Early childhood appetitive traits and eating disorder symptoms in adolescence: a 10-year longitudinal follow-up study in the Netherlands and the UK

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Summary

Background Obesity and eating disorders commonly co-occur and might share common risk factors. Appetite avidity is an established neurobehavioural risk factor for obesity from early life, but the role of appetite in eating disorder susceptibility is unclear. We aimed to examine longitudinal associations between appetitive traits in early childhood and eating disorder symptoms in adolescence.

Methods In this longitudinal cohort study, we used data from Generation R (based in Rotterdam, the Netherlands) and Gemini (based in England and Wales). Appetitive traits at age 4–5 years were measured using the parent-reported Child Eating Behaviour Questionnaire. At age 12–14 years, adolescents self-reported on overeating eating disorder symptoms (binge eating symptoms, uncontrolled eating, and emotional eating) and restrictive eating disorder symptoms (compensatory behaviours and restrained eating). Missing data on covariates were imputed using Multivariate Imputation via Chained Equations. Ordinal and binary logistic regressions were performed in each cohort separately and adjusted for confounders. Pooled results were obtained by meta-analyses. Sensitivity analyses were performed on complete cases using inverse probability weighting.

Findings The final study sample included 2801 participants from Generation R and 869 participants from Gemini. Pooled findings after meta-analyses showed that higher food responsiveness in early childhood increased the odds of binge eating symptoms (odds ratio $[OR]_{pooled}$ 1·47, 95% CI 1·26–1·72), uncontrolled eating (1·33, 1·21–1·46), emotional eating (1·26, 1·13–1·41), restrained eating (1·16, 1·06–1·27), and compensatory behaviours (1·18, 1·08–1·30) in adolescence. Greater emotional overeating in early childhood increased the odds of compensatory behaviours (1·18, 1·06–1·33). By contrast, greater satiety responsiveness in early childhood decreased the odds of compensatory behaviours in adolescence (0·89, 0·81–0·99) and uncontrolled eating (0·86, 0·78–0·95) in adolescence. Slower eating in early childhood decreased the odds of compensatory behaviours (0·91, 0·84–0·99) and restrained eating (0·90, 0·83–0·98) in adolescence. No other associations were observed.

Interpretation In this study, higher food responsiveness in early childhood was associated with a higher likelihood of self-reported eating disorder symptoms in adolescence, whereas greater satiety sensitivity and slower eating were associated with a lower likelihood of some eating disorder symptoms. Appetitive traits in children might be early neurobehavioural risk factors for, or markers of, subsequent eating disorder symptoms.

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Introduction

Eating disorders are serious mental health conditions that are rapidly increasing in prevalence worldwide.[1] Eating disorders frequently coexist with other psychiatric disorders and have high mortality rates.[2,3] Because these disorders are difficult to treat, focus is shifting towards prevention and early intervention when symptoms or episodes first arise, often in adolescence. However, there are currently no effective prevention strategies. Epidemiological research is needed to identify novel modifiable risk factors and to provide new avenues for prevention and treatment.[4]

In contrast with eating disorders, much is known about the determinants of bodyweight, indexed using body-mass index (BMI). Eating disorders and BMI share many features—both are strongly dependent on food intake regulation and are moderately to highly heritable.[5,6] Eating disorders also show genetic overlap with BMI: genetic predisposition to higher BMI was linked with binge eating and disordered eating, and genetic predisposition to lower BMI was linked with anorexia nervosa.[5,7] Appetite avidity, which is observable and measurable from early life, is robustly associated with weight development[8] and a neurobehavioural mediator of genetic susceptibility to obesity—the so-called Behavioural Susceptibility Theory.[9] High levels of food approach appetitive traits (food cue responsiveness, food enjoyment, and emotional overeating) reflect an avid appetite and are linked with overweight and

Research in context

Evidence before this study

Embase, Medline, and Cochrane were searched for English content from database conception until Nov 29, 2022, using the terms "Appetite", "Eating OR Feeding with Behav* habit* OR rhythm* OR attitude", "Food fussiness", "Satiation", "Satiety Response", "Overnutrition", "Food OR eater* with fussiness OR fussy OR responsiveness* OR satiet* OR satiat* OR overnutrition* OR overeating*", combined with terms "Feeding and Eating Disorders", "Anorexia Nervosa", "Anorexia", "Bulimia", "Binge-Eating Disorder", "Eating with disorder* OR emotional OR loss-of-control* OR binge* OR restrain* OR uncontrol* OR disturb* OR abnormal* OR problem* OR dysfunction*", "Food craving", "Adverse OR harmful* with diet*". We also included search terms "Cohort Studies", "Prediction", "Risk Factors", "prospectiv* OR longitudinal* OR cohort* OR predict OR risk with factor OR population" with "Research*" to select prospective studies, and the terms "adolescent OR adolescen*" AND "Child", "Pediatrics", "Pre-school* OR pediatric* OR paediatric* OR young* age" to narrow the search on children and adolescents. References from relevant studies were also checked. Nine studies in six population-based cohorts examined discrete eating or mealtime behaviours in childhood as appetitive traits, with subsequent eating disorder symptoms or diagnoses. Fussy eating, general overeating, general undereating, and problems around meals were the most frequently examined appetitive traits, commonly assessed using unvalidated single items. The few studies showed conflicting results. For instance, associations of undereating or fussy eating with anorexia nervosa diagnosis or symptoms were inconsistent across studies. Similar patterns were found for overeating traits and subsequent bulimia nervosa or binge eating disorder symptoms and diagnoses. None of these studies examined the full range of appetitive traits, such as hedonic eating (food responsiveness and enjoyment of food), homoeostatic appetite control (satiety responsiveness and slowness in eating), emotional eating (emotional overeating and emotional undereating), and fussy eating.

Added value of this study

This longitudinal study with up to 10 years of follow-up included adolescents (age 12–14 years) from two independent cohorts in the Netherlands and the UK, with harmonised measures of appetite and eating disorder symptoms. We used the Child Eating Behaviour Questionnaire, the most comprehensive and widely used psychometric measure of child appetitive traits that has been independently validated with objective measures of eating behaviour in children from different countries. We found several early childhood appetitive traits to be associated with eating disorder symptoms in early adolescence. Heightened responsiveness to food cues was consistently associated with higher odds of restrained eating, compensatory behaviours, binge eating symptoms, uncontrolled eating, and emotional eating. Emotional overeating in early childhood was associated with higher odds of compensatory behaviours. However, greater satiety sensitivity was associated with decreased risk of engaging in compensatory behaviours and uncontrolled eating, whereas slower pace of eating was associated with reduced risk of compensatory behaviours and restrained eating. Food fussiness, emotional undereating, and enjoyment of food in early childhood showed no association with any eating disorder symptoms in adolescence.

Implications of all the available evidence

Appetitive traits in early childhood might be neurobehavioural risk factors for eating disorder symptoms in adolescence. In particular, heightened food cue responsiveness might confer susceptibility to eating disorder pathology, whereas strong sensitivity to internal satiety signals and slower eating speed might be protective. A healthy food environment and responsive parental feeding strategies, known to influence the development of appetite in early life, might therefore have the potential to prevent both obesity as well as eating disorders.

obesity, whereas high levels of food avoidance appetitive traits (fussiness or picky eating, emotional undereating, satiety sensitivity, and slow eating rate) reflect a poor or restrictive appetite and are linked with underweight.[8]

The Behavioural Susceptibility Theory could be extended to eating disorders: early-life appetitive traits might also have a role in susceptibility to eating disorders. Key eating pathologies of anorexia nervosa (severe restriction, aversion to satiety, and emotional undereating) and bulimia nervosa or binge eating disorder (uncontrolled and rapid eating, and emotional overeating) are at the extremes of appetitive traits that drive early weight development. Few population-based studies have examined prospective associations between childhood appetite and subsequent eating disorder symptoms and diagnoses, and existing studies mostly used unvalidated single items of eating or mealtime behaviours, such as general overeating.[10–15] Findings have been inconsistent and, to date, no studies have examined prospective associations between the full range of appetitive traits in childhood with later eating disorder symptoms, using a validated measure of appetite.

This study examined associations between appetitive traits in early childhood and eating disorder symptoms in early adolescence, using a comprehensive, validated measure of appetite in two independent cohorts in the Netherlands and the UK. The focus here is on behavioural eating disorder symptoms (eg, restrictive eating and binge eating) rather than cognitive symptoms (eg, body image distortion), in line with the Behavioural Susceptibility Theory. We hypothesised that: (1) high food approach traits (and low food avoidance traits) characterising an avid appetite predispose to overeating eating disorder symptomatology (binge eating, uncontrolled eating, and

emotional eating); and (2) high food avoidance traits (and low food approach traits) characterising a poor appetite predispose to restrictive eating disorder symptomatology (restrained eating). We did not formulate a directional hypothesis for compensatory behaviours because they could be considered restrictive but also relate strongly to overeating symptomatology.

Methods

Study design and population

In this longitudinal cohort study, we used data from Generation R and Gemini. The Generation R Study is a population-based cohort situated in Rotterdam, the Netherlands, designed to examine genetic and environmental pathways towards normal and abnormal growth, development, and health. All pregnant women with an expected delivery date between April, 2002, and January, 2006, were invited to participate, resulting in 9745 live-born children.[16] Gemini is a population-based cohort of twins born in England and Wales in 2007, and originally included 4804 children (2402 twin pairs). Gemini was set up to examine genetic and environmental contributions to early growth, with a focus on the roles of appetite and the home family environment. In 2017, additional funding was awarded by MQ Mental Health Research to examine the role of appetite and parental feeding practices in the development of eating disorder symptoms in early adolescence (12–14 years).[17]

Generation R has been approved by the Medical Ethical Committee of the Erasmus Medical Center Rotterdam. Written informed consent was obtained from all parents in early childhood, and all parents and children provided written informed consent in early adolescence. Gemini was granted ethical approval in 2007 through the University College London Committee for the Ethics of non-National Health Service Human Research. Families consented to take part at study inception. Families then reconsented to take part in the new phase of the study (on appetite and eating disorders) in 2018.

Procedures and outcomes

The primary exposure of the study is appetitive traits, measured with the Child Eating Behaviour Questionnaire (CEBQ, 35 items).[18] The CEBQ is the most comprehensive psychometric measure that assesses the full range of appetitive traits in children and adolescents. It has good internal and test–retest reliability (Cronbach's alphas for all scales in this study ranged from 0·70 to 0·91) and is validated using objectively measured eating behaviour.[19] The CEBQ is freely available in 14 languages and widely used.[18,19]

When children were aged 4 years (Generation R) and 5 years (Gemini), parents completed the CEBQ,[18] consisting of eight appetitive traits (including one on drinking, excluded from this study). Higher scores on three food approach scales reflect a more avid appetite: food responsiveness measures children's responsiveness towards external food cues (five items); enjoyment of food assesses the extent of pleasure derived from eating (four items); and emotional overeating assesses the tendency to eat in order to soothe negative emotions (four items). Higher scores on four food avoidance scales reflect a poorer appetite: food fussiness assesses pickiness or selectivity in eating (six items); satiety responsiveness assesses sensitivity towards internal satiety cues (five items); slowness in eating measures eating speed (four items); and emotional undereating assesses the tendency to eat less in response to negative emotions (four items). Items were scored on a five-point Likert scale (never to always). Mean item subscale scores were calculated and ranged from 1·00 to 5·00.

When children were aged 12–13 years (Gemini) and 14 years (Generation R), they self-reported on eating disorder symptoms. The self-reported outcome measures were the same for both cohorts and include both behavioural eating disorder symptoms (compensatory behaviours and binge eating symptoms) and disordered eating behaviours (restrained eating, uncontrolled eating, and emotional eating).

Compensatory behaviours were defined as the number of compensatory behaviours that were present in the past 3 months. Eight compensatory behaviours were assessed using items from the Developmental and Well-Being Assessment (DAWBA): purging, medication use, fasting for all or most of the day, hiding or throwing away food, exercising more, eating less during meals, skipping meals, and avoiding foods that are perceived to lead to weight gain.[20] The DAWBA generates diagnoses according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) and shows good validity for eating disorders.[20] The response scale per behaviour ranged from zero (no) to three (one time per week) in Generation R, and from zero (no) to four (more than one time per week) in Gemini. Any response higher than no was considered as symptom being present. Symptom occurrences were summed (zero to eight) and, due to a skewed distribution, categorised (no behaviours; 1 behaviour; 2–3 behaviours; and ≥4 behaviours).

Restrained eating was defined as the extent to which food intake was restricted to manage or lose weight and assessed with the restrained eating subscale of the Dutch Eating Behaviour Questionnaire (DEBQ, ten items).[21] A mean item score was calculated from a four-point Likert scale (ranging from never to one or more times per week) and, due to skewed data distribution, categorised into tertiles.

Binge eating symptoms were also assessed by the DAWBA and defined as the presence of any binge eating symptom (overeating, loss of control eating, or both) in the past 3 months.[20] A description of an overeating episode was provided, followed by questions about whether and how often this happened in the past 3 months (ranging from did not happen to one or more times per week). Loss of control eating was assessed in

the same way. Due to small sample sizes in each category (overeating, loss of control eating, or both), symptoms were collapsed into presence or absence of any symptoms in the past 3 months (yes or no).

Uncontrolled eating was defined as the extent to which someone feels out of control and eats more than usual. Emotional eating was defined as the presence of eating in response to negative feelings. Both were measured with subscales of the Three Factor Eating Questionnaire (TFEQ), revised version of 18 items.[22] TFEQ items were rated on a four-point Likert scale, ranging from definitely false to definitely true. For uncontrolled eating, mean item scores from nine items were calculated and categorised into tertiles due to the skewed nature of the data. Emotional eating consisted of three items and because scores were low, we dichotomised the obtained mean item scores into presence or absence of any emotional eating (yes or no).

Several variables that have previously been associated with appetite and eating disorders were included as covariates in the analyses:adolescent's age at outcome assessment, biological sex, ethnicity, gestational age, sex-adjusted and age-adjusted BMI SD scores at age 4–5 years; and mother's education level, BMI, and household income.[23–25] Sex and ethnicity were parent-reported in both cohorts.

Statistical analysis

Cohort-specific analyses were performed in R version 4.2.1 (Gemini) and 4.2.2 (Generation R). Associations between appetite and compensatory behaviours, restrained eating, and uncontrolled eating were examined using ordinal logistic regression. Associations between appetite and binge eating symptoms and emotional eating were examined using binary logistic regression. In model one we adjusted for sociodemographic covariates (age at outcome assessment, biological sex, gestational age, ethnicity, household income, maternal education, and maternal BMI); in model two we also adjusted for child's sex-adjusted and age-adjusted BMI SD scores at age 4–5 years. Analyses in Gemini were additionally adjusted for clustering of twins in families using the survey package in R.[26] Missing data on covariates were not missing completely at random (MCAR) in both cohorts, as indicated by significant Little's MCAR tests (Generation R: p=0·00792; Gemini: p=0·00119), but were missing at random or missing not at random. In both instances, multiple imputation techniques are preferred to obtain less biased results and to preserve the maximum sample size.[27] Thus, covariates were imputed using Multivariate Imputation via Chained Equations, with extra predictor variables (eg, sex-adjusted and age-adjusted BMI SD scores at other ages) enhancing the estimation of imputed values. A maximum of 50 iterations were used to create 20 imputed datasets. Random-effects meta-analysis in Stata MP 17 was used to obtain weighted pooled results for fully adjusted odds ratios (ORs) and 95% CIs of both cohorts. Significant results were indicated by 95% CIs not crossing 1·00.

	Generation R (n=2801)		Gemini (n=869)	
	Total number	n (%) or mean (SD)	Total number	n (%) or mean (SD)
Demographic characteristics				
Sex				
Female	2801	1491 (53·2%)	869	447 (51·4%)
Male	2801	1310 (46·8%)	869	422 (48·6%)
Age at self-reported outcome assessment, years	2543	13·57 (0·30)	862	12·67 (0·41)
Birthweight, g	2800	3451·13 (579·27)	853	2462·42 (529·68)
Gestational age, weeks	2793	39·82 (1·87)	869	36·31 (2·50)
Sex-adjusted and age-adjusted BMI SD score at 4–5 years	1844	0·04 (0·90)	532	–0·29 (1·07)
Ethnicity				
Dutch or White British	2795	2054 (73·5%)	869	744 (85·6%)
Non-Dutch or Non-White British	2795	741 (26·5%)	869	125 (14·4%)
Maternal age at inclusion, years	2801	32·15 (4·21)	869	34·96 (4·32)
Maternal BMI at baseline, kg/m²*	2575	24·83 (4·51)	855	24·39 (4·19)
Maternal educational level†				
High	2676	1903 (71·1%)	869	512 (58·9%)
Medium	2676	641 (24·0%)	869	258 (29·7%)
Low	2676	132 (4·9%)	869	99 (11·4%)
Household income‡				
High	2573	1160 (45·1%)	850	231 (27·2%)
Medium	2573	1234 (48·0%)	850	439 (51·6%)
Low	2573	179 (7·0%)	850	180 (21·2%)
Appetitive traits§				
Food fussiness	2801	2·94 (0·81)	869	2·75 (0·81)
Food responsiveness	2801	1·77 (0·66)	869	2·33 (0·73)
Emotional overeating	2801	1·43 (0·58)	869	1·55 (0·50)
Emotional undereating	2801	2·78 (0·82)	869	2·71 (0·83)
Food enjoyment	2801	3·40 (0·70)	869	3·89 (0·67)
Satiety responsiveness	2801	3·08 (0·65)	869	2·87 (0·61)
Slowness in eating	2801	3·14 (0·77)	869	2·81 (0·78)
Restrictive eating disorder symptoms				
Compensatory behaviours¶				
No occurrence	2801	1348 (48·1%)	869	394 (45·3%)
Low occurrence (1 behaviour)	2801	481 (17·2%)	869	197 (22·7%)
Mild occurrence (2–3 behaviours)	2801	678 (24·2%)	869	187 (21·5%)
High occurrence (≥4 behaviours)	2801	294 (10·5%)	869	91 (10·5%)
Restrained eating‖				
Low	2801	992 (35·4%)	869	327 (37·6%)
Moderate	2801	910 (32·5%)	869	276 (31·8%)
High	2801	899 (32·1%)	869	266 (30·6%)

(Table 1 continues on next page)

Interaction effects for appetite by biological sex were examined. Because we took a conservative approach to characterising binge eating symptoms (missing data on one of the two items was imputed as no symptom), we ran sensitivity analyses excluding imputed data for binge eating symptoms. Analyses for compensatory behaviours were also repeated separately with the item exercise more, as this item might not reflect disordered eating behaviour per se. Finally, to check for the robustness of our findings,

	Generation R (n=2801)		Gemini (n=869)	
	Total number	n (%) or mean (SD)	Total number	n (%) or mean (SD)
(Continued from previous page)				
Overeating eating disorder symptoms				
Binge eating symptoms¶				
No symptoms present	2801	2472 (88·3%)	869	785 (90·3%)
Any symptoms present	2801	329 (11·7%)	869	84 (9·7%)
Uncontrolled eating**				
Low	2801	1062 (37·9%)	869	308 (35·5%)
Moderate	2801	827 (29·5%)	869	274 (31·5%)
High	2801	912 (32·6%)	869	287 (33·0%)
Emotional eating**				
Not present	2801	2127 (75·9%)	869	583 (67·1%)
Any present	2801	674 (24·1%)	869	286 (32·9%)

BMI=body-mass index. Sample characteristics are based on original data. In Generation R, missing data ranged from 0 (sex and age at outcome assessment) to 957 (child BMI at 4 years); in Gemini, this ranged from 0 (sex) to 337 (child BMI at 5 years). *Baseline is in the first trimester of pregnancy in Generation R and child age around 8 months in Gemini. †Maternal education was measured in Generation R at age 5 years (high: higher vocational training or university; medium: lower vocational training; or low: up to high school education) and in Gemini at age around 8 months (high: university-level education; medium: vocational or advanced high-school education; or low: no qualifications or basic high-school education). ‡Household income was measured in Generation R at age 5 years (high: ≥€4000 per month; medium: €1600–4000 per month; or low: <€1600 per month) and in Gemini at age around 8 months (high: >£67 500 [high income]; medium: £30 000–67 500 [average UK income]; or low: <£30 000 [less than average UK income]). §Measured with the Child Eating Behaviour Questionnaire. ¶Measured with the Development And Wellbeing Assessment. ||Measured with the Dutch Eating Behaviour Questionnaire. **Measured with the Three Factor Eating Questionnaire.

Table 1: **Sample characteristics**

See Online for appendix

analyses were repeated (model two only) in weighted complete cases (n=1475 in Generation R and n=427 in Gemini) and subsequently pooled with random-effects meta-analysis. Inverse probability weights were created in the larger cohort with data available on appetitive traits (n=4845 in Generation R and n=2070 in Gemini) to reduce potential selection bias.[28] Weights were based on the following demographic characteristics: child's biological sex, age, gestational age at birth, ethnicity, and BMI SD score at age 4–5 years, as well as maternal educational level, maternal BMI at baseline, and household income.

For the analysis plan in **Open Science Framework** see https://osf.io/tv348

The analysis plan was pre-registered in the Open Science Framework, which includes full details on measures, missing data approaches, and analyses.

Role of the funding source

The funders had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

For both cohorts, the final sample consisted of participants who had completed most items (>60%) for each appetitive trait scale, most items (≥60%) for each disordered eating scale (restrained eating, uncontrolled eating, and emotional eating), at least one of two items for binge eating symptoms, and five of eight items for compensatory behaviours.

In Generation R, consent was obtained for 7295 children to participate in the early childhood phase of the study. Of those with consent, information on appetitive traits at age 4 years was available for 4845 children. The final study sample from Generation R consisted of 2801 children who self-reported on eating disorder symptoms at age 14 years (28·7% of all live-born children). In Gemini, appetitive traits were reported by parents for 2070 children aged 5 years, and the final study sample consisted of 869 children who also self-reported on eating disorder symptoms at age 12–13 years (18·1% of those originally included at baseline). In both cohorts, participants lost to follow-up for eating disorder symptoms at age 12–14 years (n=2044 in Generation R; n=1201 in Gemini) had younger mothers, lower maternal education and household income, and a higher BMI at baseline than did those who were included in the analyses. Participants lost to follow-up were more often male, had a higher sex-adjusted and age-adjusted BMI SD score at age 4–5 years, and were more often non-Dutch or non-White-British. In Generation R, participants lost to follow-up had higher scores on emotional overeating but lower scores on emotional undereating, enjoyment of food, and slowness in eating compared with the Generation R participants in the final study sample. Those lost to follow-up in Gemini scored higher on food responsiveness compared with the Gemini participants in the final study sample (all $p<0·05$). Descriptive characteristics of the final study sample and non-response sample are shown in the appendix (pp 2–5).

Average follow-up periods were 10 years for Generation R and 7·5 years for Gemini. Complete data on exposures, outcomes, and covariates were present for 1475 (52·7%) of the final Generation R study sample and for 427 (49·1%) of the final Gemini study sample. Cohorts were similar across most characteristics, including mean scores for appetitive traits and frequency of eating disorder symptoms (table 1). In both cohorts, the presence of at least one compensatory behaviour was reported by more than half of the participants (1453 [51·9%] of 2801 in Generation R; 475 [54·7%] of 869 in Gemini), and 10·5% of participants in both cohorts (294 of 2801 in Generation R and 91 of 869 in Gemini) reported the presence of four or more compensatory behaviours. However, compared with Generation R participants, Gemini participants had lower gestational age, birthweight, and sex-adjusted and age-adjusted BMI SD score at aged 4–5 years. Mothers in Generation R were slightly younger and more often had a higher educational level and household income than did mothers in Gemini, and Generation R participants were more diverse in ethnic background.

The pooled meta-analysis results from the fully adjusted models (model two), which were similar to model one (no adjustment for child sex-adjusted and age-adjusted BMI SD score at 4–5 years of age), are presented in table 2. Cohort-specific results are presented in the appendix (pp 6–10). Forest plots of the pooled results are presented in the appendix (pp 11–17).

	Restrictive eating disorder symptoms		Overeating eating disorder symptoms		
	Compensatory behaviours*	Restrained eating†	Binge eating symptoms‡	Uncontrolled eating†	Emotional eating‡
Food approach traits					
Food responsiveness	1·18 (1·08–1·30)	1·16 (1·06–1·27)	1·47 (1·26–1·72)	1·33 (1·21–1·46)	1·26 (1·13–1·41)
Enjoyment of food	0·98 (0·89–1·07)	0·96 (0·88–1·05)	1·24 (0·81–1·90)	1·06 (0·97–1·16)	1·05 (0·91–1·21)
Emotional overeating	1·18 (1·06–1·33)	1·18 (0·97–1·44)	1·13 (0·95–1·36)	1·24 (0·92–1·66)	1·14 (1·00–1·32)
Food avoidance traits					
Satiety responsiveness	0·89 (0·81–0·99)	0·94 (0·85–1·04)	0·91 (0·69–1·22)	0·86 (0·78–0·95)	0·99 (0·87–1·11)
Slowness in eating	0·91 (0·84–0·99)	0·90 (0·83–0·98)	0·85 (0·57–1·28)	0·95 (0·87–1·02)	1·00 (0·91–1·10)
Food fussiness	1·10 (0·94–1·30)	1·10 (0·97–1·24)	0·95 (0·84–1·08)	1·01 (0·94–1·09)	1·08 (0·98–1·19)
Emotional undereating	0·96 (0·89–1·04)	0·97 (0·90–1·05)	0·94 (0·82–1·06)	1·03 (0·96–1·11)	1·01 (0·92–1·11)

Data are pooled OR (95% CI). Effect sizes presented are pooled results from Gemini and Generation R obtained by meta-analysis. For compensatory behaviours, restrained eating, and uncontrolled eating, ORs were derived from ordinal logistic regression analyses. For binge eating symptoms and emotional eating, ORs were derived from binary logistic regression analyses. Before meta-analyses, cohort-specific analyses were adjusted for adolescent's biological sex, age at outcome assessment, gestational age at birth, ethnicity, maternal education, household income, maternal BMI at baseline, and child sex-adjusted and age-adjusted BMI SD score at 4–5 years, and twin clustering for Gemini. Statistically significant results are shown by 95% CIs not crossing 1·00. OR=odds ratio. *Outcome categories were no occurrence, low occurrence (1 behaviour), mild occurrence (2–3 behaviours), and high occurrence (≥4 behaviours). †Outcome categories were low, moderate, and high. ‡Outcome categories were not present and any present.

Table 2: **Pooled odds ratios (from meta-analyses) of associations between mean item scores of appetitive traits in early childhood (4–5 years) and eating disorder symptoms in early adolescence (12–14 years) across Generation R and Gemini (n=3670)**

The results reflecting associations between food approach appetitive traits and eating disorder symptomatology are presented in table 2. In line with our hypothesis, greater food responsiveness in early childhood increased the odds of all overeating eating disorder symptoms in adolescence. The largest risk was for binge eating symptoms (OR_{pooled} 1·47, 95% CI 1·26–1·72, per unit increase in food responsiveness; table 2).

Contrary to our hypothesis, greater food responsiveness also increased the odds of restrictive eating disorder symptoms. Specifically, food responsiveness was associated with an increased risk of moderate or high restrained eating (1·16, 1·06–1·27, per unit increase).Food responsiveness and emotional overeating in early childhood were also associated with an increased risk of compensatory behaviours in adolescence (OR_{pooled} 1·18, 1·08–1·30, and 1·18, 1·06–1·33, per unit increase). No other pooled associations between food approach appetitive traits and eating disorder symptoms were observed.

Cohort-specific results showed that higher enjoyment of food increased the odds of binge eating symptoms in Gemini (OR_{Gemini} 1·62, 95% CI 1·06–2·45), but this was not observed in Generation R ($OR_{Generation\ R}$ 1·04, 0·88–1·22; appendix p 8). Furthermore, higher emotional overeating increased the odds of uncontrolled eating in Gemini (OR_{Gemini} 1·48, 1·12–1·96), but not in Generation R ($OR_{Generation\ R}$ 1·09, 0·97–1·23; appendix p 9).

The results reflecting associations between food avoidance appetitive traits and eating disorder symptomatology are presented in table 2. Contrary to our hypothesis, food avoidance traits in early childhood did not increase the odds of engaging in restrictive eating disorder symptoms in adolescence. Instead, slowness in eating decreased the odds of moderate or high restrained eating (OR_{pooled} 0·90, 95% CI 0·83–0·98, per unit increase). Greater satiety responsiveness and slowness in eating also significantly reduced the risk of engaging in compensatory behaviours (OR_{pooled} 0·89, 0·81–0·99, and 0·91, 0·84–0·99, respectively; table 2).

In line with our hypothesis, higher satiety responsiveness in early childhood decreased the odds of uncontrolled eating at age 12–14 years (OR_{pooled} 0·86, 95% CI 0·78–0·95)—ie, weaker satiety was associated with increased likelihood of uncontrolled eating. No other pooled associations between food avoidance appetitive traits and eating disorder symptoms were observed.

Cohort-specific results showed that higher slowness in eating in early childhood decreased the odds of binge eating symptoms in Gemini (OR_{Gemini} 0·67, 95% CI 0·47–0·95), but this association was not observed in Generation R ($OR_{Generation\ R}$ 1·02, 0·88–1·19; appendix p 8). Furthermore, higher food fussiness increased the odds of restrained eating in Gemini (OR_{Gemini} 1·20, 1·01–1·42) but not in Generation R ($OR_{Generation\ R}$ 1·05, 0·96–1·14; appendix p 7).

Several sensitivity analyses were run to check for the robustness of our findings. Some significant interactions were seen between appetite and biological sex on eating disorder symptoms (appendix p 18). In Generation R, the association between slowness in eating in early childhood and lower odds of restrained eating in adolescence was observed in female participants only. In Gemini, associations of emotional overeating in early childhood with higher odds of binge eating symptoms and more emotional eating in adolescence were observed in male participants only. Sensitivity analysis showed that results in adolescents with complete data on binge eating symptoms and imputed binge eating symptoms were similar (data were imputed for 36 children in Generation R and eight children in

Gemini; appendix p 19). Furthermore, findings remained similar after excluding the item exercise more from the compensatory behaviours outcome, and analyses conducted on the item exercise more only also showed similar results to the original compensatory behaviours outcome (appendix pp 20–21).

The pooled results from the weighted complete cases were mostly in line with the main results: food approach traits tended to be associated with increased odds of eating disorder symptoms, whereas food avoidance traits (specifically slowness in eating and satiety responsiveness) tended to be associated with decreased odds of eating disorder symptoms (appendix p 22). Additionally, effect sizes of pooled results from weighted complete cases tended to be larger compared with the main results, and more associations were statistically significant. However, most associations between appetitive traits and compensatory behaviours were null, with the two exceptions being enjoyment of food (OR_{pooled} 0·90, 95% CI 0·84–0·97) and emotional overeating (1·14, 1·04–1·24; appendix p 22).

Discussion

To our knowledge, this study is the first observational and longitudinal examination of associations between the full range of appetitive traits observable in early childhood and eating disorder symptoms in early adolescence. We hypothesised that higher food approach traits in early childhood would predispose to overeating eating disorder symptomatology in adolescence, whereas higher food avoidance traits would predispose to restrictive eating disorder symptomatology. Our findings suggest that some food approach traits in early childhood might predispose to eating disorder symptoms in adolescence, whereas some food avoidance traits might confer protection. Specifically, heightened responsiveness to food cues—a key food approach trait—was consistently associated with a higher frequency or occurrence of eating disorder symptoms. Furthermore, emotional overeating was associated with a higher frequency of compensatory behaviours. However, two key food avoidance traits—greater sensitivity to satiety and a slower pace of eating—were associated with decreased risks of some eating disorder symptoms (eg, restrained and uncontrolled eating). Variation in food fussiness, emotional undereating, and food enjoyment in early childhood was hardly associated with any eating disorder symptoms in adolescence.

Heightened food responsiveness is a key early-life neurobehavioural trait that appears to be a robust risk factor or early marker for broad-spectrum eating disorder symptomatology (both overeating and restrictive). This finding is important because overeating and restrictive eating disorder symptoms often co-occur within eating disorders (eg, in bulimia nervosa or anorexia nervosa binge/purge type) or might trigger one another (eg, binge eating episodes are often preceded by restrained eating, which can drive binge eating episodes). No studies have examined food responsiveness specifically as a predictor for eating disorder symptomatology, but our consistent findings across two cohorts, robustness across sensitivity analyses, and pooled effect sizes amplify its importance. A 1-unit increase in the food responsiveness scale (eg, parents reporting that their child "sometimes" *vs* "rarely" demonstrates those behaviours) corresponded with a 47% increased risk of self-reporting binge eating symptoms about 10 years later. Adolescents whose parents rated them highest (five) on the food responsiveness scale in early childhood were, therefore, nearly three times more likely to report binge eating symptoms than were adolescents whose parents scored them lowest (one). These results align with previous findings showing that general overeating or eating in the absence of hunger in childhood is associated with increased risk of binge eating disorder symptoms[10,15,29] and restrained eating[30] later in life. The association between food responsiveness and eating disorder symptoms could be explained by increased food-related attentional bias and reward sensitivity, a characteristic of adolescents with binge eating disorder and anorexia nervosa.[31,32] Our findings suggest that these aberrations in executive functions might originate early in life and that food responsiveness could be a mediator of genetic susceptibility to both eating disorders and obesity.

Our findings suggested that emotional overeating in early childhood increased the risk of compensatory behaviours in adolescence. Emotional overeating develops as a maladaptive coping strategy for negative emotions and, according to twin studies, is a learned behaviour.[33] Eating to mitigate negative emotions might result in subsequent feelings of guilt and shame, which, in turn, might lead to the urge to compensate. Surprisingly, emotional overeating in early childhood was not associated with overeating eating disorder symptoms, including emotional eating, in early adolescence. One possible explanation is that emotional overeating in early childhood is a different behaviour to emotional eating in adolescence (for example, in early childhood, it might be parent-led, whereas in adolescence it might be more self-directed). Moreover, as a behaviour that is entirely learned, the environmental influences on emotional eating might differ markedly in early childhood and adolescence, leading to lower stability over time.

Partially contradicting our hypothesis, higher scores for two food avoidance appetitive traits—satiety responsiveness and slowness in eating—were negatively associated with some eating disorder symptoms and might therefore protect adolescents from engaging in restrained and uncontrolled eating, and potentially compensatory behaviours. These findings align with studies showing that individuals living with eating disorder symptoms might experience impairments in their interoceptive awareness (ie, the individual's awareness of internal body

states, including hunger and satiety cues).[34,35] Deficits in interoceptive awareness might induce individuals to rely more on external factors or cognitions (such as external food cues or feelings of reward) regarding when and how much to eat, instead of eating intuitively (ie, in response to internal hunger and satiety cues). High interoceptive awareness to internal satiety cues might indicate greater precision in appetite regulation, such that children who are sensitive to internal satiety mechanisms might intuitively stop eating when they feel full and do not, therefore, develop feelings of loss of control while eating or the following urge to compensate.

Previous studies have suggested links between other food avoidance appetitive traits such as general undereating, fussy eating, and mealtime struggles with later anorexia nervosa symptoms,[10,12,14] yet our pooled null findings of fussy eating and emotional undereating with eating disorder symptoms do not reflect this. Comparison is, however, difficult given that our study did not include anorexia nervosa diagnosis specifically. Food avoidance traits have also been linked with avoidant restrictive food intake disorder. For instance, increased satiety sensitivity and emotional undereating, and decreased enjoyment of food, were associated with avoidant restrictive food intake disorder symptoms in a community-based sample of children at age 10 years.[36] Given that avoidant restrictive food intake disorder and anorexia nervosa share similar characteristics, such as avoidance of food, and that the eating disorder symptoms investigated in this study do not relate to avoidant restrictive food intake disorder, further exploration of the relevance of appetitive profiles for anorexia nervosa and avoidant restrictive food intake disorder in clinical and population-based samples would be useful.

In this study, eating disorder symptoms were examined in early adolescence, whereas the peak age of onset at diagnosis is currently estimated at 15·5 years for anorexia nervosa and bulimia nervosa and 19·5 years for binge eating disorder.[37] This implies that some adolescents included in this study did not experience eating disorder symptoms at this age but might develop these symptoms later. However, the later onset or diagnosis of binge eating disorder might be partially explained by less noticeable symptoms for caregivers compared with anorexia nervosa, and therefore a delay in help-seeking behaviour. Binge eating episodes often take place in secret, and physical consequences can develop more slowly (if at all) and are more difficult to observe in comparison to the apparent, rapid, and sometimes life-threatening physical consequences of anorexia nervosa. Indeed, in this study, eating disorder symptoms were already frequently observed, especially compensatory behaviours. The experience of eating disorder symptoms during early adolescence could reflect a prodromal stage in which it might be possible to intervene with low-intensity treatment to prevent diagnosed eating disorders.[38,39] Nonetheless, the aetiology of eating disorder symptom onset in early adolescence might be different from eating disorder symptom onset later in life, and the predictive value of risk factors for eating disorders might vary over time, as shown previously.[40] Therefore, further investigation on the predictive value of appetite at other ages, as well as associations with eating disorders later in adolescence and adulthood, is required.

In line with the Behavioural Susceptibility Theory,[9] appetite avidity (characterised by high responsiveness to food cues) in early life confers increased risk of eating disorder symptoms, whereas a smaller appetite (characterised by high sensitivity to satiety and slow eating) provides protection. However, future studies are needed to examine the extent to which variation in appetite mediates part of the genetic susceptibility to eating disorders to further investigate the behavioural susceptibility to eating disorders.

Our findings could provide new opportunities for treatment and prevention of eating disorders. Although appetitive traits in childhood are under moderate to high genetic influence, with heritability estimates ranging from 59% to 75% for food responsiveness, 63% to 72% for satiety responsiveness, and 63% to 84% for slowness in eating,[41,42] there is also a sizeable influence of the environment on each of these and, importantly, they are modifiable. Although relatively new, behavioural interventions targeting food cue reactivity and satiety sensitivity through appetite awareness training and cue-exposure therapy have shown promising results for reducing both binge eating and weight in adults who were living with overweight or obesity as well as binge eating.[43] These interventions might be particularly beneficial for adolescents living with prodromal eating disorders, before symptoms become persistent and severe. Furthermore, our findings highlight the potential importance of the food environment (including but not limited to density of fast-food outlets) and parental feeding strategies for eating disorders (such as controlling feeding strategies), which are already well established environmental targets for the prevention and treatment of obesity.[44,45] Parental feeding strategies are known to be modifiable. Responsive feeding (ie, responding contingently with children's hunger and satiety in the context of structured mealtimes, rather than offering food for non-nutritional purposes) can help children to eat intuitively (ie, in response to their internal hunger and satiety cues), rather than in response to hedonic hunger elicited by the presence of food cues (ie, food responsiveness).[46] Thus, teaching parents and children how to recognise and respond to internal feelings of hunger and satiety, encourage slower eating at mealtimes, and not to offer or eat food for reasons other than homoeostatic hunger (eg, in response to hedonic hunger or using food as a tool to soothe negative emotions or reward or punish behaviour) are practices that support the development of healthy eating habits. Indeed, a randomised controlled trial with a responsive feeding

intervention for mothers of infants resulted in lower scores of food responsiveness and higher scores of satiety responsiveness up to 3·5 years of follow-up.[47]

If findings of our study are replicated, the potential usefulness of the CEBQ as a screening tool for health-care professionals to identify young children at high risk of an eating disorder later in life based on their appetite could be explored. For example, the CEBQ has already been successfully adapted as a screening tool for clinically relevant selective eating according to a psychiatric interview.[48] Targeted support and education on intuitive eating and responsive feeding could be provided to families of children with an avid appetite to reduce the risk of eating disorder symptoms developing. Finally, our findings also highlight the need for up-stream public health policies to focus on regulation of the food environment to minimise food cues, which will reduce the expression of food cue responsiveness for children who are particularly vulnerable to overeating in response to environmental triggers.

A strength of our study is the use of two large population-based cohorts with harmonised, validated, and comprehensive measures of child appetite and eating disorder symptoms. We did, however, observe some differences in characteristics between cohorts (eg, in ethnic distribution and socioeconomic background), which could reflect cultural differences. Generation R has a more diverse ethnic background that reflects the multi-ethnic population of Rotterdam, and the differences in infant and child characteristics are probably because the Gemini participants are twins. Although the mean scores of appetitive traits and reports of eating disorder symptoms were highly similar between the two cohorts, associations between appetite and eating disorder symptoms might vary between populations, especially considering high and low obesogenic food environments. Although most associations between appetite and eating disorder symptoms were highly similar between cohorts, a few cohort-specific differences were observed. We therefore suggest a cautious interpretation and recommend future replication. Moreover, self-reported eating disorder symptoms might have resulted in under-reporting due to social desirability. Relatedly, to account for skewed distributions of variables, we created somewhat arbitrary cutoff points for eating disorder symptoms, rather than clinically meaningful thresholds. However, eating disorders are spectrum disorders with symptoms existing on a continuum. Additionally, final study samples were more representative of those with a higher socioeconomic status and predominantly White (Gemini) and Dutch (Generation R) backgrounds, indicating the need for replication in more economically and ethnically diverse populations. We approached this bias using two different methods: namely, multiple imputation of missing values in covariates and running a complete case analysis using inverse probability weights. Results mostly pointed in the same directions, although with slight increases in effect sizes. However, the largely null findings with compensatory behaviours in the weighted complete case analyses require further investigation in other more diverse samples. Finally, the prospective and observational nature of this study and methods used preclude any causal conclusions.

Appetitive traits, which are observable and measurable from early life, might be neurobehavioural risk factors for eating disorder symptoms, extending the Behavioural Susceptibility Theory. Consistent associations were observed between heightened food responsiveness and eating disorder symptoms, whereas greater satiety sensitivity and slower eating rate were negatively associated with some eating disorder symptoms and therefore could potentially offer protection. An appetite pathway in susceptibility to eating disorders carries notable public health implications: appetitive traits are not expressed in the absence of environmental opportunity—factors such as parental feeding practices, availability of food, and environmental food cues all potentially interact with neurobehavioural traits, such as food responsiveness. These factors are important new areas to incorporate into existing models of prevention and management for eating disorders that currently focus largely on individual psychological risk factors.

Contributors

IPMD, ZN, HAH, ARK, PWJ, and CHL designed the current study. IPMD and HAH accessed and verified the data from Generation R, and ZN and ARK accessed and verified the data from Gemini. IPMD and ZN performed the statistical analyses, in close collaboration with HAH and ARK and were supervised by PWJ and CHL. All authors contributed to the interpretation of the results. JT provided important clinical and scientific expertise. IPMD and ZN were supervised by PWJ and CHL and drafted the initial manuscript, which was read and commented on by all authors multiple times. All authors read and approved the final manuscript before submission. PWJ and CHL were responsible for the decision to submit the manuscript.

Declaration of interests

We declare no competing interests.

Data sharing

Generation R: The analysis plan, statistical code, and data from this study are available upon reasonable request to the director of the Generation R Study (generationr@erasmusmc.nl), subject to local, national, and European rules and regulations. Gemini: Anonymised data in the manuscript, code book, and analytic code can be made available upon reasonable request after publication. Requests can be made by downloading and completing the data access form available through the Gemini study website which further details the data sharing process (https://www.geministudy.co.uk/data-access). Requests will be reviewed by the Gemini Executive Committee in accordance with the Gemini Data Access Policy.

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