Genetic and environmental influences on children's food neophobia[1–3]

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ABSTRACT

Background: Food neophobia in children has been associated with a low intake of fruit, vegetables, and protein foods. The design of effective interventions to improve children's diets would be facilitated by a better understanding of the determinants of neophobia.

Objective: Our objective was to quantify the contribution of genetic and environmental differences to variation in child food neophobia.

Design: Parents of twins aged 8–11 y ($n = 5390$ pairs) completed questionnaires about their children's eating habits, including a measure of food neophobia.

Results: The results showed that neophobia is highly heritable. The heritability estimate from model fitting was 0.78 (95% CI: 0.76, 0.79). A further 22% of the variance was explained by nonshared environmental factors, with no influence of shared environmental factors.

Conclusions: Neophobia appears to be a heritable trait, but almost a quarter of the phenotypic variation is accounted for by nonshared environmental factors. An important aim for future research is the identification of influential aspects of the environment specific to individual children. *Am J Clin Nutr* 2007;86:428–33.

KEY WORDS Heritability, genetics, twins, neophobia

INTRODUCTION

Humans typically show some degree of avoidance to new foods, a trait that has been termed *food neophobia* (1). Food neophobia is widespread in omnivores and has been observed in warblers (2), rats (3), chimpanzees (4), and capuchin monkeys (5), among other species. Neophobia may have adaptive value in reducing the possibility of poisoning from unfamiliar and toxic foods, although it will also have costs in terms of limiting dietary variety.

In the modern environment where foods are generally safe to eat, neophobia appears principally to have an adverse effect on food choices, particularly on intake of fruit and vegetables (6, 7). Children scoring above the median on the Child Food Neophobia Scale (CFNS) (8) ate 35% less fruit and vegetables at a meal than did children scoring below the median, while consuming just as many sweet, fatty, and starchy foods (7). Given the importance of fruit and vegetable consumption to health in childhood and adulthood (9–11), a better understanding of the cause of individual differences in neophobia would be valuable.

A number of studies have examined family similarity for neophobia, typically finding low- to moderate-sized correlations ($r = 0.2–0.3$) between parents and children or between siblings (8, 12–16). Heritability has not been estimated, but correlations of this size would be consistent with moderate-strong heritability, given that parents and children or siblings share half of their genes on average, although it could equally be due to shared home environments.

Twin studies provide a unique opportunity to estimate the relative contribution of genes and environment to phenotypic differences, because monozygotic (MZ) twins share all their genes, whereas dizygotic (DZ) twins on average share half their genes, and both MZ and DZ pairs share a home environment in childhood. Comparing intraclass correlations between MZ pairs and DZ pairs for any phenotype gives an estimate of the contribution of genes, shared environmental factors (aspects of the environment that make family members similar), and nonshared environmental factors (aspects of the environment that make family members different) to phenotypic variation. The terms *shared* and *nonshared environments* categorize the environment on the basis of its effect, not on whether it appears to be common to both twins. Living in the same home with the same parents can make identical twins (or other siblings) more alike than would be expected by their shared genes. This does not happen for all traits, but when it does it is called a shared-environment effect. When twins (or siblings) are more different from what would be expected, this is described as a nonshared environmental effect. This will be addressed in more detail in the discussion.

Only one study has examined the heritability of reactions to food with the use of a twin design. With the use of the five-item "reaction to food" subscale of the Colorado Childhood Temperament Inventory, Plomin and Rowe (17) reported no differences in the correlations between scores for MZ and DZ twin pairs and therefore no evidence for heritability. However, the average age

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[2] Supported by grant 31/D19086 from the Biotechnology and Biological Sciences Research Council to examine obesity in the Twins Early Development Study (TEDS). The TEDS project is supported by a programme grant (G500079) from the UK Medical Research Council. JW and LC are funded by Cancer-Research-UK.

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Received December 6, 2006.

Accepted for publication April 5, 2007.

of the children was 3 y, the sample size was comparatively small (91 twin pairs), and the measure of food reactions will not have fully captured all the features of food neophobia because it assessed responses to foods in general, not to unfamiliar foods. The present study used a shortened version of the CFNS (8) in a large sample of MZ and DZ twins to examine the relative contribution of genes and environment to variation in children's food neophobia.

SUBJECTS AND METHODS

Participants

The sample for this study came from the Twins Early Development Study (TEDS), a study of twins born in 1994, 1995, and 1996 (18). TEDS was shown to be reasonably representative of families in the United Kingdom with children in terms of parental education and occupation (19, 20). Zygosity was established when the twins were aged 18 mo with the use of an extensive measure of physical similarity which was found to be >95% accurate compared with DNA testing. Agreement of 96% was achieved when the questionnaire was readministered 18 mo later (21). Where data were missing or unclear, DNA testing was conducted, but it was not feasible to perform DNA analyses on the full sample of >15 000 twin pairs. Ethical approval for the study was granted by the UCL Committee for the Ethics of Non-NHS Human Research.

Procedure

Families from the TEDS cohort were invited to take part in an eating behavior survey. Questionnaires were mailed to parents who completed them at home and returned them with the use of Freepost envelopes. A separate questionnaire was completed for each twin. If the questionnaires were not returned after 2 mo, families received ≤2 reminders.

Measures

The trait of neophobia was assessed with a four-item version of the CFNS (8), which included the following items: "My child is constantly sampling new and different foods" (reversed), "My child doesn't trust new foods," "My child is afraid to eat things s/he has never had before." and "If my child doesn't know what's in a food s/he won't try it." These questions were selected because they appeared likely to best capture responses to *new* foods. Responses were on a 4-point scale from "strongly disagree" to "strongly agree"; with higher total scores indicating higher neophobia. The original 10-item version of this scale has good reliability and validity, but the Cronbach's α for the shortened version was also high (0.88).

Statistical analyses

The neophobia composite score was calculated as a mean of the 4 items. Scores were then standardized to z scores that have a mean of 0 and a SD of 1. Because twin covariances can be inflated by variance because of age or sex (the age and sex of twins is perfectly correlated), all scores were residualized for age and sex effects with the use of a regression procedure (22).

Main effects of sex and zygosity and any potential interaction effects were examined with the use of a 2 × 2 (sex-by-zygosity interaction) analysis of variance. Intraclass correlations for neophobia were compared between MZ and DZ twin pairs. If a trait is influenced by genetics, within-pair resemblance for that trait should be higher in identical twins than in nonidentical twins.

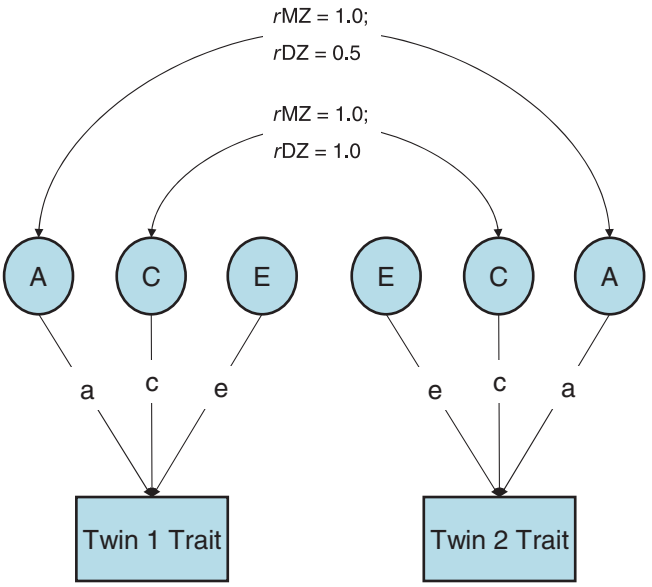

FIGURE 1. Path diagram of a simple genetic model. A, additive genetic influence; C, shared environment influence; E, nonshared environment influence. The path coefficients a, c, and e indicate effects of A, C, and E on a trait; *r*MZ, monozygotic correlation; *r*DZ, dizygotic correlation.

The proportion of the variance for a particular trait that is attributable to additive genetic influences and shared and nonshared environmental influences was estimated from twin analyses. Heritability describes the proportion of phenotypic variance that can be ascribed to heritable genetic influences (ie, it is an index of the genetic effect size) and can be estimated as twice the MZ-DZ correlation difference (23). The greater the difference between correlations for MZ and DZ twins, the higher the heritability. The shared environment component can be estimated by subtracting the estimate of heritability from the MZ correlation because that indicates how much more alike a shared family environment has made the twins than would be predicted by their being genetically identical. The remaining variance provides an estimate of the nonshared environment component (ie, aspects of the environment that make twins different from one another) plus error of measurement.

Model-fitting analyses were used to estimate parameters, provide CIs for the parameter estimates, and test the fit of alternative models (24). MX software for structural equation modeling was used to perform standard model-fitting analyses with the use of raw data which takes all of the data into account rather than using summary statistics such as correlations or variance or covariance matrices (25). Two fit indexes are reported: chi-square and Akaike's information criterion (26). The best-fitting model was chosen on the basis of a change in the chi-square test not representing a significant worsening of fit; for a change of df of 1, the statistically significant change in the chi-square test is 3.84. **Figure 1** shows the basic twin model.

The path coefficients of latent variables A (additive genetic), C (common or shared environmental), and E (nonshared environmental, including error of measurement) are represented by the lower case letters a, c, and e, respectively. The coefficient of genetic relatedness (r_G) is 1.0 for MZ twins and 0.5 for DZ twins.

Environmental relatedness, the shared environmental correlation (r_C), is assumed to be 1.0 both for MZ and DZ twins. The full ACE model dissects the phenotypic variance into these 3 components of variance. Given that a general scientific principle is parsimony (to prefer a simpler theory if it accounts equally well for the given observations), 2 subsequent nested models, the CE and AE models, were also tested to determine whether these 2 parameters adequately explained the data [for details see (24)]. A saturated phenotypic model was also fitted to the data. This model perfectly describes the data and does not partition the variance into ACE components. Therefore, the comparison of the ACE models with the saturated model provides information about the overall goodness-of-fit of these models (ie, whether they adequately explain the data).

RESULTS

Response rate and participant characteristics

Of the 12 212 families to whom the eating behavior questionnaire was sent, 8978 were active participants in TEDS at the time of data collection. Of these, 5543 (61.7%) returned completed questionnaires. Of the remaining 3234 families who were not currently active, a further 359 returned completed questionnaires, resulting in a total sample of 5902 families (representing 48.3% of the entire original sample). Responders were significantly higher than nonresponders on a composite measure of socioeconomic status based on occupational status and educational qualifications of mothers and fathers [$F(1,14556) = 155.74$, $P < 0.001$]. However, the effect size was small (0.2) and was statistically significant only because of the large sample size. We excluded from the analyses families in which either twin had a specific medical condition or was an extreme outlier for perinatal problems (eg, ≥3 SDs below mean birth weight, <471 g). Those scoring >3 SDs above or below the mean on the CFNS were also excluded. After exclusions, the final sample comprised 5390 complete pairs of twins: 1913 MZ ($n = 894$ male, $n = 1019$ female) and 3477 DZ pairs ($n = 878$ male, $n = 906$ female, $n = 1693$ opposite sex). The mean age of the twins was 9.91 y (range: 8.32–11.61 y).

Respondents were mostly of white ethnic origin (92%), with 6% other and 2% unknown. Mothers were mostly working (60%), but 21% were full-time homemakers, 3% were not working, and the remaining 15% declined to answer. Likewise 72% of fathers were employed, 3% were full-time homemakers, 2% were not working and 22% declined to answer. University degrees were held by 21% of mothers and 22% fathers, "A" levels (equivalent to US 12th grade) by 15% of mothers and 9% of fathers, and "O" levels (equivalent to US 10th grade) by 36% of fathers and 41% of mothers.

Effects of sex and zygosity on CFNS scores

Means and SDs of standardized neophobia scores and the results of a 2 × 2 analysis of variance are presented in **Table 1**. There were significant effects of both zygosity ($P = 0.003$, $\eta^2 = 0.001$) and sex ($P < 0.001$, $\eta^2 = 0.001$); MZ and female twins had lower scores than did DZ or male twins. However, the difference is small indeed, and the effect size shows that this difference explains only 0.1% of the variance. These findings do not have an effect on the individual difference analysis. No significant interaction was observed between sex and zygosity.

TABLE 1
Neophobia scores, by zygosity and sex, and ANOVA results showing significance and effect size[1]

	Neophobia score
MZ + DZ	
All ($n = 10\,780$)	2.34 ± 0.44
Male ($n = 5237$)	2.36 ± 0.45
Female ($n = 5543$)	2.32 ± 0.43
MZ	
All ($n = 3826$)	2.32 ± 0.43
Male ($n = 1788$)	2.34 ± 0.43
Female ($n = 2038$)	2.31 ± 0.43
DZ	
All ($n = 6954$)	2.35 ± 0.45
Male ($n = 3449$)	2.37 ± 0.47
Female ($n = 3505$)	2.33 ± 0.43

[1] All values are $\bar{x}$ ± SD. MZ, monozygotic; DZ, dizygotic. ANOVA showed significant effects of zygosity ($P = 0.003$, $\eta^2 = 0.001$) and sex ($P < 0.001$, $\eta^2 = 0.001$) but no significant interaction between sex and zygosity ($P = 0.522$, $\eta^2 < 0.001$).

Genetic analyses of individual differences

Intraclass twin correlations are shown in **Table 2**. They are presented for the total group of MZ, DZ same-sex, and DZ opposite-sex twins and for the males and females among same-sex pairs. In every case, MZ correlations greatly exceeded the DZ correlations, strongly implicating genetic influence. Correlations between male and female pairs were similar, and correlations for opposite-sex DZ twins (0.35) were similar to those for same-sex DZ twins (0.38).

The results of model-fitting analyses are shown in **Table 3**. The parameter estimates from the model fitting are highly similar to the estimates expected on the basis of the intraclass correlations (Table 2). Estimates show high heritability (0.78) for neophobia. Furthermore, in line with our correlations, the best-fitting and most parsimonious model is the AE model, which constrains the shared environmental parameter to be zero. The AE model was selected based on the fit statistics presented in Table 3. An analysis of quantitative and qualitative sex differences showed no significant differences in cause for males and females (data not shown).

TABLE 2
Intraclass correlations for Child Food Neophobia Scale (CFNS) data for twin pairs by zygosity and sex

	n[1]	r[2]
Monozygotic (all)	1913	0.77
Monozygotic (male)	894	0.77
Monozygotic (female)	1019	0.76
Dizygotic (all)	3477	0.36
Dizygotic (same sex)	1784	0.38
Dizygotic (opposite sex)	1693	0.35
Dizygotic (male)	878	0.35
Dizygotic (female)	906	0.41

[1] Number of complete pairs.
[2] All correlations were significant at $P < 0.001$.

TABLE 3
Individual differences model fitting for neophobia for the entire sample: model fit and parameter estimates[1]

Model	−21nL	df	Tested against	*P*	AIC	A	C	E
1. Saturated model	27 996.568	10808	—	—	—	—	—	—
2. ACE	27 998.084	10809	1	0.218	−0.484	0.78 (0.75, 0.79)	0.00 (0.00, 0.02)	0.22 (0.21, 0.24)
3. CE	28 639.279	10810	1	< 0.001	638.711			
			2	< 0.001	639.200		0.50 (0.48, 0.52)	0.50 (0.48, 0.52)
4. AE	27 998.084	10810	1	0.469	−2.484			
			2	—[2]	−2.000	0.78 (0.76, 0.79)		0.22 (0.21, 0.24)

[1] Two fit indexes are reported from the structural equation modeling analyses: *P* value based on the likelihood ratio chi-square test and Akaike's information criterion (AIC; Akaike, 1987). −21nL, −2 log likelihood; df, degrees of freedom; A, additive genetic influence; C, shared environmental influence; E, nonshared environmental influence. There were 10 816 observed statistics from 5390 complete pairs and 36 incomplete pairs. CE and AE models are nested within the ACE model. The best-fitting model, the AE model, was chosen on the basis of a change in χ not representing a significant worsening of fit (for a change in df of 1, the statistically significant change in χ^2 is 3.84). Models 2, 3, and 4 were compared with the saturated phenotypic model (model 1), which perfectly fits the data. This comparison provides information about the overall goodness-of-fit of each model to the data. Results show that the ACE and AE models fit the data (they are not significantly worse in fit in comparison with the saturated model). The AE model is the most parsimonious model that adequately explains the data. 95% CI in parentheses.

[2] Probability was incalculable because of no change in fit statistics.

DISCUSSION

To our knowledge this was the first study to examine the relative influences of genetic and environmental factors on food neophobia measured with a standard scale. The results are consistent with published literature that has found parent-child and sibling-sibling correlations to be ≈0.3 (8, 12–16). Intraclass correlations between similarly related DZ pairs in this sample were slightly higher (0.36), but this is to be expected given that twins are exactly the same age as each other. Correlations between MZ twins greatly exceeded correlations between DZ twins, indicating a strong heritable component to variation in neophobia.

The model-fitting analyses produced broadly similar results. Variation in neophobia scores because of heritable genetic differences was estimated at 78%. A further 22% of variance was due to nonshared environment effects (including measurement error), with no observed influence of shared environmental factors. This is a robust finding. Genetic research has consistently shown that shared genes rather than shared experience largely accounts for similarities in behavioral traits between family members. The family environment was found to have surprisingly little influence on within-family similarity across a variety of personality traits, on psychopathology, or, surprisingly, on adult body weight (27). Our results are consistent with these previous findings and suggest that for neophobia, despite sharing home, parents, neighborhood, and culture, siblings must experience these environmental factors differently; therefore, the outcomes differ accordingly. The idea that apparently shared environments have nonshared effects has sometimes been misinterpreted as indicating that parenting is unimportant. More plausible, however, is the possibility that parents treat different children differently, perhaps responding sensitively to each child's needs [*see* a summary in (28)]. Alternatively different children experience the same situation differently, based on what they bring to the situation (29).

In attempting to explain why parents treat siblings differently, several possibilities are proposed. One is that there is a tendency for "niche-picking" among siblings, the adoption of distinctive roles to secure individual attention and other valuable resources [known as Deidentification Theory (30)], which induces differential treatment by parents. Parents themselves might promote differentiation between siblings by accentuating differences in temperaments and behavior. In all likelihood there is considerable reciprocal influence in parent-child interactions. By extrapolating these ideas to the issue of neophobia, it is easy to imagine that, if a child refuses food on several occasions, he or she may be labeled picky and treated differently from other children at mealtimes. The child-feeding techniques that parents typically use to coax an unwilling child to eat (eg, offering rewards or withdrawal of privileges) can make the situation worse rather than better (31, 32). Accidental environmental factors may also play a part in promoting differences between siblings. For example, if one twin experiences nausea or vomiting after ingestion of an unfamiliar food, this might result in a greater reluctance to try new foods in the future, even if the cause of the reaction was, in fact, unrelated to the food (ie, the result of illness). Such explanations are speculative, but they illustrate the need for research to identify the sources of nonshared environmental effects, both within and outside the home environment, and, to do this, we have to investigate environments in a child-specific, rather than a family general way (24).

Another focus for future research is the implication of these findings for basic taste acceptance. Recent work has used twin data to examine the heritability of food preferences, finding that heritability was modest for dessert foods (0.20), moderate for vegetables (0.37) and fruit (0.51), and high for protein foods (0.78) (33). It would be of value to gather the information on the general trait (neophobic) and the specific behaviors (food acceptance) in the same study and carry out cross-twin, cross-trait investigations. There has also been a good deal of interest in variation in 6-*n*-propylthiouracil (PROP) and phenylthiocarbamide taster status, which is a genetically determined trait (34, 35). In common with neophobic persons, PROP tasters appear to like vegetables less than nontasters (36–39). Heightened taste sensitivity could result in a reluctance to taste unfamiliar foods, although to date no one has measured PROP sensitivity and food neophobia in the same sample. One study (40) found PROP tasters to be no more likely to describe themselves as "unadventurous" about food than nontasters, but another study found that super-tasters were familiar with fewer foods than mere tasters or nontasters(41). This may suggest directions for linking behavior, biology, and genetics in the field of taste perception.

Like all twin research, the results of the present study must be interpreted with caution. Although the twin method has been described as the "perfect natural experiment" (42), it is not without its detractors [see Bouchard and McGue (43) for a summary of the most frequent criticisms]. For example, it has been claimed that the experience of growing up as a twin is so different from that of a singleton that findings based on twin samples cannot be generalized to the population as a whole. However, investigations of the personality of twins have shown few differences between MZ and DZ pairs or between twins and singletons (44), and in the present sample mean neophobia scores were similar to those of singletons of a similar age (item mean, 2.34 ± 0.44) in the present sample compared with. 2.26 ± 0.59 (n = 96: unpublished data, 2004).

Other critics have challenged the "equal environments assumption" which is critical to the twin method. However, support for the validity of the equal environments assumption has come from the domains of psychiatric disorders (45, 46), sexual orientation (47), mental ability, personality traits (48), and eating attitudes and behaviors (49). It is not possible to entirely rule out inflation of heritability estimates in the present study, but, even if the estimate were slightly lower, this would not change the conclusion that neophobia is a largely heritable trait in children.

Conclusions

The aim of the present study was to obtain an estimate of the contribution of genetic and environmental influences to food neophobia in children 8–11 y of age. The results showed high heritability, but they also showed that just under a quarter of the variance in neophobia was accounted for by nonshared environmental influences. Parents can be reassured that their child's reluctance to try new foods is not simply the result of poor parental feeding practices, but it is partly in the genes. However, notwithstanding high heritability, research in laboratory and real-world settings has shown that neophobia for specific foods can be reduced through exposure-based interventions. New foods can become familiar, and disliked foods become liked, with repeated presentation (50–54) (L Cooke and J Wardle, unpublished observation, 2006), although the process might be more laborious with a highly neophobic child. Guidance in effective feeding techniques and modification of other influential environmental factors may help to minimize the negative effects of neophobia on children's diets.

The author's responsibilities were as follows—LJC: devised the study and prepared the first draft of the manuscript; JW: contributed to the design of the study and to the writing of the manuscript; and CMAH: performed the statistical analyses and contributed to the writing of the manuscript. None of the authors had a conflict of interest.

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