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Food neophobia shows heritable variation in humans

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Abstract

Food neophobia refers to reluctance to eat unfamiliar foods. We determined the heritability of food neophobia in a family and a twin sample. The family sample consisted of 28 Finnish families (105 females, 50 males, aged 18–78 years, mean age 49 years) and the twin sample of 468 British female twin pairs (211 monozygous and 257 dizygous pairs, aged 17–82 years, mean age 55 years). Food neophobia was measured using the ten-item Food Neophobia Scale (FNS) questionnaire, and its internationally validated six-item modification. The heritability estimate for food neophobia was 69 and 66% in Finnish families (h^2) and 67 and 66% in British female twins (a^2+d^2) using the ten- and six-item versions of the FNS, respectively. The results from both populations suggest that about two thirds of variation in food neophobia is genetically determined.

Keywords: Food neophobia; Quantitative trait; Heritability; Genetic; Family; Twin

1. Introduction

Food neophobia is defined as the reluctance to eat unfamiliar foods. It is distinct from finickiness (picky eating) that refers to unwillingness to eat disliked familiar foods [1,2]. In humans, food neophobia is often measured by the Food Neophobia Scale (FNS), a validated ten-item questionnaire [1]. High scores of the FNS indicate a low anticipated liking of unfamiliar foods and low familiarity of foreign cuisines [1], as well as low willingness to try unfamiliar foods [3]. Differences in measured food neophobia did not relate to actual hedonic responses to unfamiliar foods, when tasted, in one study [1], while in other studies neophobic subjects tended to like unfamiliar foods less than neophilic subjects [4,5]. Earlier tasting of food has been demonstrated to greatly enhance the willingness to eat the food again regardless of the level of food neophobia [3]. This supports the view that food neophobia captures responses to unfamiliar, but not to familiar foods [1,5]. Clearly, food neophobia influences the initial tasting of unfamiliar food, but the continuation of consumption is determined by many other factors as well [6].

Evolutionarily, food neophobia may have given a selective advantage by protecting from harmful foods, as well as a disadvantage by narrowing the variety of diet of omnivorous animals, including humans. This may have led to a situation in which the environment determined whether neophobic or neophilic behavior was advantageous. Nowadays, food safety is generally guaranteed in the developed societies, and the protective function of food neophobia may not be advantageous any longer [7]. On the contrary, the utmost food neophobic individuals may restrict their diet to ones with inadequate nutritional quality, or at least lose the potential health and hedonic advantages of new foods.

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Individuals vary widely in food neophobia [3, for a review see 7]. The hypothetical evolutionary significance of food neophobia raises the question whether the variation in food neophobia, considered as a personality trait [1], has a genetic component, and, if the genetic component is involved, how large is its influence. In mice, evidence of genetic influence on food neophobia has been found, and an effective locus mapped to chromosome eight [8]. Hence, food neophobia may show heritable variation also in humans. However, heritability estimates for the trait food neophobia in humans have not been reported to date. Thus, our major aim was to estimate the heritability of food neophobia using the FNS and its modifications in Finnish families and British female twins.

Unidimensionality of the FNS has been questioned, as well as the validity of its individual items and cross-cultural comparability of the scores [3,9]. By exploratory factor analysis, Tuorila et al. [3] separated a factor for general concern about eating from the "real" food neophobia factor. Ritchey et al. [9] studied FNS data from North American, Swedish, and Finnish populations by confirmatory factor analysis, and proposed a shortened, six-item version of the FNS for the valid cross-cultural comparisons of food neophobia. Thus, the reliability of the FNS and its modifications and their differences in the heritability analyses were also explored in the present study.

2. Subjects and methods

2.1. Data collection

The family sample consisted of 155 Finnish adults from 28 families, including 70 parent–offspring, 145 sibling, and 47 first cousin pairs. The twin sample consisted of 936 British adult female twins, including 211 complete monozygous (MZ) and 257 dizygous (DZ) twin pairs (Table 1). The subjects were originally recruited for clinical studies unrelated to food orientations: the Finnish subjects were participants of the migraine family study [10] and the British subjects came from the studies of the UK Adult Twin Registry [11]. The subjects that participated in these clinical studies during certain periods were invited to the present study. These subjects were asked to fill out the questionnaire containing the items of FNS at home and to return it when they visited the clinic for the other studies. The subjects were motivated to original studies by offering feedback regarding their health, and they participated in the present study on a voluntary basis. The ethical principles applied in the studies were approved by the appropriate ethical committees in Finland and in the UK.

Table 1
Characteristics of the study samples

	Finnish families		British female twins	
	Males	Females	MZ	DZ
Number of individuals	50	105	422	514
Age (years)				
Range	19–74	18–78	18–82	17–80
Mean (S.D.)	46.3 (16.3)	49.6 (14.5)	54.0 (14.2)	56.4 (11.0)

MZ = monozygous, DZ = dizygous.
S.D. = standard deviation.

Table 2
The items of the Food Neophobia Scale (FNS) and the factor loadings of Varimax rotated factor matrix

Item [a]	Loadings [b]			
	Finnish families		British female twins	
	Factor 1	Factor 2	Factor 1	Factor 2
1. I am constantly sampling new and different foods (rev)	**0.624**	0.051	**0.622**	0.175
2. I don't trust new foods	**0.596**	0.080	0.322	**0.615**
3. If I don't know what is in food, I won't try it	**0.483**	0.384	0.165	**0.701**
4. I like foods from different countries (rev)	**0.727**	0.192	**0.794**	0.174
5. Ethnic food looks too weird to eat	**0.693**	0.199	0.436	**0.475**
6. At dinner parties, I will try a new food (rev)	**0.631**	0.009	**0.639**	0.302
7. I am afraid to eat things I have never had before	**0.624**	0.309	0.372	**0.576**
8. I am very particular about the foods I will eat	−0.071	**0.997**	0.125	**0.540**
9. I will eat almost anything (rev)	0.289	**0.548**	**0.438**	0.346
10. I like to try new ethnic restaurants (rev)	**0.740**	0.202	**0.726**	0.319

[a] Responses to items negative to food neophobia (rev) were reversed prior to analysis.
[b] Loadings higher on either factor are bolded.

Only females were included in the twin sample because for historical reasons [11], the UK Adult Twin Registry comprises mainly female twins making the female sample far easier to collect than large male and female samples of equal size, and because in that way potential interfering non-genetic sex effects were also eliminated from twin data. Zygosity of the twins was ascertained using the standard questionnaire and, where there was uncertainty, checked by genotyping [11]. The family sample included mainly individuals diagnosed with migraine (83%) as well as their healthy relatives (17%). The original Finnish migraine family study sample consisted of consecutively identified families with at least four migraine cases in each family. For further molecular genetic studies, the pedigrees with very homogenous migraine throughout the entire family were selected [10]. The family sample in the present study came from these typical migraine families.

Food neophobia data were collected using the FNS [1]. It was included in a large questionnaire consisting of other food orientation, food frequency, and food pleasantness scales as well. Items of the FNS are listed in Table 2. Each item was rated using seven categories (endpoints: disagree strongly, agree strongly). Relative to food neophobia, half of the items were positively and half negatively worded and responses given to negatively worded items were reversed before analyses. The score proposed by Ritchey et al. [9] was calculated with six items of the FNS (items 1, 3, 4, 6, 7, and 10), denoted FNS-R herein. The responses were summed (few missing responses replaced by the individual mean), resulting in the theoretical range of 10–70 for the FNS score and 6–42 for the FNS-R score.

Table 3
Mean FNS and FNS-R scores in the Finnish and the British samples

	Theoretical range	Finnish families							British female twins						
		All		Males		Females		Sig	All		MZ		DZ		Sig
		Mean	S.D.	Mean	S.D.	Mean	S.D.		Mean	S.D.	Mean	S.D.	Mean	S.D.	
FNS	10–70	30.9	10.8	30.1	11.3	31.3	10.6	ns	31.8	12.4	30.8	13.6	32.7	12.2	*
FNS-R	6–42	18.0	7.2	17.9	7.1	18.1	7.3	ns	18.8	8.0	18.0	8.0	19.4	7.9	**

MZ = monozygous, DZ = dizygous.
S.D. = standard deviation.
Sig = the significances of the differences of group means (males/females, MZ/DZ twins): ns, not significant; *, $p<0.05$; **, $p<0.01$.

2.2. Data analysis

Basic statistical analyses (Pearson correlations, *t*-test, and reliability and factor analyses) were carried out using the SPSS statistical package version 12.0. Factor analysis was performed using Maximum likelihood factoring together with orthogonal Varimax rotation. The number of factors extracted was constrained by excluding factors with eigenvalues less than one.

Heritability estimates were computed for the FNS and FNS-R scores, and for the factor scores calculated in the factor analysis (two factors). The heritability analyses of the family data were performed using the SOLAR package [12] assuming a polygenic model. The analyses were also done using sex and age as covariates, but this did not improve the model. Heritability (h^2) is the proportion of variance attributable to genetic factors. However, when estimating heritability using family data and not twins, incorporation of shared (common) environmental influence into the heritability estimate cannot be ruled out. Thus, the heritability estimate can be regarded only as tentative evidence for genetic influence on variance of the studied traits. A more reliable estimate of genetic influence can be achieved by analyzing twin data.

Suggestive implications about genetic and environmental effects can be made by comparing intra-class correlations of MZ and DZ twins. Because MZ twins have 100% and DZ twins, on average, 50% of their segregating genes in common, higher intra-class correlations for MZ compared with DZ pairs implies a genetic influence. In addition, the structural equation modeling of twin data allows the determination of the confidence intervals of parameter estimates, and the fit of the models can be explored statistically [13]. Two models were fitted into the data: a model including an additive genetic (A), a shared (common) environmental (C), and a non-shared (individual) environmental (E) components, as well as a model that contained a non-additive genetic component (D) instead of the C component. Since we had information only on twins reared together, the C and D components cannot be estimated simultaneously. Error variation is included in the E component in these models. Age was used as a covariate. The goodness of fit of the models was tested by comparing the log-likelihood values, and the assumptions of the twin modeling were tested by comparing the log-likelihood values of the ACE/ADE models with those of saturated models, which do not make these assumptions. The modeling of the twin data was

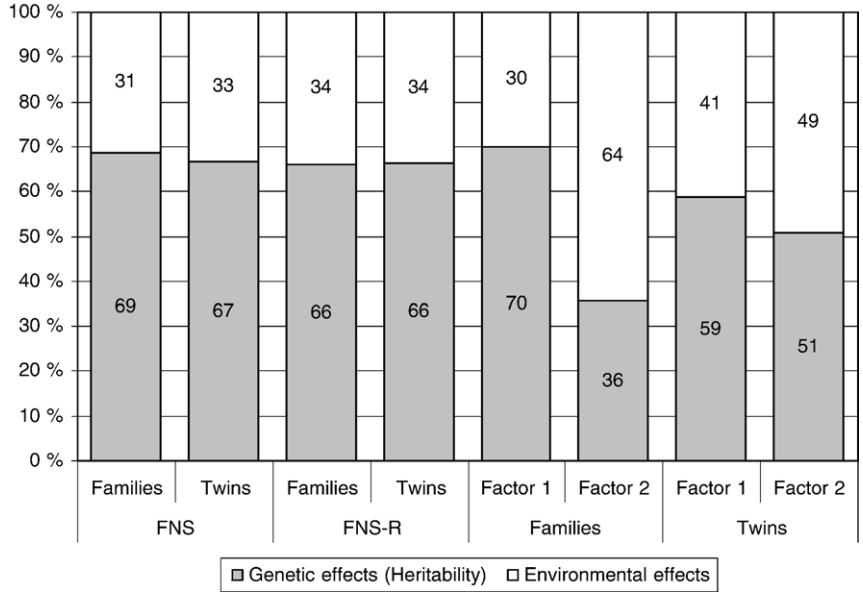

Fig. 1. Proportions of genetic and environmental effects on variation in the scores of the FNS, FNS-R, and factors 1 and 2 of the FNS in the family and twin samples.

performed using the program Mx version 1.5 [14]. Only complete twin pairs were included in the data since only they are genetically informative.

3. Results

The means of the FNS and FNS-R scores for males and females in the family sample and for MZ and DZ twins are shown in Table 3. In the family sample, the two-tailed *t*-test failed to reveal significant differences in the scores between men and women or between subjects with and without migraine. In the twin sample, the MZ twins were on average less food neophobic than the DZ twins (Table 3). There was a slight but significant positive correlation between age and the FNS score both in the Finnish ($r=0.23$, $p<0.01$) and in the British ($r=0.21$, $p<0.001$) sample.

The FNS and FNS-R scores correlated very strongly both in the Finnish ($r=0.95$; $p<0.001$) and in the British ($r=0.95$; $p<0.001$) samples after adjustment for age. Likewise, the reliabilities of these two scales described with Cronbach's α were high and similar both in the Finnish (0.84 and 0.82, for the FNS and FNS-R scores, respectively) and in the British (0.86 and 0.81, for the FNS and FNS-R scores, respectively) samples.

The factor analysis resulted in two factors in both the Finnish and the British sample when the eigenvalues of factors were constrained to values above one. In the Finnish sample, all items except eight and nine loaded mostly on the first factor (Table 2). The FNS score was very strongly correlated with the factor score of the first factor ($r=0.85$; $p<0.001$), but far less strongly with the factor score of the second factor ($r=0.49$; $p<0.001$). This suggests that the first factor reflects food neophobia purified from the confounding effect of item eight that was very strongly loaded on the second factor and negligibly on the first factor. In the British sample, negatively worded items in relation to food neophobia were loaded mostly on the first factor and positively worded items on the second factor (Table 2). FNS score was almost equally correlated with factor scores of both the first ($r=0.77$; $p<0.001$) and the second ($r=0.76$; $p<0.001$) factor score. This implies that the factors reflect rather differences in answering styles than different dimensions of food neophobia. Scores of the first and the second factor also correlated with each other in the British ($r=0.19$; $p<0.001$), but not in the Finnish ($r=-0.01$; $p>0.05$) sample, indicating more clearly separated factors and more relevant factoring in the Finnish than in the British sample.

Table 5
Model fit statistics for univariate models for the scores of the FNS, FNS-R, and the factors 1 and 2 of the FNS

	FNS	FNS-R	Factor 1	Factor 2
Saturated model				
χ^2	7165	6120	2291	2222
df	923	925	923	923
ADE model				
$\Delta\chi^2$ compared to saturated model	11	10	8	6
Δdf compared to saturated model	6	6	6	6
p-value	0.086	0.106	0.275	0.374
AE model				
$\Delta\chi^2$ compared to ADE model	6	5	1	4
Δdf compared to ADE model	1	1	1	1
p-value	0.017	0.025	0.239	0.057

ADE model = model including additive genetic (A), shared environmental (C), and non-shared environmental (E) components.
AE model = model including additive genetic (A) and non-shared environmental (E) components.

In the family data, the heritability estimates were high and nearly equal for the FNS and FNS-R scores, as well as for the score of the first factor, but only moderate for the score of the second factor (Fig. 1). In the twin data, the intra-class correlations of the MZ twins were in all cases higher than that of the DZ twins (Table 4), implying a genetic influence. The fact that the MZ correlation was more than twice higher than the DZ correlation suggested that a non-additive genetic influence may be involved and that a shared (common) environmental influence is of no account. Thus, we used the ADE model as the starting point for the analyses.

According to goodness of fit statistics (χ^2), the ADE model fitted better to the data than the ACE model. The ADE model was also appropriate for the data according to the comparison with the saturated model. When seeking a more parsimonious model, it was found that the D component cannot be left out of the model because the goodness of fit would decrease significantly in most cases (Table 5). Leaving the A component out would not significantly decrease the goodness of fit, but DE model without A component would not be biologically

Table 4
Intra-class correlations and parameter estimates of ADE-model for the FNS and FNS-R scores, and the factors formed by the factor analysis in the twin data

	Intra-class correlations		Parameter estimates					
			Additive genetic effect (a^2)		Non-additive genetic effect (d^2)		Non-shared environmental effect (e^2)	
	r(MZ)	*r*(DZ)	Estimate	95% CI	Estimate	95% CI	Estimate	95% CI
FNS	0.70	0.23	0.10	(0.00–0.56)	0.56	(0.09–0.73)	0.33	(0.27–0.41)
FNS-R	0.68	0.20	0.13	(0.00–0.59)	0.53	(0.06–0.72)	0.34	(0.28–0.41)
Factor 1	0.59	0.26	0.31	(0.00–0.64)	0.27	(0.00–0.65)	0.41	(0.34–0.50)
Factor 2	0.53	0.15	0.02	(0.00–0.50)	0.49	(0.00–0.59)	0.49	(0.41–0.59)

MZ = monozygous, DZ = dizygous.
95% CI = 95% Confidence interval.

reasonable for multigenetically inherited traits. Thus, the most appropriate univariate model for the data was found to be the ADE model, whose parameter estimates are shown in Table 4.

For both FNS and FNS-R scores, the estimates for non-additive genetic effect (d^2) were far higher than the estimates for additive genetic effect (a^2, Table 4). The confidence intervals of all estimates for genetic effects (a^2 and d^2) were wide, and their lower limits reached zero or very near to it. However, the confidence intervals of non-shared environmental effect (e^2) were narrower and their upper limits lied only at 0.41 for both FNS and FNS-R scores (Table 4). This provides further evidence of genetic contribution, as the variation of food neophobia was not entirely composed of environmental influence plus error variation. Nevertheless, the wide confidence intervals of estimates of additive and non-additive genetic effects indicate that magnitudes and the ratio of these effects cannot be estimated accurately.

4. Discussion

To our knowledge, this was the first time when the heritability estimates were determined for food neophobia in humans. In our family sample, we found an indication that food neophobia was highly heritable, which implied the existence of a genetic component. However, this familial correlation was not necessarily due to a genetic influence, but could be due to a common family environment where children learn to respond to foods similarly to their parents. To confirm the genetic influence, the twin data were analyzed.

Results from our twin data suggested that a strong genetic influence exists for food neophobia. When the ADE model was fitted to the data, the upper limit of the confidence interval of environmental effects reached 41% of variation for the FNS score, suggesting that at least 59% of variation is due to (additive and non-additive) genetic effects. We were unable to determine accurate proportions of additive and non-additive genetic effects (the confidence intervals of the estimates were wide). However, the sum of their estimates for the FNS score (67%) was close to the heritability estimate in the family sample (69%), implying that heritability estimate in the family sample was also composed of genetic contribution. In addition, no shared environmental effect was detected in the twin data. This suggests that the shared environmental effect is negligible, and the same may also apply in the family data, further supporting the existence of genetic component in the heritability estimate of families.

We assume our study samples to be appropriate for studying the heritability of food neophobia for several reasons. First, the selection bias was avoided because the subjects were not selected based on their own willingness to participate in a food related survey only, but they completed the FNS questionnaire as part of a clinical testing procedure. Second, our subjects were adults who are obviously less influenced by the shared family environment than children. Hence, genetic effects may be easier to detect in adult samples. For example, Breen et al. [15] found a substantial shared environmental effect (12–64%) in 4–5 year old children when studying the heritability of preference for different food groups. The shared environmental effect did not exist in our sample. Third, although most of the subjects were middle-aged, the age range of our subjects was still broad. Fourth, food neophobia was studied in two unrelated samples from different countries.

However, the results cannot be generalized to males without caution, because the twin sample consisted exclusively of females, and in the family sample two thirds of the subjects were females. In addition, the Finnish family sample completed a FNS questionnaire that was translated from the original English version into Finnish. Translation of questionnaires may include problems, as discussed in the case of Finnish and Swedish versions of the FNS by Ritchey et al. [9].

In general, children are more reluctant to try unfamiliar foods than adults [7,16]. Among adults food neophobia tended to decrease with age in some studies [1,16]. However, our finding that age and food neophobia are slightly positively correlated is in line with the results of a large Finnish study [3]. Yet, the practical importance of this finding may be modest, because the variation in the FNS scores was high in all age groups.

In the family sample, the factor analysis resulted in two factors, of which the first may be interpreted as a “real” food neophobia factor purified from the effect of the second factor contributed mainly by items eight and nine. Our result parallels to the result of nationally representative data by Tuorila et al. [3], with an exception that their second factor was also strongly affected by the item three. The heritability of the first factor was almost equal to that of the FNS score, suggesting that the essential content of the FNS was retained in the first factor only. Also in the twin sample, the factor analysis yielded two factors, but in this case the factor analysis was not very useful as the positively and negatively worded items loaded mostly on their own factors. The heritabilities of the factor scores were much lower than those of the FNS scores implying that either of the factors alone did not represent food neophobia. The positively and negatively worded items of the FNS formed their own factors also in a two-factor model by Ritchey et al. [9], who used confirmatory factor analysis and data from the USA.

In the Finnish sample, the reliability of the FNS measured by Cronbach’s α was very close to that reported in the Finnish population earlier (0.85; [3]). In the British sample, the reliability was even better, maybe reflecting a slight advantage of using the FNS in its original language. In both samples, the six-item FNS-R exhibited almost as high reliability as the original ten-item FNS. The heritability estimates for the scores of both scales were also similar, suggesting that both scales measured the same trait with a similar reliability and efficiency.

Pliner and Hobden [1] found the FNS score to be negatively correlated with the Experience Seeking subscale of Zuckerman’s Sensation Seeking Scale [17]. In a pilot study ($n=43$, unpublished results), we found a negative correlation ($r=-0.39$, $p<0.05$) between the FNS score and the Exploratory Excitability subscale of the Novelty Seeking dimension of Cloninger’s Temperament and Character Inventory (TCI; [18]). Thus, food neophobia seems to be inversely related with excitement related personality dimensions.

The association between novelty seeking and dopamine receptor D4 (DRD4) gene polymorphism was suggested by

several studies (e.g. [19,20]), while a few studies suggested the opposite (for meta-analysis see [21]). Hence, novelty seeking and food neophobia may be influenced by overlapping set of genes. Interestingly, in a large study with adult twins and their siblings, non-additive genetic effects were found to account for 35% phenotypic variation for novelty seeking, but no evidence of shared environmental effects was found [22]. This supports the plausibility of the substantial non-additive genetic component for food neophobia too, assuming the resemblance between food neophobia and novelty seeking.

The genetic component underlying food neophobia may also have clinical implications. When there is a need to change the diet of an individual, it may be challenging to introduce unfamiliar foods to the diets of individuals with genotype predisposing them to high food neophobia. However, the consequences of food neophobia can be successfully attenuated, at least temporarily, by several strategies. Introducing unfamiliar food accompanied by a component with familiar, liked, and appropriate flavor (flavor principle) is one such strategy [23]. Repeated taste exposure, but not mere visual and olfactory exposure, enhances the preference for initially unfamiliar foods [24]. Prior exposure to palatable unfamiliar foods has been found to increase subsequent willingness to taste also other unfamiliar foods [25]. In addition, information concerning the origin or ingredients of the product [4] or suggesting the good taste or beneficial nutritive value of unfamiliar food tends to increase positive responses to such food [16]. Although the overt food neophobic response can be modified using such strategies, the underlying neophobic personality trait may persist.

In conclusion, our results suggest that, at least in females, approximately two thirds of variation in food neophobia were attributable to genetic factors. Further research is needed to establish the heritability of food neophobia among males, to determine the proportions of additive and non-additive genetic effects, and to search for underlying genes and to associate their variants with different levels of food neophobia.

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