratios as high as 9:1²⁴. Prior population-based studies indicate that this ratio is perhaps not as extreme, with estimated gender ratios closer to 3:1^{22, 25}. However, the prior studies in youth rely on self-report. Indeed, when focusing on adolescent reports, our

results align with these prior epidemiologic studies (e.g., binge eating as reported by the adolescent was 2-4 times more common in females than males). When comparing parent reports for boys and girls, the gender ratio appeared to be nearly 1:1. At minimum, these results underscore the importance of the source of information when assessing the prevalence and distribution of disorders in a population. Long-held beliefs that eating disorders are much more common in females than males are rooted in research focused on treatment-seeking samples or epidemiologic studies using only self-report, and thus the presentation and course of illness in males has been a neglected area of research. Indeed, several recent studies have suggested current measurements of eating disorders are not well suited to boys. For example, cognitive features may manifest differently due to different ideal body types (valuing muscularity over thinness), and males may not accurately or consistently report objective overeating and loss of control (i.e., components of binge eating)²⁶⁻²⁸. As eating disorder assessments continue to evolve, incorporating parental reports could aid our understanding of these constructs as they present in males.

As stated above, this study does not address whether the information provided by the parent or adolescent is more or less valid across all possible studies or research questions. Nonetheless, we can describe their relative relevance in predicting later outcomes expected to be strongly correlated with bulimic behaviors and prior bodyweight²³. Indeed, both informants' reporting of symptoms was predictive of future BMI and body composition measures. The magnitude of difference between the expected outcomes with binge eating compared to without was slightly larger when considering parent reports. One possible interpretation is parental reports better recognize the facets of the binge eating construct that are predictive of later BMI and body composition; alternatively, parents may only be recognizing more severe behaviors, and thus when they do recognize the behavior it is strongly predictive.

Some limitations warrant consideration. Because different questionnaires were given to the parents and adolescents, question wording for each symptom did not perfectly align. Different answer choices forced us to dichotomize behaviors as any vs. none; future research is needed to assess how frequency of behaviors may modify concordance. Parents were asked whether the behaviors occurred in the prior three months while adolescents were asked whether they occurred in the prior year; thus, it is possible that some (although likely not all) of the discordance when an adolescent endorsed a behavior that the parent did not is due to symptoms that occurred between three and twelve months ago. In particular, it is unlikely that the discordance is completely explained by this temporal discrepancy because parents more often reported thinness. Moreover, given the relationship between lifetime and period prevalences seen in prior studies, it is unlikely the temporal discrepancy would influence these results^{22, 25}. The strengths of this study likely outweigh these limitations. It is a large cohort with repeated measurements of many important eating disorder symptoms, as well as important body mass and composition measures. Our modeling approaches allowed us to present interpretable results while efficiently using all collected data and advanced techniques to address missingness^{12, 18}.

Eating disorders are of great public health concern, particularly during adolescence, because these disorders are prevalent and are risk factors for severe medical complications and

suicide^{22, 25, 29, 30}. Thus, it is critical that epidemiologic studies of eating disorders obtain all relevant information so we can study prevention, intervention, and consequences of these disorders using valid measurements. Future studies of eating disorders may consider incorporating both parent and adolescent report, particularly if studying sex differences, bearing in mind that each informant may be providing different yet potentially valuable information.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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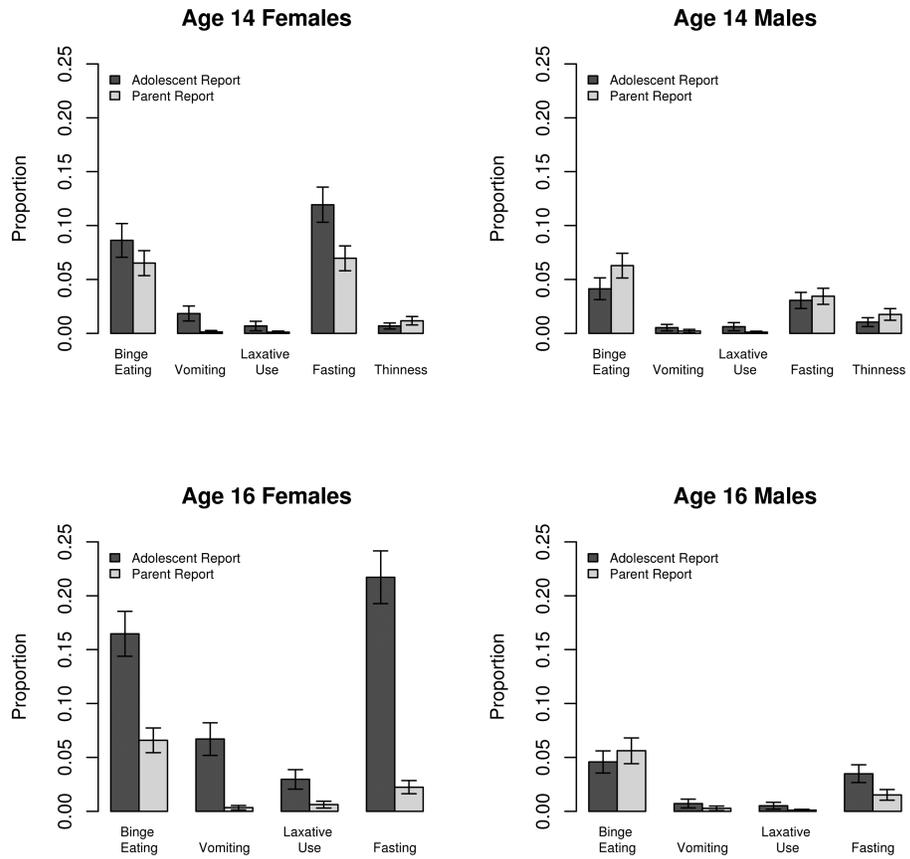


Figure 1. Model-based symptom prevalence by age, sex, and informant among first-born or only children with lower maternal education levels

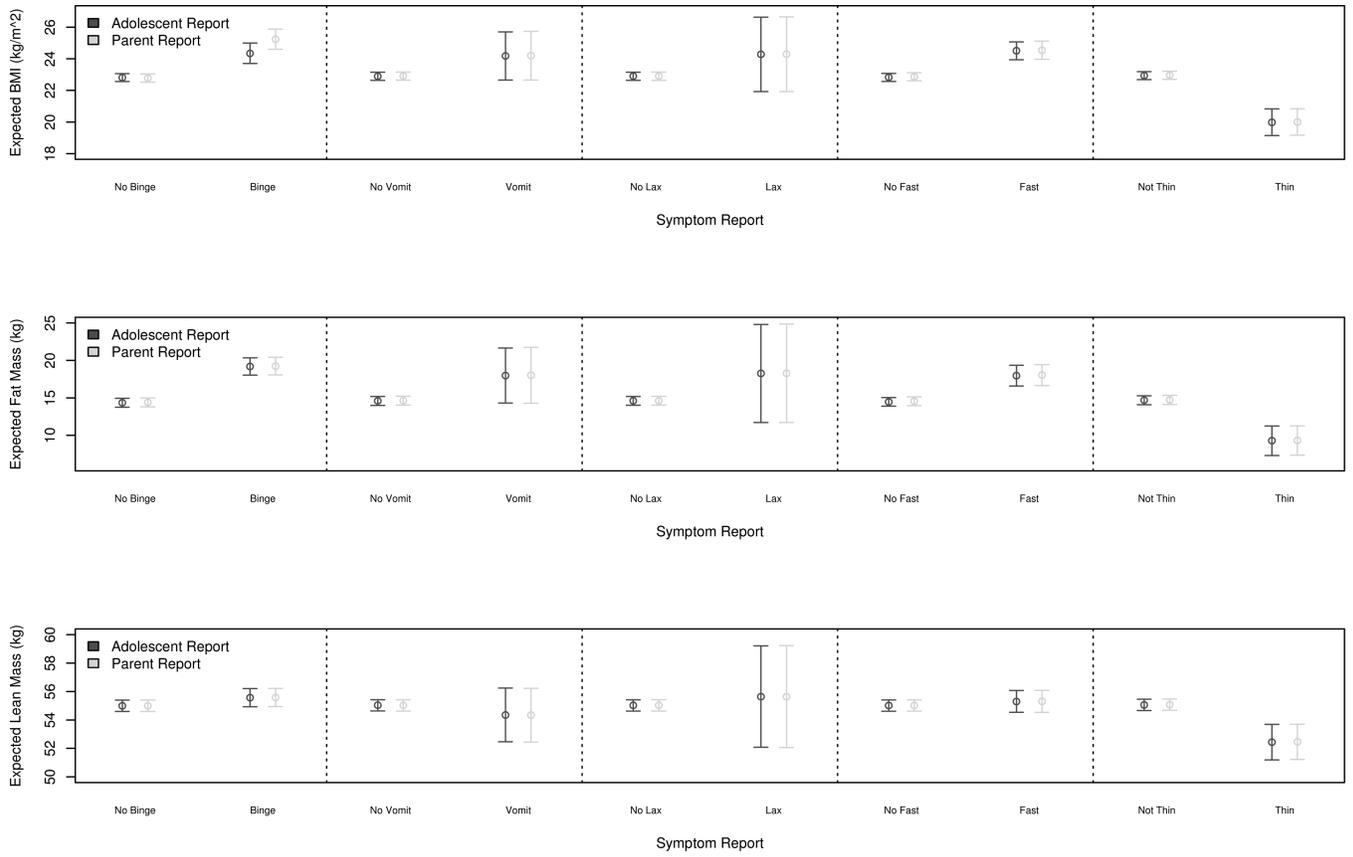


Figure 2. Model-based expected BMI, fat mass, and lean mass at 17.5 years by age-14 symptom report and informant among first-born or only children with lower maternal education levels

Table 1

Description of cohort (N=7,968)

	Full Population	Male	Female
Male, N (%)	3,834 (48.1)		
Maternal education A-level or above, N (%) *	3,350 (42.4)	1,580 (42.5)	1,670 (42.1)
Primi-parity, N (%) *	3,594 (46.6)	1,749 (47.0)	1,845 (46.2)
BMI at 17.5 years, Mean kg/m ² (SD) *	22.7 (3.9)	22.4 (3.6)	22.8 (4.1)

* Maternal education, parity, and BMI (i.e., height and weight) were measured in 7,679, 7,714, and 4,264 respondents, respectively.

Table 2

Prevalence of eating disorder symptoms according to adolescent report only, parent report only, and using the “OR rule” method, % (Number Reporting Symptom/Number Measured)

	Adolescent Report	Parent Report	Parent or Adolescent Report *
Age 14			
Binge Eating	5.74 (327/5,693)	4.87 (322/6,618)	12.17 (612/5,027)
Vomiting	1.37 (79/5,765)	0.18 (12/6,594)	1.80 (89/4,942)
Laxative Use	0.43 (25/5,739)	0.08 (5/6,621)	0.61 (30/4,935)
Fasting	6.69 (284/5,739)	4.70 (309/6,576)	12.77 (648/5,074)
Thinness	0.98 (57/5,803)	1.67 (112/6,689)	3.16 (159/5,024)
Age 16			
Binge Eating	10.87 (515/4,738)	5.07 (271/5,344)	18.13 (735/4,053)
Vomiting	4.93 (236/4,788)	0.30 (16/5,252)	6.30 (246/3,905)
Laxative Use	1.88 (90/4,792)	0.38 (20/5,214)	2.74 (105/3,836)
Fasting	13.23 (633/4,786)	1.63 (80/4,894)	18.06 (697/3,860)

* Note the “OR rule” method is only defined for subjects with both an adolescent and parent report.

Table 3Symptom prevalence model estimates, odds ratios (95% CI)^{*}

	Binge Eating	Vomiting	Laxative Use	Fasting	Thinness
Age 14 Models					
Parity (Multiparous)	0.78 (0.62, 0.97)	1.54 (1.03, 2.31)	0.87 (0.43, 1.73)	1.18 (1.01, 1.39)	1.12 (0.82, 1.51)
Maternal Education (A level or above)	0.71 (0.61, 0.84)	0.83 (0.55, 1.26)	0.62 (0.31, 1.24)	0.63 (0.54, 0.74)	1.20 (0.88, 1.62)
Sex (Female)	1.04 (0.84, 1.29)	0.64 (0.21, 1.97)	1.10 (0.53, 2.28)	2.10 (1.65, 2.68)	0.66 (0.49, 0.89)
Informant (Child)	0.64 (0.48, 0.87)	2.54 (1.10, 5.90)	6.43 (2.65, 15.62)	0.88 (0.65, 1.21)	0.59 (0.42, 0.81)
Sex*Informant	2.11 (1.50, 2.96)	5.59 (1.60, 19.45)		2.05 (1.42, 2.96)	
Parity*Informant	1.46 (1.08, 1.96)				
Maternal Education*Informant					
Age 16 Models					
Parity (Multiparous)	1.05 (0.91, 1.22)	1.33 (1.03, 1.71)	1.31 (0.92, 1.84)	1.16 (0.99, 1.36)	NA
Maternal Education (A level or above)	0.66 (0.53, 0.83)	1.15 (0.89, 1.48)	0.80 (0.55, 1.16)	0.77 (0.66, 0.90)	NA
Sex (Female)	1.19 (0.93, 1.51)	1.22 (0.50, 2.95)	6.00 (3.28, 10.99)	1.48 (0.99, 2.21)	NA
Informant (Child)	0.81 (0.61, 1.07)	2.61 (1.09, 6.30)	4.93 (3.08, 7.89)	2.34 (1.61, 3.42)	NA
Sex*Informant	3.47 (2.51, 4.81)	8.31 (3.07, 22.53)		5.19 (3.29, 8.21)	NA
Parity*Informant					NA
Maternal Education*Informant	1.41 (1.07, 1.85)				NA

NA: no thinness measure available at age 16

^{*} Models used multiple imputation methods for missingness. Predictors of missingness are summarized in eTable 4.

Table 4BMI model (kg/m²) estimates, coefficients (SE)^{*}

	Binge Eating	Vomiting	Laxative Use	Fasting	Thinness
Age 14 Models					
Intercept	22.78 (0.13)	22.91 (0.13)	22.90 (0.13)	22.86 (0.13)	22.96 (0.42)
Parity (Multiparous)	0.01 (0.11)	-0.01 (0.11)	-0.003 (0.11)	-0.02 (0.11)	0.00002 (0.11)
Maternal Education (A level or above)	-0.67 (0.10)	-0.70 (0.10)	-0.70 (0.10)	-0.66 (0.10)	-0.69 (0.10)
Sex (Female)	0.33 (0.11)	0.35 (0.11)	0.36 (0.11)	0.26 (0.11)	0.35 (0.11)
Symptom	2.46 (0.31)	1.29 (0.74)	1.39 (1.15)	1.68 (0.25)	-2.95 (0.42)
Informant (Child)	0.03 (0.07)	-0.02 (0.06)	-0.006 (0.06)	-0.04 (0.06)	-0.02 (0.06)
Symptom*Informant	-0.92 (0.45)				
Age 16 Models					
Intercept	22.80 (0.13)	22.92 (0.13)	22.90 (0.13)	22.93 (0.13)	NA
Parity (Multiparous)	-0.01 (0.11)	-0.01 (0.11)	-0.01 (0.11)	-0.01 (0.11)	NA
Maternal Education (A level or above)	-0.67 (0.10)	-0.70 (0.10)	-0.69 (0.10)	-0.69 (0.10)	NA
Sex (Female)	0.28 (0.11)	0.34 (0.11)	0.34 (0.11)	0.28 (0.11)	NA
Symptom	2.56 (0.34)	0.55 (0.37)	4.33 (1.20)	0.89 (0.23)	NA
Informant (Child)	0.01 (0.07)	-0.02 (0.07)	-0.002 (0.06)	-0.10 (0.07)	NA
Symptom*Informant	-1.38 (0.39)		-3.34 (1.16)		NA

NA: no thinness measure available at age 16

^{*} Models used multiple imputation methods for missingness. Predictors of missingness are summarized in eTable 4.