Eating behaviours in childhood and later eating disorder behaviours and diagnoses: a longitudinal study

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Background

Eating behaviours in childhood have long been considered as risk factors for eating disorders (ED) behaviours and ED diagnoses in adolescence. However, only few longitudinal studies have examined this association.

Aim

We investigated associations between childhood eating behaviours during the first ten years of life and ED behaviours (binge eating, purging, fasting, and excessive exercise) and diagnoses (anorexia nervosa [AN], binge-eating disorder [BED], purging disorder [PD], and bulimia nervosa [BN]) at 16 years.

Methods

Data on 4,760 participants from the Avon Longitudinal Study of Parents and Children were included. Longitudinal trajectories of parent rated childhood eating behaviours (8 time points, 1.3-9.yrs) were derived using latent class growth analyses. ED diagnoses were derived from self-, parent –report and objectively measured anthropometric data at age 16. We estimated associations between childhood eating behaviours and ED behaviours and diagnoses using multivariable logistic regression models.

Results

Childhood overeating was associated with increased risk of adolescent binge eating (risk difference [RD]: 7%; 95% CI: 2, 12) and BED (RD: 1%; 95%CI: 0.2, 3). Persistent undereating was associated with higher AN risk in adolescent girls only (RD: 6%; 95%CI: 0, 12). Persistent fussy eating was associated with greater AN risk (RD: 2%; 95%CI: 0, 4).

Conclusions

Our results suggest continuities of eating behaviours into ED from early life to adolescence. It remains to be determined whether childhood eating behaviours are an early manifestation of a specific phenotype or whether the mechanisms underlying this continuity are more complex. Findings have the potential to inform preventative strategies for ED.

Introduction

Eating behaviours reflect our relationship with food and behavioural responses to food cues in the environment (1). Eating behaviours develop early and track according to distinct trajectories (2). While childhood eating behaviours have typically been studied in the context of obesity (3), their role in the development of eating disorders (EDs) in adolescence has received little attention. Importantly, in addition to overeating and undereating, food fussiness—the tendency to eat only certain foods and to refuse to try new foods—is common during childhood (2), and might be a precursor to adolescent dieting, a previously suggested risk factor for AN (4). The aetiology of EDs remains poorly understood and primary prevention of EDs, especially in children, is in need of improvement (5). A study following 800 children from six to 22 years highlighted that conflicts and struggles around meals in childhood were associated with higher risk for anorexia nervosa (AN) in adolescence and young adulthood, whereas childhood undereating was associated with later bulimia nervosa (BN) (6). This study was limited by its relatively small sample size, low number of ED cases, and focus on diagnosed EDs excluding ED behaviours, which may be prodromal expressions of ED risk. Additionally, data from the longitudinal 1970 National Child Development Study showed that infant feeding problems and childhood undereating were risk factors for adult AN at age 30 (7). However, in contrast to these previous studies, infant and childhood eating behaviours were not associated with adult bulimic or compulsive eating (8). These studies, however, might have benefited from a more comprehensive assessment of childhood eating. We have recently showed a longitudinal relationship between childhood growth and ED (9). Further, we have described longitudinal trajectories of eating behaviour in childhood and their association with BMI at age 11 years (2). In this study, we aimed to extend this work by establishing the associations between these eating behaviour trajectories during the first ten years of life and ED behaviours and diagnoses in adolescence, leveraging data from a large UK based birth cohort. We hypothesized that continuity would be observed from eating behaviours in childhood to ED behaviours and disorders in adolescence. Specifically, overeating during childhood would be associated with increased risk for binge eating, purging, binge-eating disorder (BED), and purging disorder (PD) in adolescence. Conversely, we hypothesized that undereating and fussy eating would be associated with greater risk for fasting, excessive exercise, and AN in adolescence.

Method

Ethics

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees.

Study population

Data were from the Avon Longitudinal Study of Parents and Children (ALSPAC), a population based, longitudinal cohort of mothers and their children born in the southwest of England (10, 11). All pregnant women expected to give birth between the 1st April 1991 and 31st December 1992 were invited to enrol in the study, providing informed written consent. 14,451 pregnant women opted to take part; by one year, 13,988 children were alive. One sibling per set of multiple birth (N=203 sets) was randomly selected from these analyses to guarantee independence of participants. Please note that the study website contains details of all the data that is available through a fully searchable data dictionary and variable search tool. (www.bristol.ac.uk/alspac/researchers/our-data).

Outcomes

ED diagnoses and behaviours

Binge eating, purging, fasting, and excessive exercise were obtained by self-report at age 16 years. We adapted questionnaire items from the Youth Risk Behaviour Surveillance System (12), which have previously been validated in a population-based study (13). Binge eating was defined as eating a large amount of food at least once a week and having a feeling of loss of control during that episode. In order to assess purging behaviour, participants indicated if they used of laxatives or self-induced vomiting in order to lose weight or avoid gaining weight. Fasting was described as not eating at all for at least a day in order to lose weight or avoid gaining weight. Participants who indicated that they exercised for weight loss purposes, felt guilty about missing exercise, and finding it hard to meet other obligations, such as

schoolwork, due to exercise regime were coded as engaging in excessive exercise. EDs diagnoses according to DSM-5 criteria (i.e., AN, BN, BED, PD) were obtained as described previously (14).

Analyses were restricted to adolescent ED diagnoses and behaviours at age 16 years to balance the loss of participants at later ages (18 years) and the low prevalence of the outcomes at earlier ages (age 14 years).

Exposures

Trajectories of childhood eating behaviours

Trajectories of overeating, undereating, and fussy eating were included from repeated measures of parent-reported child eating behaviours available at eight time points (around the ages of 1.3yrs, 2yrs, 3.2yrs, 4.6yrs, 5.5yrs, 6.9yrs, 8.7yrs, and 9.6yrs), (see Herle et al, 2019 for details (2)). Parents indicated how worried they were about their child's overeating and undereating. Fussy eating was rated across three questions, "being choosy", "refusing food" and "general feeding difficulties". Response options for the five tiems were "did not happen", "happened, but not worried" and "a bit/greatly worried". Trajectories were derived using Latent Class Growth Analyses with children which had data on at least one time point (n=12,002). Analyses included children with data on child eating behaviours trajectories and at least one of the outcomes at age 16 years (n=4,760).

Overeating trajectories included*:* "Low overeating" [children described as having no overeating in childhood, n=3,311, 70%], "Low transient overeating" [children with some degree of overeating during the first five years, which subsided by 9.6 years, n=606, 13%]; "Late increasing overeating" [children with low levels of overeating until age 5 yrs and increasing levels from this age onwards, n=552, 12%]; and "Early increasing overeating" [children with an early onset of overeating which progressively increased over time, n=291, 6%].

Undereating trajectories included: "Low undereating" [children with no undereating, n=1,134, 24%]; "Low transient undereating" [children with some undereating during which decreased to no undereating by 4.6yrs, n=1,805, 38%]; "Low and decreasing undereating" [children with some undereating during childhood slowly, which completely subsided by 9.6yrs, n=927, 19%]; "High transient undereating" [children with high levels

of undereating at 1.3yrs which rapidly decreased by 5.5yrs, n=628, 13%]; "High decreasing undereating" [children with high levels of undereating which slowly decreased to no undereating by 9.6 years, n=181, 4%] and "High persistent undereating" [children who were persistently undereating across childhood, n=82, 2%].

Fussy eating was represented in six trajectories: children with "Low fussy eating" [children characterised by no fussy eating, n=983, 21%]; "Low transient fussy eating" [low levels of fussy eating in first 5 years of life, which decreased to no fussy eating by 9.6yrs, n=737, 15%]; "Low and increasing fussy eating" [characterized by a low levels of fussy eating that slowly increase with time, n=1,258, 26%]; "Early and decreasing fussy eating" [high levels of fussy eating at 1.3yrs which decreased gradually, n=736, 15%]; "Rapidly increasing fussy eating" [low levels of fussy eating at 1.3 months, increasing rapidly with age, n=607, 13%] and "High persistent fussy eating " [children who were fussy eaters throughout the period assessed, n=439, 9%].

Groups are illustrated in Supplementary figures 1-3.

Covariates

The following covariates were included to control for confounding of the association between the exposures and outcomes: (i) Maternal education status (A-Levels or higher, lower than A-Levels; A-Levels are needed to enrol in university in the UK), to proxy socio-economic status of the family (ii) maternal age at birth; (iii) size at birth (gestational age and birthweight).

Statistical methods

We estimated the association between the eating behaviour trajectories of overeating, undereating, and fussy eating and ED outcomes during adolescence using multivariable logistic regression models. The results are presented in two ways: (i) Estimated mean probabilities of outcomes per class, averaged over the distribution of the confounders (marginal effects); (ii) estimated risk differences [RD] relative to the reference group i.e., the estimated difference in risk of ED behaviours and diagnoses for each group of eating behaviours, relative to the reference group. In addition, we also report Baseline risks of the outcomes in these reference groups, averaged over the distribution of the confounders, which indicate the risk of

endorsing disordered eating outcomes and ED diagnoses for adolescents classified with low levels of child overeating, undereating and fussy eating. As commonly observed in longitudinal cohorts, data are subject to loss to follow-up. We assumed outcome data were missing at random (MAR), conditionally on the variables included in the models, which are associated with drop-out: maternal education and maternal age. ED behaviours and diagnoses tend to be more common in girls. Hence, we repeated regression analyses in girls only (Supplement tables 1a-c). Regression analyses were conducted in Stata 15. Two-sided tests were used to assess significance with Bonferroni corrections implemented, dividing 0.05 by the number of comparisons for each eating behaviour. This lead to p≤0.02 for overeating (0.05 divided by 3), and p≤0.01 for undereating and fussy eating (0.05 divided by 5).

Results

There were no cases of BN in boys and few (<0.5%) cases of PD in either boys or girls, hence BN and PD were dropped from our analyses (Table 1).

TABLE 1

Associations between eating behaviour trajectories and ED behaviours and diagnoses

Overeating

Children in the low overeating reference trajectory had an estimated baseline risk (BR) of 10% (95%CI: 9, 11) for binge eating at 16 years; 2% (95%CI: 0, 5) for purging and 1% (95%CI: 0, 1) met diagnostic criteria for BED (Supplement Figure 4). Children with late increasing overeating had a 6% increased risk of engaging in binge eating in comparison to the reference low overeating group (RD: 5%, 95%CI: 2, 8). Children with early increasing overeating had a 7% greater risk of binge eating (RD=7%, 95%CI: 2, 11). Those with late increasing overeating had a 1% increase in risk of BED (RD: 1%, 95%CI: 0, 3) (see **Table 2**). There was no association between overeating trajectories and purging.

TABLE 2 HERE

Undereating

Children in the reference group had an estimated (BR) of 6% (95%CI: 4, 7) for excessive exercise, 15% (95%CI: 13, 17) for fasting, and 2% (95%CI: 1, 2) of AN. In comparison, low transient and low, but slowly decreasing, undereating was associated with a 3% lower risk of fasting (RD: -3%, 95%CI: -5, 0 and RD: -3, 95%CI; -6, 0) and purging (both groups; RD: -3%, 95%CI: -5, -1) compared with the reference group with no undereating at all (**Table 3, Supplement Figure 5**). In addition, children with low and decreasing levels of undereating had a 2% risk reduction for excessive exercise (RD: -3%, 95%CI: -4, -1). There was no association between undereating trajectories and AN. However, when investigating girls only, children in the persistent high undereating children had a 6% increased risk of meeting AN diagnostic criteria in comparison to the reference group (RD: 6%, 95%: 0, 12) **(Supplement 1b)**.

TABLE 3 HERE

Fussy eating

Children in the reference group, low fussy eating, had an estimated BR of 6% (95%CI: 4, 7) excessive exercising, 15% (95%CI: 13, 17) fasting, and 1% (95%CI: 0, 2) of AN. Children with high persistent fussy eating had a 2% risk increase for AN, in comparison to the children never reported to be fussy eaters (RD: 2%, 95%CI: 0, 0.4%) (Supplement Figure 6, Table 4). Similarly, the children who were fussy eaters only in early life ("Early and decreasing"), had 2% increased risk of AN (RD: 2%, 95%CI: 1, 4%) in comparison to the low fussy eating reference group. There were no associations between fussy eating trajectories and excessive exercise.

TABLE 4 HERE

Sensitivity analyses

Given the possibility that the associations between high persistent undereating, high persistent fussy eating, and later AN might be driven by children who were both persistent under- and fussy eaters we performed sensitivity analyses to investigate the overlap between girls with persistent undereating and persistent fussy eating. However, among the 69 girls meeting criteria for AN, only four fell into both the persistent undereating and persistent fussy eating group. This indicates that both eating behaviours may have a unique association with AN.

Discussion

We have conducted the most comprehensive investigation of the association between child eating behaviours and ED behaviours and diagnoses in adolescence. In line with our hypotheses, overeating during childhood was associated with increased risk for binge eating and BED at 16 years. Children with elevated overeating were found to have 6-7% greater risk of engaging in binge eating than children in the persistently low group, relative to the baseline risk of 10%. In addition, those with an increase in overeating in mid-late childhood had a 1% greater probability of BED compared to the reference group, suggesting that overeating behaviour linked to later BED might start in mid childhood at about age 5 years. Results highlight how increasing rates of childhood overeating could foreshadow later binge eating, and support the results from smaller longitudinal studies, indicating that eating in absence of hunger in childhood is associated with greater binge eating at 15 years (15). Additional research, using molecular genetic approaches, proposed that genetic variants associated with BMI were also associated with adolescent binge eating, suggesting a shared genetic aetiology between overeating and binge eating (16).

Low levels of undereating were associated with lower risk of fasting and excessive exercise at age 16 years. Low levels of undereating might be interpreted as a sign of a healthy appetite in early life, which could act as a protective factor, reducing the risk of later AN, fasting, purging and excessive exercise. In accordance, previous research has suggested that higher BMI at 10 years was associated with body dissatisfaction and dieting at age 14 (17). In contrast to our hypotheses, we found no associations between persistent undereating and later ED behaviours or diagnoses; this is likely due to the small number of children and this is reflected in the wide CIs estimates found in the results. In comparison when repeating the analyses in girls only, there was an indication that persistent undereating was associated with a 6% increase in risk for AN relative to baseline risk of 1%. However, due to the reduced sample, the persistent undereating trajectory only included 54 girls and the confidence intervals were wide (from 0 to 13%). Previous longitudinal research has suggested that early feeding problems and undereating in childhood are linked to adolescent AN (6, 7). In addition, adolescents with EDs have been found to have different patterns of growth in the first 12 years of life, with AN patients showing consistent premorbid low BMI (18). Similar to

the association between overeating, binge eating and obesity, shared genetic vulnerability might contribute to the association between consistent levels of undereating in childhood and risk for AN.

In line with our hypotheses, early life and persistent food fussiness was associated with a 2% risk increase in AN in comparison to no fussy eating (baseline risk 1%). Fussy eating in childhood might, in some cases, be an early manifestation of later AN. However, the shared aetiology between fussy eating and disordered eating is unknown. The newly described diagnosis of avoidant/restrictive food intake disorder (ARFID) (19) is of interest. ARFID is characterized by severe restriction and lack of interest in food (20), resulting in failure to meet appropriate energy and/or nutritional needs. The persistent fussy eating group might include children with ARFID, suggesting a potential association between childhood ARFID and adolescent AN. ARFID in childhood has been suggested as a risk factor for adolescent AN (21), and more longitudinal research is needed. Severe and persistent fussy eating can be very stressful for families, and parents, often understandably, respond by pressuring their child to eat more or offering treats as rewards to entice their child to eat (22). Parental pressure to eat has been associated with weight and shape concerns (23), and to fasting in adolescence (24), which might unintentionally steer fussy children toward more extreme behaviours. However, it is important to note that parent-child conflicts in adolescence are more likely to be the consequence of disordered eating, rather than the cause (25). Our results presented here provide support for the hypothesis that persistent levels of child fussy eating might be a contributing factor or potentially an early manifestation of adolescent AN. However, the mechanism behind this observation is unknown.

Strength and Limitations

To our knowledge, this is the largest and most comprehensive investigation to examine the prospective association between childhood Eating behaviours and adolescent ED behaviours and diagnoses. Data were from a population-based cohort, and longitudinal latent trajectories were used as exposures to acknowledge the heterogeneity of childhood eating behaviours, superseding the few previous longitudinal studies in this field. In addition, diagnoses of EDs were derived from a combination of self- and parental report. As commonly observed in longitudinal cohort studies, participants tend to drop out as time goes on. This loss of follow-up has been associated with socio-economic position of the participants (26), and their

families, reducing the generalisability of the findings. Hence, we included maternal education and maternal age at birth in the analyses. ED behaviours and diagnoses were not very common, especially in boys, leading to low statistical power. Questionnaires to assess ED diagnoses and behaviours tend to be geared towards girls, and separate assessments using tools specifically for boys would have been beneficial (27). Additionally, we were not able to estimate the association between eating behaviour trajectories and adolescent ARFID, as the study, and measures to assess ED, were developed prior to the definition of ARFID. A new psychometric tool for ARFID is now available and can be used in future observational studies (28).

Implications

Adolescent ED behaviours and ED are complex and influenced by interactions of biological, behavioural, and environmental factors (29). Our results support the hypothesis that there is continuity between early life Eating behaviours and later ED behaviours and ED. This notion has important implications for future preventive strategies. Findings suggest that identifying children with specific eating behaviours might be a promising approach for targeted intervention to prevent progression to disordered eating and ED in adolescence. Previous trials highlighted that childhood eating behaviours are modifiable through interventions targeting parental feeding and education (30). However, these intervention studies focus on the development of healthy eating behaviours in the context of obesity prevention and not ED, and may have unintended consequences. For example it has been observed that parental pressure to eat can lead to increased food refusal (31). Importantly, recent evidence suggests that family-based treatment intervention for weight loss might also positively influence disordered eating behaviours such as binge eating in adolescents with obesity (32). Our results suggest that children who show high levels of fussy eating in early childhood might be at increased risk of developing AN, the mechanisms behind this associations are unknown and need to be further explored.

In conclusion, the results from this study extend the previous longitudinal studies and support the hypothesis of continuity from childhood eating behaviours to ED behaviours and disorders in adolescence, particularly for childhood overeating and later binge eating, and for childhood fussy eating and later AN. There was an indication of an association between persistent undereating and AN in girls only. Our

observations pave the way for improved understanding of risk pathways to ED, including the role of early phenotypic manifestations, and exploration of genetic risk. Future investigations should explore protective factors that buffer children who display high persisting levels of overeating and fussy eating from developing ED behaviours or ED later on.

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Declaration of interest

CB reports: Shire (grant recipient, Scientific Advisory Board member) and Pearson and Walker (author, royalty recipient). All other authors have indicated they have no conflicts of interest to disclose.

Author Contribution

MH and BDS analysed the data. MH, BDS and NM drafted the manuscript

All authors substantially contributed to the conception and interpretation of the work, revised the manuscript for important intellectual content and approved the final version. All authors agree to be accountable for all aspects of this work.

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Table 1. Distribution of baseline variables by completeness of eating behaviours and outcome data

Baseline characteristics		**Participants with at least one measure of eating behaviour (N=12,002)**	**Participants with at least one measure of eating behaviour and at least one outcome measure (N=4760)**
		Mean (SD) or N (%)	**Mean (SD) or N (%)**
Sex	Boys	6,208 (52%)	1,956 (41%)
	Girls	5,840 (48%)	2,804 (59%)
Gestational age at birth (weeks)		39.45 (1.86)	39.48 (1.84)
Birthweight (grams)		3415 (546)	3416 (535)
Maternal age at birth (years)		28.3 (4.5)	29.4 (4.6)
Maternal education			
	Less than A-levels	7,271 (60%)	2,416 (51%)
	A-levels or higher	4,831 (40%)	2344 (49%)
Eating behaviour trajectories			
Overeating	Low overeating	8,240 (69%)	3,311 (70%)
	Low transient	1,756 (15%)	606 (13%)
	Late increasing	1,276 (11%)	552 (12%)
	Early increasing	730 (6%)	291 (6%)
Undereating	Low undereating	2,940 (24%)	1,134 (24%)
	Low transient	4,413 (37%)	1,805 (38%)
	Low and decreasing	2,454 (20%)	927 (19%)
	High transient	1,548 (13%)	628 (13%)
	High decreasing	437 (4%)	181 (4%)
	High persistent	214 (2%)	84 (2%)
Fussy eating	Low fussy eating	2,713 (23%)	983 (21%)
	Low transient	1,718 (14%)	737 (15%)
	Low and increasing	3,272 (27%)	1,258 (26%)

	Early and decreasing	1,710 (14%)	736 (15%)
	Rapidly increasing	1,590 (13%)	607 (13%)
	High persistent	1,045 (9%)	439 (9%)

Table 2. Distribution of outcome variables measured at age 16 years, separate for boys and girls

Outcome Measures		
	Boys (n = 1956)	**Girls (n = 2,804)**
Eating disorder behaviours		
Fasting	58/1921 (3%)	562/2760 (20.4%)
Purging	25/1927 (1.3%)	263/2779 (9.5%)
Binge eating	75/1921 (3.9%)	438/2767 (15.8%)
Excessive exercise	28/1828 (1.5%)	175/2526 (6.9%)
Eating disorder diagnoses		
Anorexia nervosa	19/1956 (1%)	69/2804 (2.5%)
Bulimia nervosa	n<0/1956 (0%)	37/2804 (1.3%)
Binge-eating disorder	11/1956 (0.6%)	42/2804 (1.5%)
Purging disorder	n<5/1956 (0.3%)	69/2804 (2.5%)

Table 3. Estimated baseline risks (BR) and risk differences (RD) by overeating trajectories and outcomes at age 16 years, adjusted for sex, gestational age, birthweight, maternal age, and maternal education

Childhood Overeating Trajectories			
Binge eating (N=4354)			
	BR	**95% CI**	***p-value***
Low overeating (N=3271)$	0.096	0.086**,** 0.106	<0.001*
	RD	**95% CI**	***p***
Low transient (N=598)	0.024	-0.005, 0.053	0.106
Late increasing (N=548)	**0.057**	**0.027**, **0.087**	**<0.001***
Early increasing (N=290)	**0.069**	**0.025**, **0.112**	**0.002***
Purging (N=4706)			
	BR	**95% CI**	***p-value***
Low overeating (N=3271)$	0.024	-0.005, 0.053	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=598)	0.011	-0.009, 0.034	0.269
Late increasing (N=548)	0.011	-0.007, 0.037	0.184
Early increasing (N=290)	0.017	-0.003, 0.065	0.078
Binge-eating disorder (N=4706)			
	BR	**95% CI**	***p-value***
Low overeating (N=3311)$	0.009	0.001, 0.012	<0.001*
	RD	**95% CI**	***p-value***
Low transient (N=606)	0.005	-0.001, 0.016	0.335
Late increasing (N=552)	**0.014**	**0.002, 0.026**	**0.024**
Early increasing (N=291)	0.006	-0.01**,** 0.02	0.357

BR= Baseline risk; CI = Confidence interval, N = number, RD = Risk difference; $= reference group
Associations between overeating trajectories and PD were not adjusted for sex due to collinearity, as PD was not common in boys (<0.5%). * below p-value after Bonferroni correction by the number of comparisons, 0.02.

Childhood Undereating Trajectories			
Excessive exercise (N=4354)			
	BR	**95% CI**	***p-value***
Low undereating (N=1023)[$]	0.058	0.043, 0.073	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=1672)	-0.010	-0.028, 0.008	0.284
Low and decreasing (N=839)	-0.025	-0.044**,** -0.005	0.012
High transient (N=576)	-0.009	-0.032**,** 0.015	0.477
High decreasing (N=167)	-0.016	-0.050**,** 0.019	0.380
High persistent (N=77)	-0.008	-0.060, 0.044	0.762
Fasting (N=4681)			
	BR	**95% CI**	***p-value***
Low undereating (N=1112)[$]	0.149	0.129, 0.170	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=1777)	**-0.026**	**-0.051, -0.001**	**0.043**
Low and decreasing (N=914)	**-0.031**	**-0.059, -0.002**	**0.034**
High transient (N=614)	-0.012	-0.046, 0.022	0.488
High decreasing (N=176)	0.004	-0.053, 0.062	0.881
High persistent (N=84)	0.024	-0.059, 0.108	0.564
Anorexia nervosa (N=4760)			
	BR	**95% CI**	***p-value***
Low undereating (N=1134)[$]	0.016	0.008, 0.023	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=1805)	-0.002	-0.012, 0.007	0.612
Low and decreasing (N=927)	0.008	-0.004, 0.020	0.207
High transient (N=628)	0.007	-0.007, 0.021	0.301
High decreasing (N=181)	0.014	-0.014, 0.042	0.335
High persistent (N=85)	0.037	-0.004, 0.079	0.077

Table 4. Estimated **b**aseline risks (BR) and risk differences (RD) by undereating trajectories and outcomes age 16 years, adjusted for sex, gestational age, birthweight, maternal age and maternal education

BR= Baseline risk; RD = Risk difference; $= reference group

Associations with excessive exercise were not adjusted for sex, and maternal education due to collinearity

Table 5 Estimated **b**aseline risks (BR) and risk differences (RD) by fussy eating trajectories and outcomes age 16 years, adjusted for sex, gestational age, birthweight, maternal age and maternal education

Childhood Fussy Eating Trajectories			
Excessive exercise (N=4354)			
	BR	**95% CI**	***p-value***
Low fussy eating (N=895)$	0.055	0.040, 0.070	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=681)	-0.014	-0.036,0.007	0.189
Low and increasing (N=1132)	-0.003	-0.023**,** 0.017	0.779
Early and decreasing (N=684)	-0.015	-0.036**,** 0.006	0.168
Rapidly increasing (N=554)	-0.015	-0.037**,** 0.007	0.178
High persistent (N=406)	-0.011	-0.037**,** 0.014	0.389
Fasting (N=4681)			
	BR	**95% CI**	***p-value***
Low fussy eating (N=971)$	0.149	0.129, 0.170	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=726)	-0.030	-0.061, 0.001	0.060
Low and increasing (N=1232)	-0.007	-0.035, 0.021	0.621
Early and decreasing (N=722)	-0.003	-0.035, 0.029	0.868
Rapidly increasing (N=599)	-0.016	-0.049, 0.017	0.339
High persistent (N=431)	-0.019	-0.059, 0.108	0.309
Anorexia nervosa (N=4760)			
	BR	**95% CI**	***p-value***
Low fussy eating (N=938)$	0.012	0.005, 0.019	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=737)	-0.002	-0.012, 0.008	0.712
Low and increasing (N=1258)	0.005	-0.005, 0.014	0.371
Early and decreasing (N=736)	**0.020**	**0.005**, **0.034**	**0.007**

Rapidly increasing (N=607)	0.001	-0.011, 0.012	0.903
High persistent (N=439)	**0.022**	**0.004**, **0.040**	**0.017**

BR = Baseline risk; RD = Risk difference; $= reference group

Supplement Tables

Supplement Table 1a: Associations between overeating trajectories and disordered eating and EDs outcomes age 16 years, girls only

Childhood overeating			
Binge eating, girls only (n= 2,767)			
	BR*	**95% CI**	***p-value***
Low overeating$ (N=1929)	0.15	0.13, 0.16	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=333)	0.01	-0.04, 0.05	0.705
Late increasing (N=369)	**0.07**	**0.03, 0.12**	**0.001**
Early increasing (N=136)	0.07	-0.01, 0.15	0.052
Purging, girls only (n=2,779)			
	BR	**95% CI**	***p-value***
Low overeating $ (N=1938)	0.08	0.07, 0.10	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=334)	0.03	-0.01, 0.07	0.104
Late increasing (N=370)	0.02	-0.01, 0.05	0.185
Early increasing (N=137)	0.06	-0.004 , 0.13	0.066
Binge eating disorder, girls only (n= 2,804) (not adjusted for maternal education)			
	BR	**95% CI**	***p-value***
Low overeating$ (N=1937)	0.01	0.01, 0.02	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=336)	0.01	-0.01, 0.03	0.217

Late increasing (N=372)	**0.02**	**0.00, 0.04**	**0.016**
Early increasing (N=138)	0.01	-0.02, 0.04	0.501

BR= Baseline risk; RD = Risk difference, $ Reference class

Supplement Table 1b. Associations between undereating trajectories and disordered eating and EDs outcomes age 16 years, girls only

Childhood undereating			
Anorexia Nervosa, girls only **(N=2,804)**			
	BR*	**95% CI**	***p-value***
Low undereating[$] (N=679)	0.02	0.01, 0.03	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=1,050)	0.00	-0.01, 0.02	0.760
Low and decreasing (N=565)	0.01	0.00, 0.03	0.156
High transient (N=363)	0.01	-0.01, 0.03	0.273
High decreasing (N=93)	0.02	-0.02, 0.06	0.376
High persistent (N=54)	**0.06**	**0.00, 0.13**	**0.043**
Fasting, girls only (N=2,760)			
	BR*	**95% CI**	***p-value***
Low undereating [$] (N=667)	0.23	0.20, 0.26	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=1,035)	**-0.04**	**-0.08, 0.00**	**0.033**
Low and decreasing (N=557)	-0.04	-0.09, 0.01	0.113
High transient (N=359)	-0.02	-0.08, 0.03	0.442
High decreasing (N=89)	0.00	-0.09, 0.09	0.975
High persistent (N=53)	0.00	-0.13, 0.12	0.991
Bulimia Nervosa, girls only (N= 2,804)			
	BR*	**95% CI**	***p-value***
Low undereating [$] (N=679)	0.02	0.001, 0.03	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=1,050)	-0.01	-0.02, 0.001	0.336
Low and decreasing (N=565)	-0.01	-0.02, 0	0.060
High transient (N=363)	0.01	-0.01, 0.022	0.454
High decreasing (N=93)	0.02	-0.02, 0.05	0.364
High persistent (N=54)	0.01	-0.01, 0.05	0.605
Excessive exercise, girls only (N=2,526)			
	BR*	**95% CI**	***p-value***

Low undereating [$] (N=600)	0.08	0.06, 0.11	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=962)	-0.01	-0.04, 0.02	0.476
Low and decreasing (N=503)	**-0.03**	**-0.06, 0.00**	**0.040**
High transient (N=330)	-0.02	-0.05, 0.02	0.327
High decreasing (N=83)	0.01	-0.05, 0.08	0.699
High persistent (N=48)	0.00	-0.08, 0.09	0.920

[$] Reference class; BR= Baseline risk; RD = Risk difference

Supplement Table 1c. Associations between fussy eating trajectories and disordered eating and EDs outcomes age 16 years, girls only

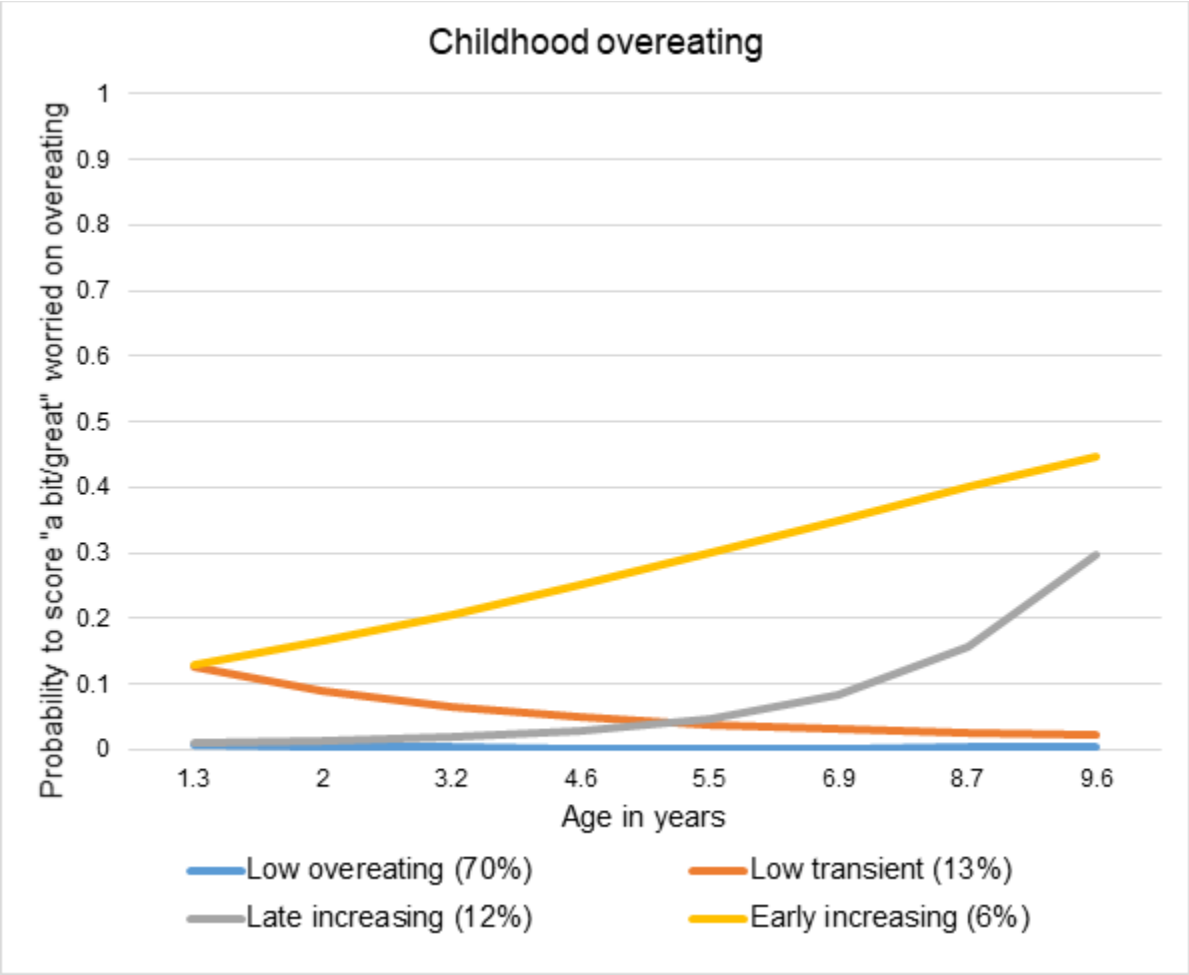
Childhood fussy eating			
Anorexia Nervosa, Girls only (N=2,804)			
	BR*	**95% CI**	***p-value***
Low fussy eating [$] (N=605)	0.02	0.01, 0.03	<0.001
	RD*	**95% CI**	***p-value***
Low transient (N=404)	0.00	-0.02, 0.01	0.780
Low and increasing (N=753)	0.00	-0.01, 0.01	0.935
Early and decreasing (N=455)	0.03	0.01, 0.05	0.009
Rapidly increasing (N=347)	-0.01	-0.02, 0.01	0.414
High persistent (N=240)	0.03	0.00, 0.06	0.029
Fasting, girls only (N=2,760)			
	BR*	**95% CI**	***p-value***
Low fussy eating [$] (N=598)	0.21	0.18, 0.24	<0.001
	RD*	**95% CI**	***p-value***
Low transient (N=400)	-0.04	-0.09, 0.01	0.153
Low and increasing (N=735)	0.00	-0.04, 0.05	0.961
Early and decreasing (N=447)	-0.01	-0.06, 0.04	0.766
Rapidly increasing (N=345)	0.01	-0.05, 0.06	0.801
High persistent (N=235)	-0.03	-0.09, 0.03	0.302
Excessive exercise, girls only (N=2,526)			
	BR*	**95% CI**	***p-value***
Low fussy eating [$] (N=538)	0.08	0.06, 0.10	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=369)	-0.02	-0.05, 0.02	0.338
Low and increasing (N=669)	0.00	-0.03, 0.03	0.920
Early and decreasing (N=420)	-0.03	-0.06, 0.00	0.087
Rapidly increasing (N=314)	-0.02	-0.05, 0.02	0.378
High persistent (N=216)	-0.02	-0.06, 0.03	0.460

Bulimia Nervosa, girls only (N=2,804)			
	BR*	**95% CI**	***p-value***
Low fussy eating [$] (N=605)	0.01	0.00, 0.02	<0.001
	RD	**95% CI**	***p-value***
Low transient (N=404)	0.01	-0.01, 0.03	0.253
Low and increasing (N=753)	-0.00	-0.01, 0.01	0.602
Early and decreasing (N=455)	0.00	-0.01, 0.02	0.558
Rapidly increasing (N=347)	-0.01	-0.02, 0.00	0.239
High persistent (N=240)	-0.01	-0.02, 0.01	0.502

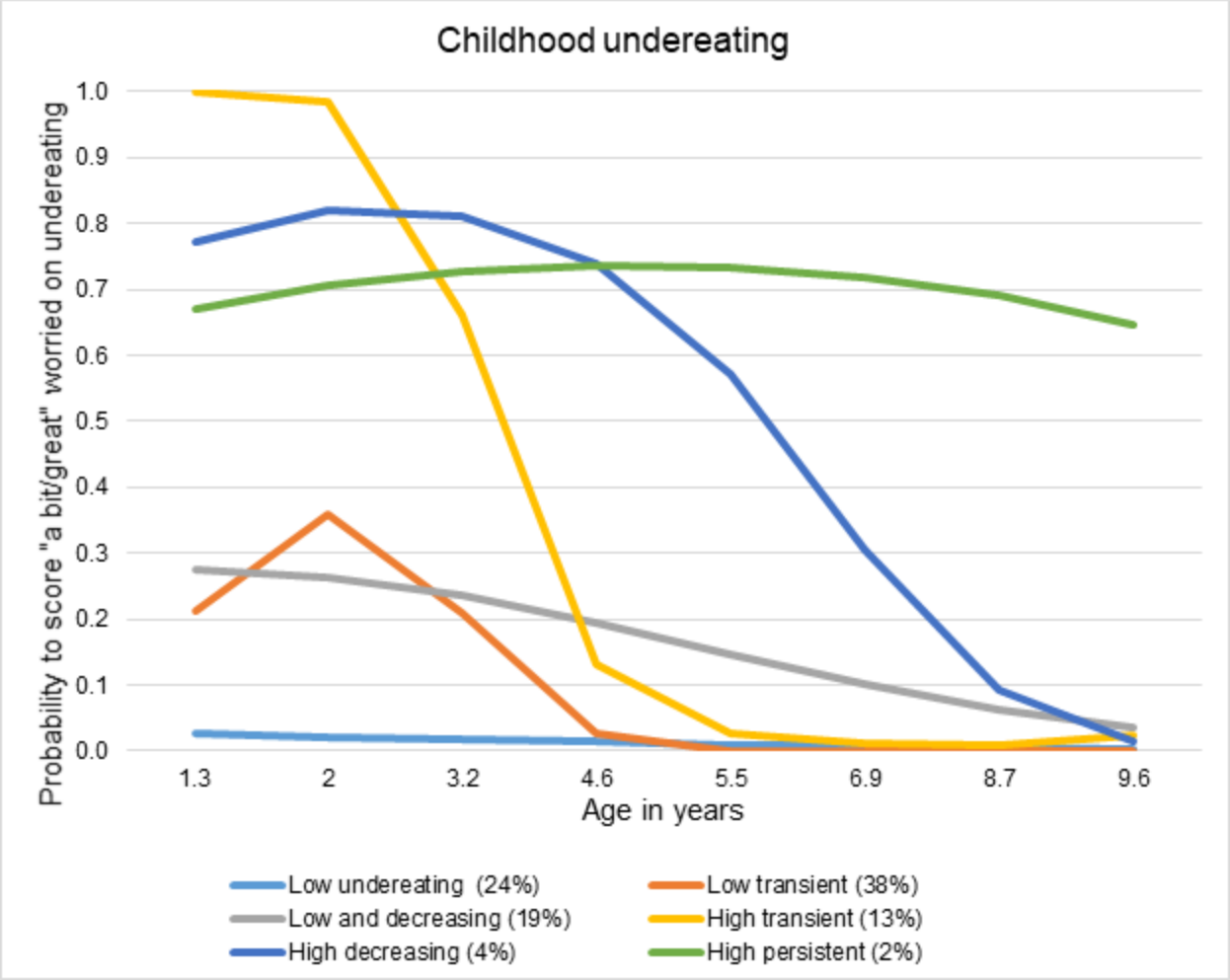
[$] Reference class; BR= Baseline risk; RD = Risk difference

Supplementary Figures

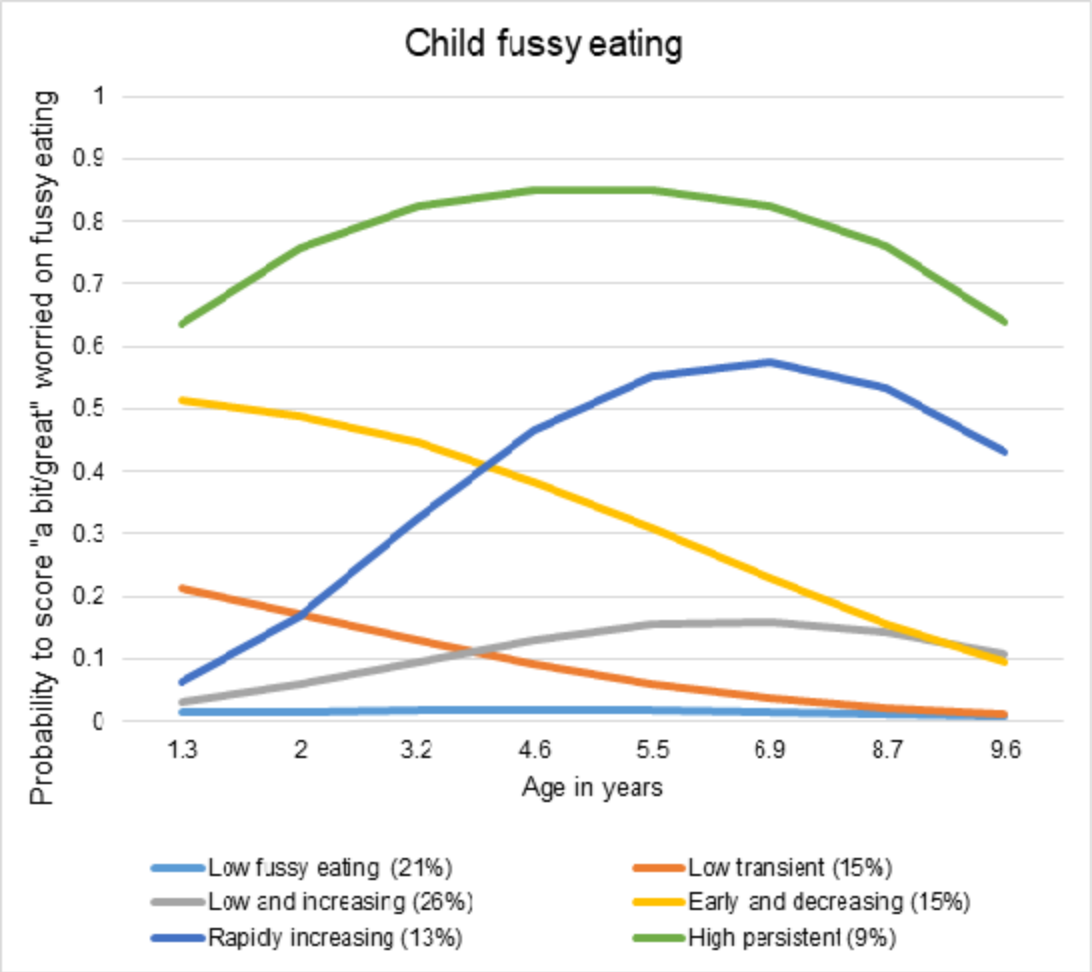
Supplement Figure 1. Groups of childhood overeating across the first 10 years of life

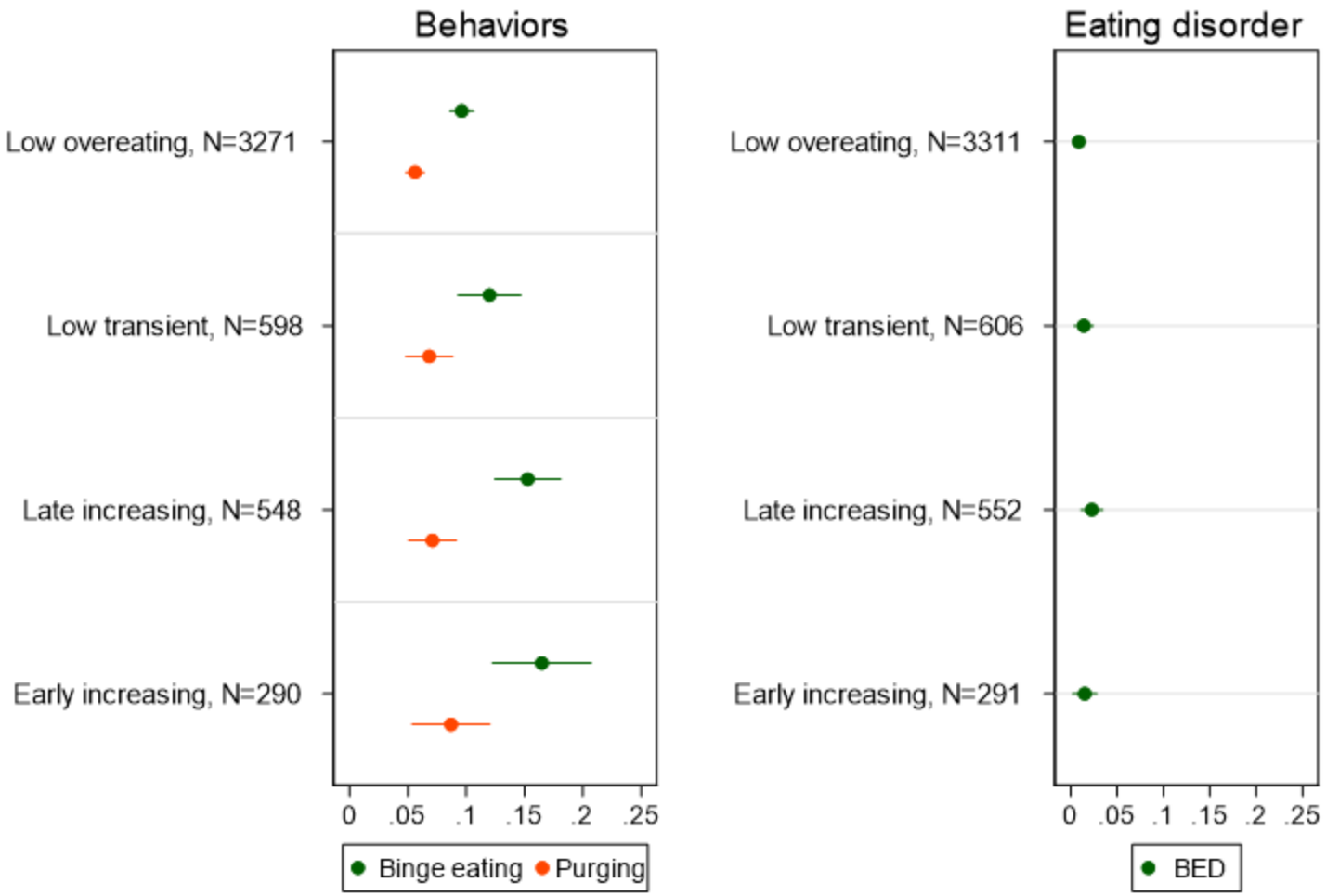


Supplement Figure 2. Groups of childhood undereating across the first 10 years of life

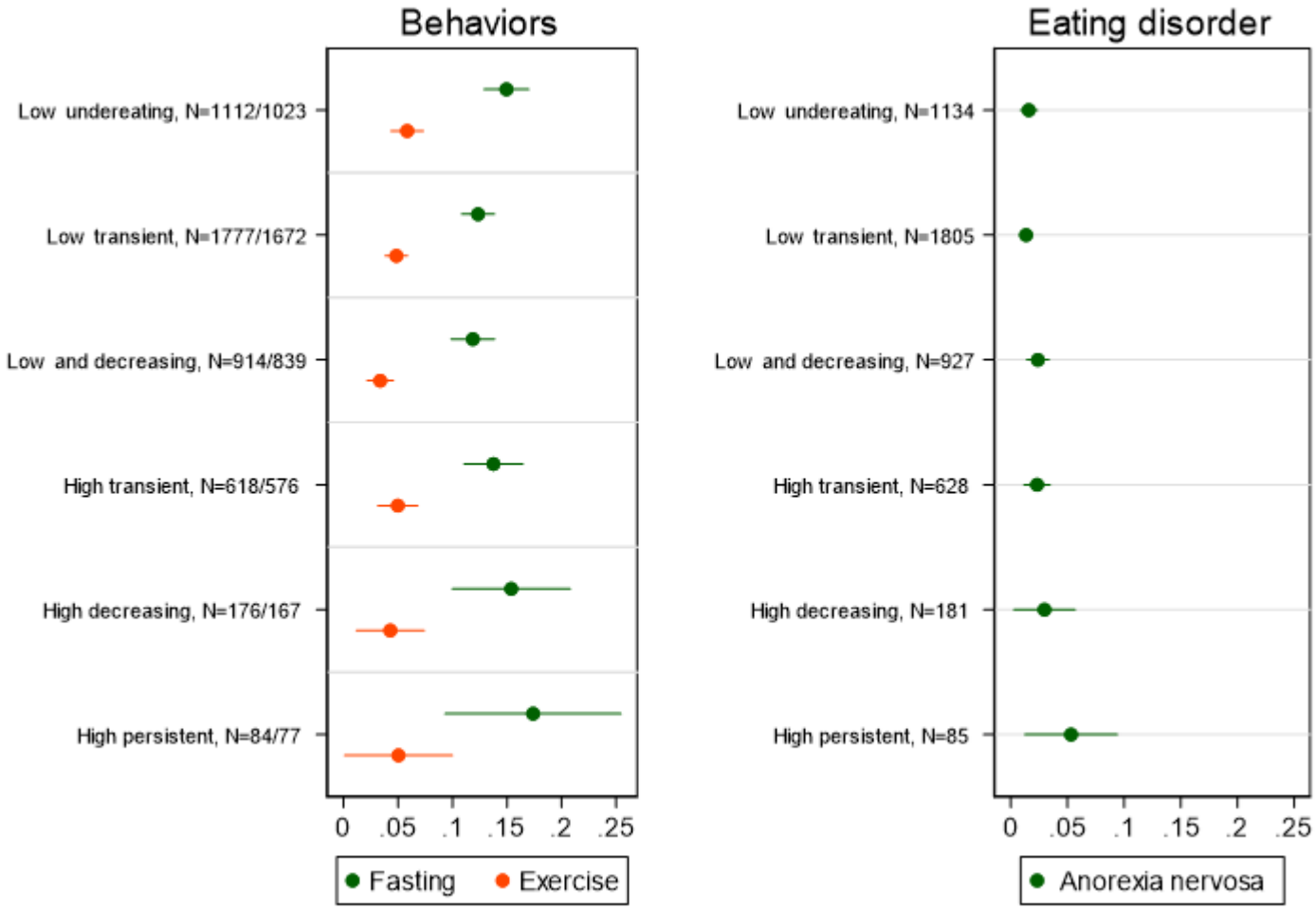


Supplement figure 3. Groups of childhood fussy eating across the first 10 years of life

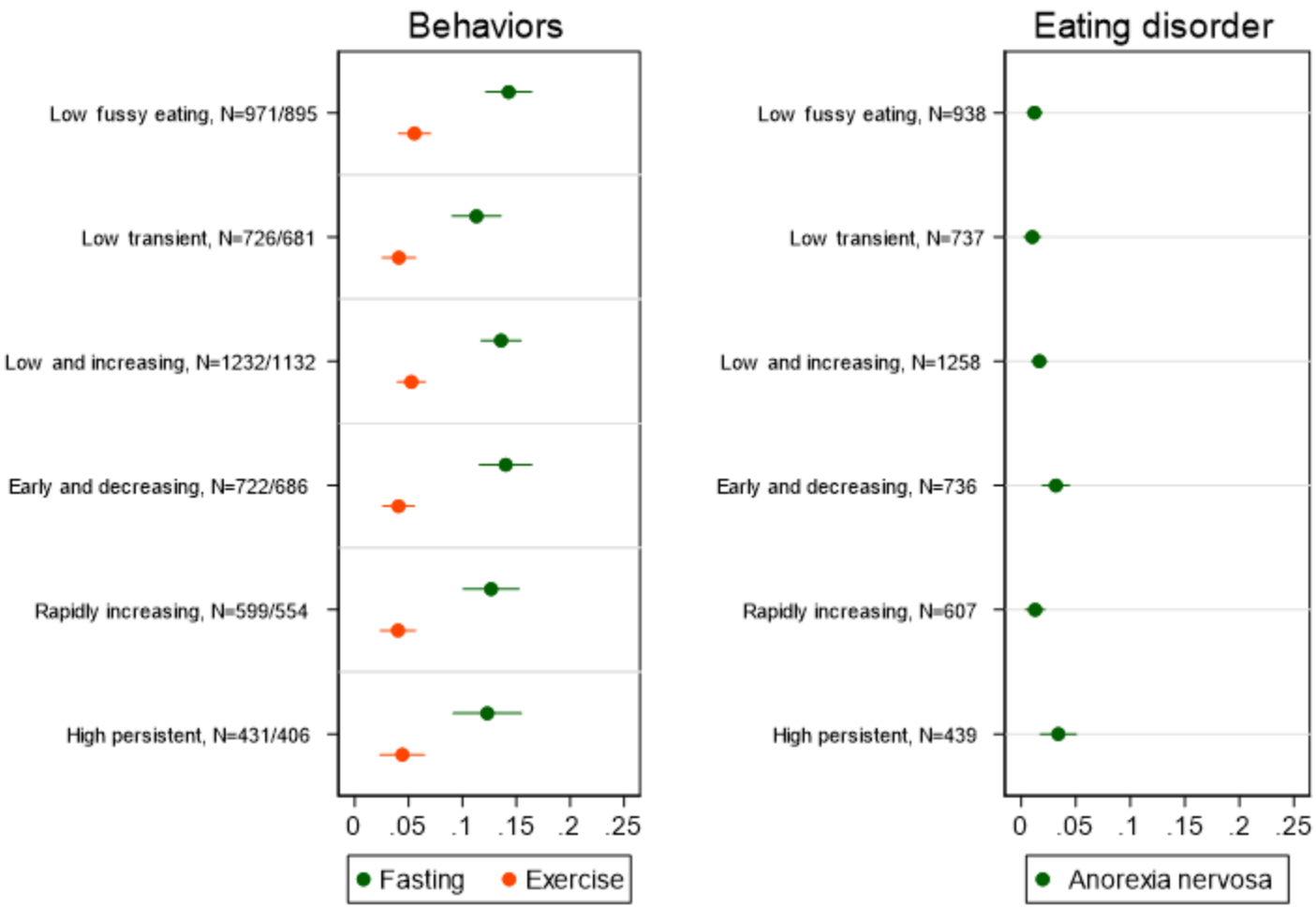




Supplement Figure 4 Estimated mean probability of disordered eating behaviors and eating disorder (ED) diagnoses by overeating trajectory, derived from logistic regression models adjusted for sex, gestational age, birthweight, maternal age, and maternal education. BED = binge-eating disorder



Supplement Figure 5 Estimated mean probability of disordered eating behaviors and eating disorder (ED) diagnoses by undereating, derived from logistic regression models adjusted for sex, gestational age, birth weight, maternal age, and maternal education



Supplement Figure 6 Estimated mean probability of disordered eating behaviors and eating disorder (ED) diagnoses by fussy eating trajectory, derived from logistic regression models, adjusted for sex, gestational age, birthweight, maternal age, and maternal education