Child Food Neophobia Is Heritable, Associated with Less Compliant Eating, and Moderates Familial Resemblance for BMI

Myles S. Faith1, Moonseong Heo2, Kathleen L. Keller3 and Angelo Pietrobelli4,5

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

Design and Methods: 66 same-sex twin pairs, including 37 monozygotic (MZ) and 29 dizygotic (DZ) pairs were studied. 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.

Conclusion: 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.

Obesity (2013) 21, 1650-1655. doi:10.1002/oby.20369

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 preferences (6), greater anxiety (4), greater emotionality (7), and more negative reaction to food (7). 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 study (8).

Family factors appear critical to the development of food neophobia. Parents shape the home food environment and child food preferences (9), and the same may be true of food neophobia. Indeed, parents who consumed a more varied diet had children who were less food neophobic (10). Children’s apprehension about trying new foods was reduced by increasing the availability of these foods at home and by parents tasting these items themselves (11). 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 5-11 years old, Pliner and Lowen (7) reported significant mother-child correlations for maternally reported food neophobia ($r = 0.23, P < 0.01$). Galloway and Birch (4) reported significant correlations among mothers and their 7-year-old daughters ($r = 0.20, P < 0.01$), whereas Falciglia et al. (10) reported significant parent-child correlations for food neophobia in 9-11-year-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 years (12).

Although suggestive of home environmental influences, family correlations also may reflect genetic factors. Genes influence behavioral traits in childhood, including eating in the absence of hunger (13) and 24-hour dietary intake patterns (14). The same may be true of food neophobia in early childhood. Cooke et al. (16) estimated genetic and environmental influences on food neophobia in 8-11-year-old twins enrolled from the United Kingdom (16,17). Results indicated that 78% of the variance in parent-reported food neophobia was because

1 Department of Nutritional Sciences and Food Science, Gillings School of Global Public Health, University of North Carolina, Chapel Hill, NC, USA. Correspondence: Myles S. Faith (mfaith@unc.edu) 2 Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY 10461, USA 3 Department of Health and Nutritional Sciences and Food Science, Pennsylvania State University, University Park, PA 16802, USA 4 Department of Pediatrics and Nutrition, University of Verona, Verona Italy 5 Pennington Biomedical Research Center, Baton Rouge, LA, USA

Funding agencies: Supported by NIH grant K08MH01530 to Dr. Faith.
Disclosure: The author declare no conflict of interest.
Received: 15 December 2013 Accepted: 3 January 2013 Published online 20 March 2013. doi:10.1002/oby.20369
of additive genetic factors, with the remaining variance attributable to nonshared (i.e., “unique” or “random”) environmental factors. Similar estimates were reported in a twin study of Finnish adults (18), which found that 69% of the variance in reported food neophobia was because of 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 specific (19), 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, because of 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. (21) did not find a significant association between food neophobia and BMI (kg/m²) z-score in 3-6-year-old children. 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 4-7-year-old children. On the basis of 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 (aim 2), but did not make predictions regarding potential associations with child body fat (aim 3).

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 elsewhere (22).

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 $z = 0.88$ 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. Parental feeding styles were measured by the Child Feeding Questionnaire (CFQ) (26). 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; and 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. This eight-item questionnaire measures the extent to which parents have demanding beliefs with respect to feeding relations with their children (29). The Feeding Demand Questionnaire (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 Demand- ingness, the extent to which parents believe that their children should eat a specific amount of food; and Food Type Demand- ingness, the extent to which parents believe that their children should eat specific types of foods. Cronbach’s $z$-coefficients for the subscales ranged from 0.70 to 0.86, and the instrument was written at the 4.8th grade level (29).

Division of responsibility feeding questions. Three division of responsibility (DoR) feeding questions from the National Longitudinal Survey of Youth (30) 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 sample (30).

Anthropometry and body composition. Children’s weight and height were measured by digital scale and stadiometer, respectively, and converted to BMI. BMI values in turn were converted to BMI z-scores and percentiles according to appropriate age- and sex-specific cut-offs (31). 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.

Sociodemographic measures. Child sex was dummy coded for final analyses. Child race/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.

Original Article

PEDIATRIC OBESITY

www.obesityjournal.org

Obesity | VOLUME 21 | NUMBER 8 | AUGUST 2013

1651
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 seven twin pairs, for whom parent-report questions determined child zygosity (32).

Procedures. Details of the research protocol are provided elsewhere (27). In brief, families came to the New York Obesity Research Center, St. Luke’s-Roosevelt Hospital, for four assessments over 2 weeks (27). The first two visits were primarily dedicated to conducting an “energy compensation” protocol (28), 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 approximately 11:30 AM until approximately 1:00 PM. 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 ± 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 analyses (33) formally estimated the magnitude of genetic, shared environmental, and nonshared 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 (e.g., 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 nonshared 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 sometimes referred to as “narrow-sense heritability.” In contrast, model 2 allows for and “fits” potential nonadditive 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 Visscher (40). The $\chi^2$ statistic and Akaiake Information Criterion (AIC) were used to evaluate the goodness-of-fit of the competing models. A nonsignificant $\chi^2$ 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 $\chi^2$. 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 data. 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 versus 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 $z = 0.05$. For any significant findings for our second and third study aims, we reran 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 race 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, that is, BMI-for-age ≥85%. Food neophobia did not differ between boys and girls (Ms = 41.54 vs. 38.89, $P = 0.29$), Caucasian and non-Caucasian children (Ms = 38.40 vs. 42.10, $P = 0.14$), and MZ and DZ twins (Ms = 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 (72). The $\chi^2$ statistic and Akaiake Information Criterion (AIC) were used to evaluate the goodness-of-fit of the competing models. A nonsignificant $\chi^2$ 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 $\chi^2$. 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 data. 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).
of dominant genetic influences, with the remaining variance estimated to be because of nonshared 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 among both 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 (\(r > 0.05\)) (Table 2) (We note that when using a Bonferroni-adjusted \(\alpha = 0.05\), 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 \((r = -0.003, P = 0.78)\), 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 \((r = 0.03)\) (Table 3). This association did not remain significant at the adjusted alpha level.)

### Table 1 Results of biometric analyses testing genetic and environmental influences on children’s food neophobia tendencies

| Model | \(\chi^2\) | \(df\) | \(P\) | 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%   |

A, additive genetic influences; D, dominance (nonadditive) genetic influences; C, common (shared) environmental influences; E, error + nonshared 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\(^c\)           | -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] |

Notes:
\(^a\)From the Child Feeding Questionnaire (26)
\(^b\)From the Feeding Demands Questionnaire (29)
\(^c\)From the National Longitudinal Survey of Youth (30).

\(^*P < 0.05,
\(^{*}\)\(P < 0.001\). 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 because of 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.
Child Food Neophobia is Heritable

Regarding environmental influences food neophobia, we found that a range of eating patterns that emerge during childhood. Thus, genes appear to influence response to external food cues and internal satiety signals (35), as children's propensity to eat in the absence of hunger (13) and eat in constant across the developmental spectrum. Genes also influence in model

| Predictor variables in model | BMI z-value | %fat | Waist circumference |
|-----------------------------|------------|------|---------------------|
| Maternal body mass index (BMI) | $\beta = -0.47$ | $\beta = -0.43$ | $\beta = -0.13$ |
| $t = -1.74$ | $t = -1.25$ | $t = -0.45$ |
| $P = 0.08$ | $P = 0.22$ | $P = 0.66$ |
| Child food neophobia | $\beta = -0.77$ | $\beta = -0.66$ | $\beta = -0.50$ |
| $t = -2.09$ | $t = -1.21$ | $t = -1.31$ |
| $P = 0.04$ | $P = 0.23$ | $P = 0.20$ |
| Interaction term | $\beta = 1.03$ | $\beta = 0.88$ | $\beta = 0.66$ |
| $t = 2.35$ | $t = 1.34$ | $t = 1.35$ |
| $P = 0.02$ | $P = 0.18$ | $P = 0.18$ |
| Overall model fit | $F = 2.67$ | $F = 0.69$ | $F = 2.66$ |
| $R^2 = 0.05$ | $R^2 = 0.06$ | $R^2 = 0.02$ | $R^2 = 0.08$ |

$\beta$ refers to the standardized association between the respective predictor variable and the child body fat index used in the particular regression model. $\beta = 0.08, P = 0.55$). 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 8-11-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 hunger (13) and eat in response to external food cues and internal satiety signals (35), as well as 24-hour dietary patterns (14). 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 development (34). Child-specific environments can include parental feeding practices or food exposures, which may differ among siblings (36), as well as social factors out of the home. Peers have a powerful influence on child development, including eating behavior (37). 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 obesity (9). 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 correlation (38) 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 contrary, a more varied diet compared to a less varied one is associated with overeating in controlled studies (39). 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 neophobia (23). 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 versus 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 effect of child or temperament, which would have been informative as food neophobia is associated with anxiety (4) and negative emotionality (7). 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 this 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’ efforts to coax eating. Finally, food neophobia moderated the association between parent and 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.

Acknowledgments
We thank the families enrolled in “Project Grow-2-gether” for their generous participation in this study.

© 2013 The Obesity Society

References
1. Pliner P. Development of measures of food neophobia in children. Appetite 1994; 23:147-163.
2. Pliner P, Hobden K. Development of a scale to measure the trait of food neophobia in humans. Appetite 1992;19:105-120.
3. Cooke L, Wardle J, Gibson EL. Relationship between parental report of food neophobia and everyday food consumption in 2-6-year-old children. Appetite 2003;41: 205-206.
4. Galloway AT, Lee Y, Birch LL. Predictors and consequences of food neophobia and pickiness in young girls. J Am Diet Assoc 2003;103:692-698.
5. Falcioglia GA, Couch SC, Gribble LS, Pabst SM, Frank R. Food neophobia in childhood affects dietary variety. J Am Diet Assoc 2000;100:1474-1481.
6. Russell CG, Worsey A. A population-based study of preschoolers’ food neophobia and its associations with food preferences. J Nutr Educ Behav 2008;40:11-19.
7. Pliner P, Loewen ER. Temperament and food neophobia in children and their mothers. Appetite 1997;28:239-254.
8. Wright CM, Parkinson KN, Shipton D, Drewett RF. How do toddler eating problems relate to their eating behavior, food preferences, and growth? Pediatrics 2007;120:e1069-e1075.
9. Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. Pediatrics 1998;101:539-549.
10. Falcioglia GA, Pabst S, Couch S, Goody C. Impact of parental food choices on child food neophobia. Children’s Health Care 2006;33:217-225.
11. Birch LL, McPhee L, Shoba BC, Pirok E, Steinberg L. What kind of exposure reduces children’s food neophobia? Looking vs. tasting. Appetite 1987;9:171-178.
12. Hursti Uk K, Spoden P. Food and general neophobia and their relationship with self-reported food choice: familial resemblance in Swedish families with children of ages 7-17 years. Appetite 1997;29:89-103.
13. Fisher JO, Cai G, Jaramillo SJ, Cole SA, Comuzzie AG, Butte NF. Heteritability of Hyperphagic eating behavior and appetite-related hormones among Hispanic children. Obesity (Silver Spring) 2007;15:1484-1495.
14. Faith MS, Rhea SA, Corley RP, Hewitt JK. Genetic and shared environmental influences on children’s 24-h food and beverage intake: sex differences at age 7 y. Am J Clin Nutr 2008;87:903-911.
15. Cooke LJ, Haworth CM, Wardle J. Genetic and environmental influences on children’s food neophobia. Am J Clin Nutr 2007;86:428-433.
16. Oliver BR, Plomin R. Twins’ Early Development Study (TEDS): a multivariate, longitudinal genetic investigation of language, cognition and behavior problems from childhood through adolescence. Twin Res Hum Genet 2007;10:96-105.
17. Trouton A, Spinath FM, Plomin R. Twins early development study (TEDS): a multivariate, longitudinal genetic investigation of language, cognition and behavior problems in childhood. Twin Res 2002;5:444-448.
18. Knaapila A, Tuuttil H, Silventoinen K, et al. Food neophobia shows heritable variation in humans. Physiol Behav 2007;91:573-578.
19. Klump KL, McGue M, Iacono WG. Age differences in genetic and environmental influences on eating attitudes and behaviors in preadolescent and adolescent female twins. J Abnorm Psychol 2000;109:239-251.
20. Silventoinen K, Roholm B, Kaprio J, Sorensen TI. The genetic and environmental influences on childhood obesity: a systematic review of twin and adoption studies. Int J Obesity 2010;34:29-40.
21. Lameng JC, Cardinal TM, Sitto JR, Kannan S. Ability to taste 6-n-propylthiouracil and BMI in low-income preschool-aged children. Obesity (Silver Spring) 2008;16: 1522-1528.
22. Faith MS, Pietrobelli A, Heo M, et al. A twin study of self-regulatory eating in early childhood: Estimates of genetic and environmental influence, and measurement considerations. Int J Obes (Lond) 2012;36:93-97.
23. Cooke L, Carnell S, Wardle J. Food neophobia and mealtime food consumption in 4·5 year old children. Int J Behav Nutr Phys Act 2006;3:14.
24. Pliner P, Lahteenmaki L, Tuorila H. Correlates of human food neophobia. Appetite 1998;30:93.
25. Pliner P, Pelchat M, Grabski M. Reduction of neophobia in humans by exposure to novel foods. Appetite 1993;20:111-123.
26. Birch LL, Fisher JO, Grimm-Thomas K, Markey CN, Sawyer R, Johnson SL. Confirmatory factor analysis of the Child Feeding Questionnaire: a measure of parental attitudes, beliefs and practices about child feeding and obesity proneness. Appetite 2001;36:201-210.
27. Faith MS, Keller KL, Matz PE, Lewis R, Ridle C, Han H, Heo M, Pietrobelli A, Heymsfield SB, Allison DB. “Project Grow-2-gether”: A twin study of the genetic and environmental influences on children eating and obesity. Twin Research 2002;5: 472-275.
28. Faith MS, Keller KL, Johnson SL, Pietrobelli A, Matz PE, Must S, Jorge MA, Cooperberg J, Heymsfield SB, Allison DB. Familial aggregation of energy intake in children. American Journal of Clinical Nutrition 2004;79:844-850.
29. Faith MS, Storey M, Kral TV, Pietrobelli A. The feeding demands questionnaire: assessment of parental demand cognitions concerning parent-child feeding relations. J Am Diet Assoc 2008;108:624-630.
30. Faith MS, Heshka S, Keller KL, et al. Maternal-child feeding patterns and child body weight: findings from a population-based sample. Arch Pediatr Adolesc Med 2003;157:926-932.
31. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. Vital Health Stat 11 2002;1:190.
32. Rietveld MJ, van Der Valk JC, Bongers IL, Stroot TM, Slagboom PE, Boomsma DI. Zygosity diagnosis in young twins by parental report. Twin Res 2000;3:134-141.
33. Neale MC, Cardon LR. Methodology for Genetic Studies of Twins and Families. Dordrecht: Kluwer; 1992.
34. Rowe DC. The Limits of Family Influence. Genes, Experience, and Behavior. New York: The Guilford Press; 1994.
35. Carnell S, Haworth CM, Plomin R, Wardle J. Genetic influence on appetite in children. Int J Obes (Lond) 2006;32:1408-1473.
36. Keller KL, Pietrobelli A, Johnson SL, Faith MS. Maternal restriction of children’s eating and encouragements to eat as the ‘non-shared environment’: a pilot study using the child feeding questionnaire. Int J Obes (Lond) 2006;30:1670-1675.
37. Salvy SJ, Kieffer E, Epstein LH. Effects of social context on overweight and normal-weight children’s food selection. Eat Behav 2008;9:190-196.
38. Agran WS, Hammer LD, McNicholas F, Kramer HC. Risk factors for childhood overweight: a prospective study from birth to 9.5 years. J Pediatr 2004;145:20-25.
39. Epstein LH, Robinson JL, Temple JL, Roemmich JN, Marusewski AL, Nadbrzuch RL. Variety influences habituation of motivated behavior for food and energy intake in children. Am J Clin Nutr 2009;89:746-754.
40. Wray N, Visscher P. Estimating trait heritability. Nat Educ 2008;1(1).