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Child food neophobia is heritable, associated with less compliant eating, and moderates familial resemblance for BMI

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Abstract

Objective—The heritability of food neophobia, the tendency to avoid new foods, was tested in 4- to 7-year-old twins. We also examined whether food neophobia is associated with parent-child feeding relations or child body fat.

Design and Methods—We studied sixty-six same-sex twin pairs, including 37 monozygotic (MZ) and 29 dizygotic (DZ) pairs. Food neophobia was assessed by parent questionnaire (Child Food Neophobia Scale, CFNS), as were child feeding practices and “division of responsibility” feeding relations. Child anthropometry and percent body fat were directly measured.

Results—MZ and DZ twin pair correlations for food neophobia were $r=0.71$ and $r=-0.01$, respectively; heritability= 72%. Greater food neophobia was associated with reduced child eating compliance of prompted foods ($p<0.001$) reduced eating compliance of initially refused foods ($p<0.001$), and – among girls only – fewer parental food demands ($p=0.01$). Interestingly, the correlation between maternal BMI and child BMI z-score was significant only for children high ($p=0.03$), but not low ($p=0.55$), in food neophobia.

Conclusions—Child food neophobia, a highly heritable trait previously linked to emotionality, was associated with less compliant parent-child feeding relations. Strategies to combat food neophobia and foster more harmonious feeding relationships may have a role in obesity prevention.

Keywords

Food Neophobia; Behavioral Genetics; Childhood Obesity; Feeding Relationships

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Introduction

Food neophobia is the tendency to avoid eating unfamiliar foods^{1, 2}. In children, increased food neophobia is associated with reduced fruit and vegetable intake^{3, 4}, a poorer “healthy eating index”⁵, less varied food preferences⁶, greater anxiety,⁴ greater emotionality⁷, and a more negative reaction to food⁷. Perhaps in part for these reasons, consumption of a “limited variety” diet by children was one of the two most common eating problems reported by parents in a UK population-based study⁸.

Family factors appear critical to the development of food neophobia. Parents shape the home food environment and child food preferences⁹, and the same may be true of food neophobia. Indeed, parents who consumed a more varied diet had children who were less food neophobic¹⁰. Children’s apprehension about trying new foods was reduced by increasing the availability of these foods at home and by parents tasting these items themselves¹¹. Finally, multiple studies have shown that food neophobia is familial, which may reflect similarities in the home environment. In a study of 81 siblings pairs who were 5 to 11 years old, Pliner and Lowen⁷ reported significant mother-child correlations for maternally reported food neophobia ($r = 0.23, p < 0.01$). Galloway and Birch⁴ reported significant correlations among mothers and their 7-year old daughters ($r = 0.20, p < 0.01$), while Falciglia et al.¹⁰ reported significant parent-child correlations for food neophobia in 9–11 years old youth. A population-based study of 722 Swedish families reported that food neophobia scores were correlated among mothers and children at ages 11, 13, 15, and 17 years¹².

While suggestive of home environmental influences, family correlations also may reflect genetic factors. Genes influence behavioral traits in childhood, including eating in the absence of hunger¹³ and 24-hour dietary intake patterns¹⁴. The same may be true of food neophobia in early childhood. Cooke et al.¹⁶ estimated genetic and environmental influences on food neophobia in 8 to 11 year old twins enrolled from the UK^{16, 17}. Results indicated that 78% of the variance in parent-reported food neophobia was due to additive genetic factors, with the remaining variance attributable to non-shared (i.e., ‘unique’ or ‘random’) environmental factors. Similar estimates were reported in a twin study of Finnish adults¹⁸, which found that 69% of the variance in reported food neophobia¹² was due to genetic factors. To our knowledge, no study to date has explored potential genetic influence on food neophobia in early childhood. As genetic influences on eating traits can be age specific¹⁹, heritability estimates for food neophobia may not be generalizable to earlier childhood. Genetic influences on BMI are in fact age specific, increasing during childhood.²⁰

It is also unclear whether food neophobia is associated with variations in child body fat. Food neophobic children may tend to underconsume, relative to their energy needs, due to their reluctance to try new foods, although data on this topic are lacking. In one of the few studies conducted with children, Lumeng et al. did not find a significant association between food neophobia and body mass index (BMI: kg/m^2) z-score in 3 to 6 year old children²¹. To our knowledge no study has examined the association using refined body fat measures.

We conducted a pediatric twin study that was designed to quantify genetic contributions to child eating patterns and body fat, “*Project Grow-2-Gether*”²². The present report had three

aims. First, we tested the relative influence of genetic and environmental factors on food neophobia in this sample of 4 to 7 year old children. Based on prior studies with older children and adults^{15, 18}, we hypothesized that genes would account for at least 50% of the variance in this eating trait. We also hypothesized that greater food neophobia would be associated with poorer parent-child feeding relations, but did not make predictions regarding potential associations with child body fat.

Methods

Participants

Sixty-six same-sex twin pairs, 4 – 7 years of age, and their mothers participated. Families were recruited through twins' clubs, *Twins Magazine*, and mailings to parents of twins. All ethnicities and both sexes were eligible. Only same-sex twin pairs were recruited. Participating twins were in good health and had no food allergies or other medical conditions that would prohibit participation. Additional details of the sample are provided elsewhere²².

Measures

Child Food Neophobia Scale—The CFNS is a 10-item instrument that assesses the degree of food neophobia, or avoidance of new foods^{1, 2}. The instrument was completed by mothers for each twin. The CFNS has a range of 10 – 70, with higher scores reflecting greater amounts of the trait. Each question is answered on a 1 (Disagree Strongly) to 7 (Agree Strongly) likert scale. Sample items include: “*My child is constantly sampling new and different foods.*” and “*My child is very particular about the foods she will eat.*” The CFNS has an internal consistency of Cronbach’s $\alpha=0.88$ and a test-retest correlation of Pearson’s $r=0.80-0.90^2$. The CFNS has been used often with young children^{3, 5, 23–25}.

Child Feeding Questionnaire (CFQ)—Parental feeding styles were measured by the Child Feeding Questionnaire²⁶. The following three feeding styles were evaluated: “Restriction,” the extent to which parents attempt to restrict their child’s eating during meals. “Pressure to Eat,” parents’ inclination to pressure their child to consume more food. “Monitoring,” the degree to which parents monitor their child’s fat intake. The CFQ has been used extensively in the literature^{27, 28}.

Feeding Demand Questionnaire (FEEDS)—This 8-item questionnaire measures the extent to which parents have demanding beliefs with respect to feeding relations with their children²⁹. The FEEDS has three subscales: *Anger and Frustration*, the extent to which parents experience anger or frustration when children do not comply when eating; *Food Amount Demandingness*, the extent to which parents believe that their children should eat a specific amount of food; and *Food Type Demandingness*, the extent to which parents believe that their children should eat specific types of foods. Cronbach’s α coefficients for the subscales ranged from 0.70 – 0.86, and the instrument was written at the 4.8th grade level²⁹.

Division of Responsibility (DoR) Feeding Questions—Three DoR feeding questions from the National Longitudinal Survey of Youth³⁰ were completed by mothers.

The first question was *Child Eating Compliance of Prompted Foods* and read “When it is mealtime, how often does your child eat what you want him/her to eat?” Response options were (1) almost never, (2) less than half the time, (3) half the time, (4) more than half the time, and (5) almost always. The second question was *Child Eating Compliance of Initially Refused Foods* and read “When your child doesn’t eat what you want him/her to eat and you tell him/her to do so, how often does he/she obey and eat?” Again, response options were (1) almost never, (2) less than half the time, (3) half the time, (4) more than half the time, and (5) almost always. The third question was *Mother-Allotted Food Choice* and read “How much choice is your child allowed in deciding what foods he/she eats at breakfast and lunch?” Response options were (1) no choice, (2) little choice, (3) some choice, and (4) a great deal of choice. Previous research has examined these DoR feeding questions in relation to child weight status in a population-based sample³⁰.

Anthropometry and Body Composition—Children’s weight and height were measured by digital scale and stadiometer, respectively, and converted to body mass index (BMI). BMI values, in turn were converted to BMI z-scores and percentiles according to appropriate age- and sex-specific cut-offs³¹. Children’s waist circumferences were measured in the standing position from midway between the last rib and the iliac crest. Because waist circumference is positively correlated with child age, we computed an age-adjusted (“residualized”) waist circumference score using regression analysis. Among children of parents who completed the CFNS, dual energy X-ray absorptiometry (DXA) scans were conducted to measure children’s percent (%) body fat. Height, weight, and BMI measures were collected on 128 children, waist circumference was measured on 107 children, and DXA body fat measures were obtained on 95 children, because certain parents/children declined these measures. Mothers were asked to self-report their weights and heights, from which BMI was calculated.

Socio-demographic measures—Child sex was dummy coded for final analyses. Child ethnicity categories were: European American, African American, Hispanic, Asian, Native American, and Other/Mixed. A dummy-coded variable of European American (0) and non-European American (1) was constructed for analyses.

Zygosity—Zygosity determination was based on analysis of 10 highly polymorphic genetic markers obtained from cheek cells using buccal swabs. Genetic analyses were conducted by an independent laboratory (<http://www.affiliatedgenetics.com/>). Twin pairs who were identical for all markers were classified as monozygotic (MZ); all others were classified as dizygotic (DZ). Genetic analyses did not yield interpretable results for 7 twin pairs, for whom a parent-report questions determined child zygosity³².

Procedures—Details of the research protocol are provided elsewhere²⁷. In brief, families came to the New York Obesity Research Center, St. Luke’s-Roosevelt Hospital, for 4 assessments over two weeks²⁷. The first two visits were primarily dedicated to conducting an “energy compensation” protocol²⁸, with body composition assessed either on the third or fourth visit. Visit 1, which is when parents completed the CFNS and other questionnaires,

lasted from ~11:30AM until ~1:00PM. This study received full approval from the Institutional Review Board of St. Luke's-Roosevelt Hospital.

Data Analytic Plan—Descriptive statistics are presented as means \pm standard deviations (SD) or percentages. We tested whether food neophobia scores differed by child sex, ethnicity (dummy coded), and zygosity using independent sample t-tests. To test our first study aim, biometrical genetic analyses³³ formally estimated the magnitude of genetic, shared environmental, and non-shared environmental influences on food neophobia. The 'shared environment' refers to aspects of the environment that are identical for twins, such as foods in home cupboards and meal-time rules for all children (eg, "*no dessert until vegetables are eaten*"). The 'non-shared' environment refers to aspects of the home environment that differ among twins, including differential treatment by or interactions with parents at home as well as peers and unrelated adults out of the home. The non-shared environment also reflects measurement error.

Biometric analyses tested the goodness of fit of five competing models that fit the following parameters in different combinations: Additive Genetic influences (A), referring to multiple alleles that work additively to impact the trait; Dominant Genetic influences (D), referring to multiple alleles at the same genetic locus that work interactively to impact the trait; Shared Environment influences (C), and; Non-Shared Environment influences (E). The five competing biometrical models were: (1) A-C-E; (2) A-D-E; (3) A-E; (4) C-E and (5); and E. These five models are typically fit and tested in behavioral genetics studies, as described in Neale and Cardon's classic text.³³ We note that models 1, 2, and 3 provided estimates of "heritability" (i.e., the proportion of phenotypic variance in CFNS due to genetic factors). Models 1 and 3 only fit "additive" genetic influences – that is, the average effect on the phenotype of alleles at different genetic loci. This model does not allow for or "fit" potential interactions among alleles at same genetic loci, and so the resulting heritability estimate is sometime referred to as "narrow-sense heritability." In contrast, model 2 allows for and "fits" potential non-additive or interactive effects among alleles at the same genetic loci as well as additive genetic influences. To the extent that the observed data are more consistent with non-additive (dominant) genetic influences on the phenotype, model 2 – which includes both parameters A and D – should provide a superior fit to models 1 or 3. Heritability estimated in model 2 includes results from both parameters A and D. For further discussion, see Wray and Visscher⁴⁰. The χ^2 statistic and Akaike Information Criterion (AIC) were used to evaluate the goodness-of-fit of the competing models. A non-significant χ^2 value suggests that the data do not significantly deviate from the posited model and signifies a relatively "good" fitting model. A progressively lower AIC value signifies a progressively better fitting model. The difference in fit between competing models was tested by a likelihood ratio test asymptotically distributed as χ^2 . Our study had sufficient power (80%) to detect an additive genetic effect of 20% or greater, when assuming an "A-E" model fit to the data⁴². Because we did not know in advance which model would provide the best fit, we considered A-E to be a reasonable working model for power considerations.³⁴

For our second study aim, Pearson's correlations tested whether greater food neophobia was associated with reduced child eating compliance of prompted foods, including initially rejected foods, and reduced mother-allotted child food choice. To test our third aim,

Pearson's correlations tested whether greater food neophobia was associated with a higher child BMI z-score, waist circumference, and percent body fat. Additionally, multiple regression models examined whether food neophobia moderates the association between maternal BMI and child body fat indexes. The predictor variables in these models were maternal BMI, child CFNS scores, and the interaction between maternal BMI and CFNS. Outcome measures were child BMI z-score, waist circumference, and percent body fat, respectively, in separate models. A significant interaction would imply that the association between maternal BMI and the respective child body fat index was dependent upon child food neophobia level; a significant interaction, when detected, was probed by examining mother-child associations among children low vs. high in food neophobia based on median split (i.e., CFNS score of 40).

For all analyses, statistical tests were conducted with a two-tailed significance level $\alpha = 0.05$. For any significant findings for our second and third study aims, we re-ran the analyses using generalized estimating equations to ensure that results did not change when controlling for family membership (i.e., among twins).

Results

Sample Characteristics

Out of the original 69 families participating in *Project Grow2gether*, 66 parents completed the CFNS. Among these 66 families, the mean child age was 58.0 ± 17.5 months and 53% of the sample was female. The ethnicity breakdown was 55.3% European American, 15.2% African American, 14.4% Hispanic, 3.0% Asian, and 12.1% other or mixed racial background. Sixteen percent of the children were overweight or obese, ie, BMI-for-age 85th %. Food neophobia did not differ between boys and girls ($M_s = 41.54$ vs. 38.89 , $p = 0.29$), Caucasian and non-Caucasian children ($M_s = 38.40$ vs. 42.10 , $p = 0.14$), and MZ and DZ twins ($M_s = 39.51$ vs. 40.98 , $p = 0.56$). The mean (SD) BMI for participating mothers was 25.98 (6.9).

Heritability of Food Neophobia

The correlation for food neophobia was $r = 0.71$ and $r = -0.01$ among MZ and DZ twin pairs, respectively. Biometric analyses indicated that the model including additive genetic, dominant genetic, and non-shared environmental influences provided the best fit to the data ($\chi^2 = 3.869$, $df = 3$, $p = 0.28$, $AIC = -2.13$) (see Table 1). Specifically, 0% of the variance was estimated to be due to additive genetic influences, 72% of the variance was estimated to be due to dominant genetic influences, with the remaining variance estimated to be due to non-shared environmental factors.

Association of the Food Neophobia Scale with the Child Feeding Questionnaire, Feeding Demands Questionnaire, and Division of Responsibility (DoR) Feeding Questions

Greater food neophobia was associated with reduced child eating compliance of prompted foods ($r = -0.36$, $p < 0.001$) and reduced eating compliance of initially refused foods ($r = -0.48$, $p < 0.001$) (see Table 2). These associations were significant both among boys and girls, when the sexes were analyzed separately. Among girls only, mothers of children who

were higher in food neophobia had fewer demanding thoughts about the particular types of foods that their daughters should eat ($r = -0.29$, $p = 0.01$) (Table 2). These findings did not change in GEE analyses that controlled for family membership. Food neophobia was not associated with parental feeding styles as measured by the CFQ ($p > 0.05$) (Table 2). [We note that when using a Bonferroni-adjusted α of $0.05/29 = 0.0018$, all but one of the unadjusted associations remained significant. The association between food neophobia and maternal demand cognitions did not remain significant at the adjusted alpha level].

Association of the Food Neophobia Scale and Child Body Fat Indices

Food neophobia was not significantly associated with child BMI z-score ($r = -0.003$, $p = 0.78$), waist circumference ($r = -0.17$, $p = 0.08$), or percent body fat ($r = -0.07$, $p = 0.60$). These null findings did not change when controlling for child sex or ethnicity, nor did these findings change when testing correlations within each sex ($p > 0.05$ across analyses).

Multiple regression models found that food neophobia moderated the relationship between maternal BMI and child BMI z-score (interaction $p = 0.02$, see Table 3). This finding did not change when controlling for family membership (interaction $p = 0.018$). Probing this interaction, there was a significant correlation between maternal BMI and child BMI z-score among children at or above the median CFNS score of 40 ($r = 0.27$, $p = 0.03$); however this association was not significant among children below the median CFNS score ($r = 0.08$, $p = 0.55$). In multiple regression models in which percent body fat and age-adjusted waist circumference were the dependent variables, there was no significant interaction between maternal BMI and child food neophobia scores (see Table 3).

Discussion

The main finding of this study is that genes play a substantial role in young children's tendency to avoid new foods, accounting for 72% of the variance in this trait. This heritability estimate is comparable to those reported for 8 to 11 year old children, $h^2 = 78\%$ ¹⁵, and adults, $h^2 = 69\%$ ¹⁸. These studies also reported a significant dominant genetic effect. Thus, the magnitude of genetic influence appears to be constant across the developmental spectrum. Genes also influence children's propensity to eat in the absence of hunger¹³ and eat in response to external food cues and internal satiety signals³⁵, as well as 24-hour dietary patterns¹⁴. Thus, genes appear to influence a range of eating patterns that emerge during childhood.

Regarding environmental influences food neophobia, we found that unique life experiences that are not shared by siblings make children differ in this trait. Thus, parents' greatest influence on children's food neophobia may reside in how they treat their children differently rather than similarly, which is typical of child development³⁴. Child-specific environments can include parental feeding practices or food exposures, which may differ among siblings³⁶, as well as social factors out of the home. Peers have a powerful influence on child development, including eating behavior³⁷. Experiences that promote and protect against food neophobia need to be identified, most interestingly among siblings residing in the same household.

Another main finding of our study was that greater food neophobia was associated with reduced child eating compliance of prompted foods, including foods initially refused by children. Anecdotally, parents of food neophobic children report needing to coax their children to eat often, which can be met with child resistance and mutual frustration. The long-term impact of this dynamic on children's self-regulatory eating and energy balance is unclear. Coercing feeding patterns may promote childhood obesity⁹. Prospective studies should clarify whether food neophobia elicits parenting practices that impede children's energy balance.

We found, unexpectedly, that food neophobia moderated the association between parent BMI and child BMI z-score. That is, the well-established positive correlation³⁸ was present only for youth high in food neophobia. Heavier parents did not tend to have heavier children in families where youth were low in food neophobia. This finding needs replication. Children who are less fearful of new foods may be more inclined to sample and select a wider variety of foods, including less energy-dense and more nutrient-dense choices that better support energy balance. They might also eat a greater variety of fruits and vegetables that mitigate obesity risk. On the other hand, a more varied diet compared to a less varied one is associated with overeating in controlled studies³⁹. Prospective and experimental studies should examine these questions and replicate the interaction reported in this study.

The present findings have implications for health professionals and parents. First, our results underscore the important role of 'nature' on young children's tendency to avoid new foods. This information may be valuable to parents who believe that they alone 'make' their children food neophobic through 'bad parenting.' Pediatricians may be in a unique position to disabuse parents of such beliefs, which discussing strategies to encourage acceptance of new foods.

A second implication of our study concerns the potential importance of child-specific environments in the development of food neophobia. Efforts to reduce food neophobia within the family may need to be individualized or tailored to each child, taking their idiosyncrasies into consideration, rather than treating all children the same. Certain food exposure strategies might help increase acceptance for one siblings (e.g., role modeling by parents at the dinner table) whereas alternate strategies might work better for other siblings (e.g., providing novel foods as snacks while reading). Exposure is an effective strategy for combating food neophobia²³. How children's unique characteristics can be incorporated into food exposure strategies warrants research.

Finally, as food neophobia moderated the association between parent and child weight status, strategies to combat food neophobia may have a role in obesity prevention. Childhood obesity treatment studies might assess food neophobia at baseline, to see whether it predicts treatment outcome. More generally, it would be interesting to better understand obese children who differ in food neophobia status. These may be two different sub-groups of obese children with potentially different etiological pathways and prognoses for treatment.

Our findings should be interpreted in light of study limitations. First, we only used parent-report measures of food neophobia, without complementary behavioral assessments. This is especially important when considering the sizable difference in correlations for MZ vs. DZ twin pairs for food neophobia. This large difference may reflect, at least in part, a contrast phenomenon, wherein parents of MZ twins over-emphasize similarities between twins and parents of DZ twins over-emphasize differences. Objective behavioral assessments that are not dependent upon parent report would help to bypass this potential influence. Second, measures of 24-hour dietary intake and physical activity were not assessed. Third, we did not measure child affect or temperament, which would have been informative as food neophobia is associated with anxiety⁴ and negative emotionality⁷. Fourth, the sample size was too small to conduct sex- or ethnicity-specific biometric analyses. Fifth, the novel finding that child food neophobia moderates the association between parent and child weight status needs to be replicated with a larger sample and wider distribution of CFNS scores. The median split used in the present study to probe the two-way interaction in the regression model may not necessarily generalize to other samples with different CFNS distributions.

In sum, we found that young children differ in food neophobia tendencies largely because of genetic factors, which accounted for 72% of the variance. Similar heritability estimates were reported previously in older children and adults. Greater food neophobia was associated with reduced eating compliance with parental prompts, which may also reflect children's resistance to parents' effort to coax eating. Finally, food neophobia moderated the association between parent-child weight status, such that heavier parents did not necessarily tend to have heavier children for those youth who were not avoidant of foods. Strategies to reduce food neophobia warrant greater investigation, with potential secondary benefits for family relations and child energy balance.

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Results of biometric analyses testing genetic and environmental influences on children's food neophobia tendencies.

Table 1

Model	χ^2	df	p	AIC	a ²	d ²	c ²	e ²
A-C-E	7.758	3	0.05	1.758	69%	--	0%	31%
A-D-E	3.869	3	0.28	-2.131	0%	72%	--	28%
A-E	7.758	4	0.10	-0.240	69%	--	--	31%
C-E	18.33	4	0.001	10.334	--	--	65%	35%
E	26.76	5	<0.001	16.76	--	--	--	100%

Notes: A= Additive genetic influences; D= dominance (non-additive) genetic influences; C= Common (shared) environmental influences; E= Error + Non-shared environmental influences. AIC= Akaike Information Criterion.

Table 2

Pearson's correlations between child food neophobia and measures of parental feeding strategies, parental demand cognitions regarding child eating, and division of responsibility (DoR) feeding practices.

Food Neophobia Scale association with:	Full Sample	Girls Only	Boys Only
Restriction of Child Feeding ^a	0.10 [132]	0.11 [70]	0.07 [62]
Encouragement to Eat ^a	-0.04 [132]	-0.11 [70]	0.07 [62]
Monitoring Fat Intake ^a	-0.00 [132]	0.01 [70]	-0.02 [62]
Anger and Frustration ^b	-0.03 [132]	-0.12 [70]	0.07 [62]
Food Amount Demandingness ^b	-0.10 [132]	-0.20 [70]	0.01 [62]
Food Type Demandingness ^b	-0.15 [132]	-0.29 [*] [70]	0.04 [62]
Mother-allotted child food choice ^c	0.14 [108]	0.12 [56]	0.14 [52]
Child Eating Compliance of Prompted Foods ^c	-0.36 ^{**} [106]	-0.44 ^{**} [54]	-0.36 ^{**} [52]
Child Eating Compliance of Initially Refused Foods ^c	-0.48 ^{**} [108]	-0.60 ^{**} [56]	-0.36 ^{**} [52]

^aFrom the Child Feeding Questionnaire³²;

^bFrom the Feeding Demands Questionnaire³⁵;

^cFrom the National Longitudinal Survey of Youth³⁷.

* p < 0.05,

** p < 0.001

Note: The number in the parenthesis is the sample size for the given correlation coefficient. The full sample is less than N=132 for certain association due to missing data (i.e., questionnaires that were not completed by the parent). This is also the reason why the N's were less than 70 and 62 in certain analyses for girls and boys, respectively.

Table 3

Results of multiple regression models testing the independent and interactive associations of maternal body mass index and child food neophobia on child body fat indexes

Predictor Variables in Model	Child Body Fat Indexes:		
	BMIz	%fat	Waist Circumference
Maternal Body Mass Index (BMI)	$\beta=-0.47$ $t=-1.74$ $p=0.08$	$\beta=-0.43$ $t=-1.25$ $p=0.22$	$\beta=-0.13$ $t=-0.45$ $p=0.66$
Child Food Neophobia	$\beta=-0.77$ $t=-2.09$ $p=0.04$	$\beta=-0.66$ $t=-1.21$ $p=0.23$	$\beta=-0.50$ $t=-1.31$ $p=0.20$
Interaction Term	$\beta=1.03$ $t=2.35$ $p=0.02$	$\beta=0.88$ $t=1.34$ $p=0.18$	$\beta=0.66$ $t=1.35$ $p=0.18$
Overall Model Fit	$F=2.67$ $p=0.05$ $R^2=0.06$	$F=0.69$ $p=0.56$ $R^2=0.02$	$F=2.66$ $p=0.05$ $R^2=0.08$

Note. Beta (β) refers to the standardized association between the respective predictor variable and the child body fat index used in the particular regression model.