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Genetic and environmental contributions to food use patterns of young adult twins☆

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Abstract

The contribution of genetic factors to individual differences in food use was estimated in a large population-based twin cohort of young adults (22- to 27-year-old). Male and female twins (n=2009 complete twin pairs) evaluated use-frequencies of 24 food items using 5 categories (1=never–5=several times a day) in a postal questionnaire. Foods were categorized by factor analysis. Estimates of the relative proportions of additive genetic, shared environmental, and unshared environmental effects on the use-frequency of food items and factor scores were obtained by quantitative genetic modeling of twin data based on linear structural equations. Four factors of food use were identified: "healthy" foods, high-fat foods, sweet foods, and meats. The variance of the use-frequency of food items and food categories was explained by additive genetic and unshared environmental influences, whereas shared environmental factors did not contribute to food use. The average proportions of genetic effects on the total variance of the use-frequency of food items and food categories were 40% and 45%, respectively. Sex differences were observed in the magnitude of genetic influences for use-frequency of four food items (chocolate, other sweets, fried foods, and meat), and in genetic factors underlying the use of three (fresh vegetables, fruits, and cheeses) items. In conclusion, family environment does not appear to influence the food use of young adults and thus nutritional education should be targeted at this age group to support development of healthy eating patterns. In addition, the results illuminate the importance of the sex-specific genetic effects on food use.

Keywords: Food-frequency questionnaire; Twins; Genetic effects; Food habits; Young adults

1. Introduction

Food choice is a luxury strongly affecting people's health in Western countries [1]. Besides nutritional impact, food choice has economic, political, and social relevance. Nevertheless, its underlying mechanisms in the population at large remain poorly understood. Children's food preferences are often determined by taste (especially sweetness) and familiarity, while adults' choices and preferences are also influenced by attitudinal (e.g. towards nutrition), cultural, situational, physiological, and economic factors [2].

Previous studies have evaluated the contribution of genetic factors in food consumption [3–5] and nutrient intake [6–8] of

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adults. Both a study of 18- to 77-year-old twins reared apart and their husbands [6] and a study of twins aged ≥50 years [5] showed that approximately 30% of the variance in food use was attributable to genetic factors. They also concluded that shared environment, i.e. factors common to family members, did not contribute to food use of adults. However, a recent twin study [9] showed that more than half of the variation in dessert and vegetable preferences of 4- to 5-year-old children was due to shared environment. Thus, the contribution of genetic factors likely evolves with age, and studies of specific age groups are needed to investigate the genetic architecture of food use and preferences.

Our aim was to examine the relative proportions of genetic and environmental effects on food use of 22- to 27-year-old subjects. To our knowledge, no earlier studies have investigated factors affecting food use at an age at which subjects are becoming independent and starting to make their own food choices. We estimated the relative influence of genetic and environmental factors on the use-frequency of 24 food items and on four patterns of food use, constructed using factor analysis, in a uniquely large twin study sample with males and females, a narrow age distribution and high population representativeness.

2. Materials and methods

2.1. Data collection

Data were derived from five consecutive and complete one-year cohorts of Finnish twins born in 1975–1979 [10]. The response rate to the baseline survey at age 16 was 88%. The data used in this study were based on the fourth wave of that study, conducted in 2000–2002. The questionnaire was mailed to baseline respondents in each birth cohort at semi-annual intervals such that it was mailed to those born in 1975 in autumn of 2000, those born in 1976 in the spring of 2001, and so on, with those born in 1979 receiving the questionnaire in autumn of 2002. Thus, at the time of study, the subjects were aged 22–27 years (mean age 24.4 years, SD 0.82 years). A total of 5240 subjects participated in the fourth study wave. The response rate in the fourth wave was 88%, thus including 77% of all twin individuals belonging to the five birth cohorts. The study protocol was approved by the Ethics Committee of Helsinki University Hospital.

Replies to the food-frequency questionnaire from 4667 subjects (89% of the participants) were available at the time of analysis. Only the subjects whose zygosity could be classified

Table 1
Response frequencies (%) of males and females

Variable	Males (n=1962)						Females (n=2462)					
	Never	A couple times a month or more rarely	A couple times a week	Once a day	Several times a day	Missing	Never	A couple times a month or more rarely	A couple times a week	Once a day	Several times a 2day	Missing
Cooked or mashed potatoes	0.9	22.3	54.6	20.5	1.5	0.2	1.3	29.1	53.5	15.0	1.0	0.2
Fried potatoes or French fries [a]	3.3	66.7	29.8	–	–	0.2	9.1	76.9	13.8	–	–	0.2
Rice or pasta	1.7	32.4	55.7	9.3	0.8	0.2	0.7	20.9	69.8	7.9	0.4	0.2
Porridge, muesli, cereals	15.5	41.6	21.8	19.2	1.6	0.3	8.0	33.8	30.6	25.1	2.0	0.4
Yoghurt	6.3	27.1	36.5	24.6	5.1	0.3	3.9	16.6	36.7	35.4	6.8	0.6
Reduced-fat cheeses	35.1	36.4	16.0	8.4	3.3	0.7	19.4	35.5	20.2	14.3	9.9	0.6
Other cheeses (e.g. Emmental, Brie)	5.5	17.5	29.1	28.4	19.4	0.1	7.9	26.4	27.0	21.0	17.7	0.1
Fish (hot or cold) [b]	7.4	64.6	26.2	1.0	–	0.7	7.8	63.9	26.5	0.7	–	1.1
Chicken, turkey (hot or cold)	4.4	48.2	43.9	2.8	0.3	0.3	5.8	37.1	52.5	3.7	0.7	0.3
Meat (hot or cold)	2.4	8.9	52.5	28.1	7.7	0.4	7.4	17.7	55.9	14.5	3.6	0.9
Sausage (hot or cold)	4.1	25.2	41.7	21.8	6.5	0.8	15.7	40.9	30.4	9.0	3.5	0.6
Eggs (boiled, fried, omelette)	5.3	60.0	31.8	2.3	0.3	0.3	6.1	69.6	22.9	1.1	0.1	0.2
Fresh vegetables	3.4	24.8	37.9	28.8	4.6	0.5	0.7	14.0	35.9	35.1	13.9	0.4
Cooked vegetables	12.4	46.0	34.7	6.2	0.7	0.3	5.8	34.9	43.9	13.2	2.0	0.3
Fruits	1.8	27.4	43.5	20.8	6.2	0.2	0.6	11.5	34.1	32.9	20.5	0.4
Berries	5.9	63.0	27.0	3.2	0.5	0.4	3.5	56.1	32.0	6.4	1.3	0.7
Sweet desserts (sweet pastries, cakes, ice-cream, etc.)	2.8	49.5	41.0	5.8	0.5	0.3	1.8	45.7	44.0	7.7	0.4	0.4
Chocolate [b]	8.0	61.8	28.6	1.3	–	0.3	4.8	53.2	37.2	4.5	–	0.2
Other sweets	4.6	46.0	43.9	4.8	0.3	0.3	6.0	76.0	17.0	0.7	0.0	0.3
Salty snacks (e.g. chips, popcorn, salted peanuts) [b]	6.2	69.8	22.9	0.8	–	0.3	6.0	76.0	17.0	0.7	–	0.3
Pizza [b]	2.3	75.7	21.2	0.6	–	0.2	3.7	87.6	8.3	0.1	–	0.2
Hamburgers [a]	7.2	74.6	18.0	–	–	0.2	14.0	79.9	5.9	–	–	0.2
Fried foods [b]	6.1	62.0	29.4	2.2	–	0.3	14.6	70.7	13.5	0.8	–	0.4
Creamy foods [b]	7.8	68.5	22.6	0.7	–	0.4	11.9	72.1	15.4	0.2	–	0.3

[a] Response alternatives “a couple times a week”, “once a day” and “several times a day” combined.
[b] Response alternatives “once a day” and “several times a day” combined.

(94%) were included in the study, yielding a sample size of 4388 twin individuals (44.7% males, 55.3% females). This sample consisted of 1408 monozygotic (MZ) (38.4% males, 61.6% females), 1470 same-sex dizygotic (SSDZ) (48.0% males, 52.0% females), and 1510 opposite-sex dizygotic (OSDZ) twin individuals (47.5% males, 52.5% females). The data included 2009 complete twin pairs (12.4% MZ male, 20.6% MZ female, 15.3% SSDZ male, 17.9% SSDZ female, 33.7% OSDZ pairs) of known zygosity with both members phenotyped. Zygosity was assessed by a deterministic algorithm using questions on physical similarity during school age, which was shown to have high validity in another Finnish twin cohort [11].

The food-frequency questionnaire included 24 food items that are common in the Finnish diet. The questionnaire was modified from a previous national food-frequency questionnaire [12] and covered the main food groups (cereals, rice, pasta, meat, poultry, fish, eggs, fresh and cooked vegetables, fruits, berries, milk products, yoghurt, cheese, fats, oils, sweets, and fast food). Participants were asked to evaluate how often they consume these food items using five categories (1=never, 2=a couple times a month or more rarely, 3=a couple times a week, 4=once a day, 5=several times a day).

2.2. Statistical analysis

Factor analysis with a maximum likelihood extraction method and orthogonal Varimax rotation was conducted for the use-frequency of the 24 food items of the entire sample. The number of factors (food categories) was decided from scree plots of eigenvalues and by screening based on meaningful contents. The reliability of the components was further evaluated by Cronbach's alpha value. Factor scores were computed using the Anderson–Rubin method, yielding latent variables with the mean value of 0 and standard deviation of 1. Thus, for each subject, a factor score was obtained based on the use-frequencies of the food items loading to the factor in question. Except for quantitative genetic modeling, all statistical analyses were performed with the SPSS statistical package [13].

Classical twin modeling relies on the assumption that MZ twins are genetically identical, whereas DZ twins share approximately half of their segregating genes [14]. Genetic variation can be divided into additive genetic variation, which consists of the sum of the allelic effects on the phenotype over all relevant loci, and nonadditive genetic variation, which includes the interaction of alleles in the same locus (dominance) as well as between loci (epistasis). The correlations of both additive and nonadditive genetic effects are 1 within MZ pairs. Within DZ pairs, the correlations are 0.5 for additive and 0.25 for nonadditive genetic effects.

Thus, if correlations within MZ pairs are higher than within DZ pairs, this provides evidence that genetic factors affect the trait. If within-pair MZ correlations are about twice the DZ correlations, the genetic effects are likely to be additive. If the MZ correlations are higher than double the DZ correlations, nonadditive genetic effects are probably also involved. Different correlation patterns for male and female twin pair groups and lower correlations for opposite-sex than same-sex DZ pairs suggest that sex-specific differences exist in genetic influences or environmental influences, or both [16].

Environmental variation can be divided into environmental factors shared and unshared by co-twins. A shared environment, having a similar effect on MZ and DZ pairs, includes all environmental factors that make the twin pair similar for the trait, such as shared childhood experiences, parental socioeconomic status, and the same friends. An unshared environment includes all environmental factors and experiences that make siblings in the family dissimilar, including measurement error. Thus, the correlations of shared and unshared environmental effects are defined as 1 and 0, respectively, within both MZ and DZ twin pairs. Random mating with respect to the traits in question and the absence of gene–environment interactions or correlations are also assumed in the model. The assortative mating would result in genetic correlations of DZ twin pairs above 0.5 which would lead to increase in common environmental variance and decrease in the estimates of genetic effects. Both the gene–environment interactions (individuals with different genotypes responding differently to an environment) and correlations (nonrandom distribution of environments among different genotypes) increase the effect of additive genetic factors [15].

On the basis of these assumptions, the proportion of phenotypic variance explained by additive genetic effects (A), dominant (nonadditive) genetic effects (D), common (shared) environmental effects (C), and unshared environmental effects

Table 2
Correlations of food items within twin pairs according to zygosity and sex

Food item	MZM [a]	DZM [b]	MZF [c]	DZF [d]	OSDZ [e]
Cooked or mashed potatoes	0.49	0.27	0.47	0.21	0.15
Fried potatoes or French fries	0.41	0.27	0.39	0.30	0.10
Rice or pasta	0.49	0.28	0.38	0.32	0.18
Porridge, muesli, cereals	0.44	0.24	0.42	0.23	0.14
Yoghurt	0.58	0.08	0.38	0.22	0.15
Reduced-fat cheeses	0.51	0.18	0.47	0.08	0.24
Other cheeses	0.44	0.16	0.40	0.20	0.10
Fish	0.52	0.20	0.44	0.23	0.12
Chicken, turkey	0.46	0.28	0.44	0.36	0.23
Meat	0.23	0.17	0.42	0.32	0.11
Sausage	0.38	0.26	0.48	0.28	0.20
Eggs	0.34	0.16	0.44	0.08	0.09
Fresh vegetables	0.46	0.23	0.49	0.26	0.12
Cooked vegetables	0.40	0.20	0.51	0.19	0.22
Fruits	0.55	0.26	0.52	0.29	0.16
Berries	0.42	0.24	0.46	0.30	0.13
Sweet desserts	0.26	0.24	0.35	0.13	0.12
Chocolate	0.45	0.17	0.39	0.27	0.06
Other sweets	0.42	0.40	0.58	0.28	0.11
Salty snacks	0.44	0.27	0.44	0.25	0.13
Pizza	0.36	0.23	0.49	0.21	0.13
Hamburgers	0.65	0.15	0.55	0.35	0.16
Fried foods	0.24	0.21	0.44	0.21	0.09
Creamy foods	0.36	0.19	0.28	0.20	0.14

[a] Monozygotic male (n=413) pairs.
[b] Dizygotic male (n=360) pairs.
[c] Monozygotic female (n=250) pairs.
[d] Dizygotic female (n=308) pairs.
[e] Opposite-sex dizygotic (n=678) pairs.

(E) can be estimated. In genetic modeling, these variance components are treated as latent (unmeasured) and standardized independent variables, which are used to explain the variation of the trait, treated as the dependent variable in the model. Thus, the regression coefficients of these latent variables are the square roots of the genetic and environmental variance components affecting the trait. Because simultaneous estimation of the shared environment (C) and dominant (D) genetic variance components requires adopted twins or other relatives, we were unable to separate these components in this study [14].

Genetic modeling was carried out with the Mx statistical package, version 1.5 [17]. As Mx expects that there are cases in every response alternative in each of the five zygosity and sex groups (MZ males and females, DZ males and females, DZ opposite sex), the response alternatives of some of the food items had to be combined. For six food items (fish, chocolate, salty snacks, pizza, fried foods, and creamy foods), the two response alternatives "once a day" and "several times a day" were combined, and for two food items (fried potatoes or French fries and hamburgers) the three response alternatives "a couple times a week", "once a day", and "several times a day" were combined. These transformed variables were used in all of the analyses.

Linear structural equation models estimating variance components A, C or D, and E were built for use-frequencies of individual food items and for food category (factor) scores. In the analyses, individual food items were regarded as ordinal variables assuming a normally distributed liability function behind the observed phenotypes. The food category scores were regarded as continuous variables. The model fits were assessed using chi-square (χ^2) goodness-of-fit statistics. First, the chi-square change ($\Delta\chi^2$) between the twin model and the saturated model was examined. The saturated model did not make any of the assumptions of the twin model and tests whether birth order, zygosity, sex, age effects on mean and variance of the traits exist. If the change in χ^2-values compared with the change in degrees of freedom measured by a p-value was more than 0.05, the twin model was considered to fit the data. Next, we tested whether the estimates of males and females differed from each other and whether there were sex-specific genetic effects underlying the trait, i.e. different genes affecting the trait in males and females. Finally, we examined which model best describes the variation of the trait in question. If exclusion of a parameter made the model fit significantly worse than the general overall (ACE) model, the parameter was judged to be statistically significant.

3. Results

Frequencies of responses to different response alternatives are shown in Table 1, separately for males and females. The within-pair polychoric correlations of individual food items by sex and zygosity are presented in Table 2. MZ twins resembled each other more than DZ twins, suggesting that genetic effects

Table 3
Estimates of proportional influences of additive genetic effects (a^2) and unshared environmental effects (e^2) on use of 24 food items with no sex-specific genetic effects allowed in the model

Food item	Males				Females			
	a^2	CI 95%	e^2	CI 95%	a^2	CI 95%	e^2	CI 95%
Cooked or mashed potatoes	0.46	(0.35–0.55)	0.54	(0.45–0.65)	0.44	(0.35–0.53)	0.56	(0.47–0.65)
Fried potatoes or French fries	0.40	(0.25–0.53)	0.60	(0.47–0.75)	0.38	(0.26–0.49)	0.62	(0.51–0.74)
Rice or pasta	0.49	(0.38–0.60)	0.51	(0.40–0.62)	0.40	(0.30–0.50)	0.60	(0.50–0.70)
Porridge, muesli, cereals	0.42	(0.31–0.52)	0.58	(0.48–0.69)	0.41	(0.32–0.49)	0.59	(0.51–0.68)
Yoghurt	0.48	(0.38–0.57)	0.52	(0.43–0.62)	0.37	(0.29–0.45)	0.63	(0.55–0.72)
Reduced-fat cheeses	0.46	(0.36–0.56)	0.54	(0.44–0.64)	0.43	(0.34–0.50)	0.57	(0.50–0.66)
Other cheeses [a]	0.38	(0.27–0.48)	0.62	(0.52–0.73)	0.37	(0.29–0.45)	0.63	(0.55–0.71)
Fish	0.45	(0.32–0.56)	0.55	(0.44–0.53)	0.44	(0.33–0.53)	0.57	(0.47–0.67)
Chicken, turkey	0.47	(0.36–0.57)	0.53	(0.43–0.64)	0.49	(0.40–0.57)	0.51	(0.43–0.60)
Meat	0.22	(0.11–0.32)	0.78	(0.68–0.89)	0.44	(0.35–0.52)	0.56	(0.48–0.65)
Sausage	0.40	(0.30–0.49)	0.60	(0.51–0.70)	0.46	(0.39–0.53)	0.54	(0.47–0.61)
Eggs	0.30	(0.16–0.44)	0.70	(0.56–0.84)	0.37	(0.25–0.48)	0.63	(0.52–0.75)
Fresh vegetables [a]	0.40	(0.30–0.50)	0.60	(0.50–0.70)	0.48	(0.39–0.55)	0.52	(0.45–0.61)
Cooked vegetables	0.38	(0.27–0.48)	0.62	(0.52–0.73)	0.50	(0.41–0.57)	0.50	(0.43–0.59)
Fruits [a]	0.51	(0.40–0.60)	0.49	(0.40–0.60)	0.49	(0.41–0.56)	0.51	(0.44–0.59)
Berries	0.37	(0.25–0.49)	0.63	(0.51–0.75)	0.44	(0.35–0.53)	0.56	(0.47–0.65)
Sweet desserts	0.23	(0.17–0.41)	0.71	(0.59–0.84)	0.33	(0.23–0.43)	0.67	(0.57–0.77)
Chocolate [b]	0.37	(0.23–0.50)	0.63	(0.50–0.77)	0.38	(0.27–0.48)	0.62	(0.52–0.73)
Other sweets [c]	0.55	(0.40–0.68)	0.45	(0.32–0.60)	0.54	(0.42–0.60)	0.46	(0.35–0.58)
Salty snacks	0.43	(0.28–0.56)	0.57	(0.44–0.72)	0.41	(0.29–0.52)	0.59	(0.48–0.71)
Pizza	0.34	(0.18–0.49)	0.66	(0.51–0.82)	0.47	(0.28–0.63)	0.53	(0.37–0.72)
Hamburgers [b]	0.55	(0.41–0.68)	0.45	(0.32–0.60)	0.54	(0.42–0.64)	0.46	(0.36–0.58)
Fried foods	0.22	(0.09–0.34)	0.78	(0.66–0.91)	0.43	(0.32–0.52)	0.57	(0.48–0.68)
Creamy foods	0.36	(0.22–0.49)	0.64	(0.51–0.78)	0.29	(0.18–0.39)	0.71	(0.61–0.82)

[a] Allowing sex-specific genetic effects would provide a better model fit.
[b] ACE model would provide a better fit for females.
[c] ACE model would provide a better fit for males.

Table 4
Factor analysis of 21 food items in 4388 twin individuals

Item	F1 (17.0%, α=0.73)	F2 (13.4%, α=0.72)	F3 (7.6%, α=0.66)	F4 (6.7%, α=0.64)
Fresh vegetables	0.66			
Fruits	0.63			
Cooked vegetables	0.58			
Berries	0.52			
Porridge, muesli, cereals	0.45			
Reduced-fat cheeses	0.39			
Rice or pasta	0.37			
Chicken	0.35			
Yoghurt	0.34			
Fish	0.34			
Fried foods		0.61		
Hamburgers		0.61		
Pizza		0.53		
Fried potatoes or French fries		0.50		
Creamy foods		0.48		
Salty snacks		0.43	0.32	
Other sweets			0.66	
Chocolate			0.65	
Sweet desserts			0.51	
Sausage				0.67
Meat				0.64

Derived attributes and item loadings greater than 0.30 of items on each factor. Explanation rates and Cronbach's alphas are shown in parenthesis.

underlie the use-frequencies of all food items. As correlations within MZ pairs were on average twice those of DZ pairs, the genetic effects are mainly additive. Thus, based on the within-pair correlation pattern, the ACE model was chosen as a general model instead of the ADE model. Modeling was carried out to formally find the best-fitting model.

The AE model with no sex-specific genetic effects provided the best fit for most of the use-frequencies of individual food items. Even in cases in which allowance of sex-specific genetic effects or inclusion of common environmental effects (C) provided higher χ^2-values, a model without these parameters fit the data well when compared with the saturated model (poorest change in fit $\Delta\chi^2_{35}=41$, $p=0.234$ for reduced-fat cheeses). Therefore, from here onwards we focus on the results of the AE models only. For most of the food items, a model with the same variance components for men and women would have provided a good fit. However, for four food items (meat, fried foods, chocolate, other sweets), the estimates of males differed significantly from those of females. Thus, models with different magnitudes of genetic and environmental influences for men and women were calculated for all food items.

The proportional effects of the additive genetic and specific environmental variance components with their 95% confidence intervals are provided in Table 3. On average, the contribution of genetic factors to use-frequency of individual food items was 40%. The highest heritability estimates were obtained for hamburgers (55% for males, 54% for females), other sweets except chocolate (55% for males, 54% for females), and fruits (51% for males, 49% for females). The items with the lowest heritability estimates were creamy foods (36% for males, 29% for females) and eggs (30% for males, 37% for females); in the models for males only also fried foods, meat, and sweet desserts (22%, 22%, and 23%, respectively).

A small proportion (3.2%) of the subjects was identified as vegetarians, determined as not consuming meat and chicken/turkey. The tetrachoric within-pair correlation coefficients for vegetarianism were 1.00 for MZ male, 0.85 for MZ female, 0.77 for SSDZ male, 0.79 for SSDZ female, and 0.54 for OSDZ pairs. The assumption that a single liability to the frequency of use underlies the reported use was tested by examining whether the cross-tabulation of the frequency of use within twin pairs deviated from expectation of a bivariate normal liability distribution by tests on normality by using a maximum likelihood ratio test. We found that nearly all variables followed within-pair bivariate latent normal distribution and that the observed deviations were not systematic with respect to sex or inclusion/exclusion of vegetarians. We thus considered that the same mechanisms underlie use versus no use and the frequency of use.

Factor analysis was conducted on the use-frequencies of all 24 food items. Three items (cooked or mashed potatoes, other cheeses, and eggs) were omitted since they did not load on any factor or they considerably weakened the reliability of a factor measured by Cronbach's alpha. After this, factor analysis was performed again on the use-frequencies of the 21 remaining food items. These ratings loaded on four factors, explaining 45% of the variance, and we chose to call the factors according to essential content (Table 4). Factors 1 ("healthy" foods) and 2 (high-fat foods) reflect lighter and heavier eating patterns, respectively, and factors 3 (sweet foods) and 4 (meats) sensory or nutritional content of the foods. Each food item loaded on only one component, the only exception being "salty snacks" which loaded on both factor 2 and factor 3. This exception may arise from popcorns, which may be sweet or salty, being among the examples of food items for this variable. When calculating Cronbach's alpha values, the variable "salty snacks" was considered to belong only to factor 2, on which it loaded more strongly. The within-pair correlations (Pearson's r) of the food category factor scores are shown in Table 5. Again, the correlations within MZ pairs are higher than those of DZ pairs. In addition, the correlations within opposite-sex DZ pairs are markedly lower than those of same-sex DZ (DZF and DZM) pairs, especially in the case of factor 3, suggesting that the genetics underlying the use-frequency of sweet foods in males and females are different.

Table 5
Correlations of factor scores within monozygous and dizygous twin pairs

Component	MZM [a]	DZM [b]	MZF [c]	DZF [d]	OSDZ [e]
F1 "Healthy" foods	0.51	0.26	0.56	0.27	0.18
F2 High-fat foods	0.41	0.24	0.44	0.29	0.16
F3 Sweet foods	0.37	0.29	0.42	0.29	0.08
F4 Meat	0.39	0.24	0.40	0.32	0.20

[a] Monozygotic male ($n=413$) pairs.
[b] Dizygotic male ($n=360$) pairs.
[c] Monozygotic female ($n=250$) pairs.
[d] Dizygotic female ($n=308$) pairs.
[e] Opposite-sex dizygotic ($n=678$) pairs.

Table 6
Estimates of proportional influences of additive genetic effects (a^2) and unshared environmental effects (e^2) on factor scores with sex-specific genetic effects allowed in the model

Factor	Males				Females			
	a^2	CI 95%	e^2	CI 95%	a^2	CI 95%	e^2	CI 95%
F1 "Healthy" foods	0.49	(0.40–0.56)	0.51	(0.44–0.60)	0.54	(0.47–0.60)	0.46	(0.40–0.53)
F2 High-fat foods	0.44	(0.34–0.53)	0.56	(0.47–0.66)	0.47	(0.39–0.53)	0.53	(0.47–0.61)
F3 Sweet foods	0.42	(0.32–0.51)	0.58	(0.49–0.68)	0.43	(0.36–0.50)	0.57	(0.50–0.64)
F4 Meat[a]	0.39	(0.30–0.48)	0.61	(0.52–0.70)	0.44	(0.36–0.51)	0.56	(0.49–0.64)

[a] ACE model with no sex-specific genetic effects would provide a better fit.

The models presented for the food categories (Table 6) are AE models with sex-specific genetic effects allowed, the fits of which were good compared with a saturated model ($p>0.05$). If sex-specific genetic effects were not allowed, it caused a decrease in the χ^2-value of the model, significantly worsening the model fit ($p<0.05$) for all food categories except factor 4. This implies that there are partly different genes in males and females underlying the use of foods loading on the other three factors. The variance components of males and females differed significantly from each other in factor 2 (high-fat foods) model. To enable comparison between the models of food categories, all were estimated separately for males and females. The proportion of genetic effects of the total variation was for all food categories around 45%, varying from 39% (factor 4, meats in males) to 54% (factor 1, "healthy" foods in females). Having lived together, on average, until the age of 18.8 years, almost all (92%) of the twin pairs now lived apart. Quantitative genetic models of only these pairs (results not presented here) were virtually identical to the models of the entire study sample.

4. Discussion

4.1. Genetic and environmental influences on males and females

Our results support the view that use-frequency of foods is influenced by genetic differences among individuals and that the common environmental factors do not influence food use in young adulthood. The average proportions of additive genetic effects of the total phenotypic variance in use-frequency of individual food items and in the four food categories were 40% and 45%, respectively, and the rest of the variance was explained by unique environmental factors. Young adulthood is typically a transition period between childhood at home and independent adult life. This change in life may lead to alterations in food habits. For example, the liability for weight gain is increased in young adulthood, and this may lead to a discordance in weight even in genetically identical monozygotic twins [18], highlighting the importance of individual lifestyle and environmental factors in the health status of young adults.

In children, food preferences are determined by genetic and unshared environmental factors, as well as by shared environmental factors [9]. However, in line with the present study, several previous studies have shown that a shared childhood family environment does not affect food use [3–5] or nutrient intake [6–8] in adults. The phenotypes measured, the statistical models applied, the size, sex, age, and zygosity distributions of the samples and the representativeness of the study population have varied widely in these studies. Despite methodological differences and the lack of longitudinal studies, all data indicate that the influence of family on the variation in food behavior ceases after childhood. Other factors, e.g. social considerations, time pressure and structure of the day, may start influencing the food use in young adulthood [19]. This presents new challenges for targeting nutritional education at adolescents and young adults to increase their awareness of nutrition as well as their ability to implement this knowledge in daily food choices.

The heritability estimates were very similar for the use-frequencies of different foods. Thus, no clear trends were observed in the heritability of use of foods with certain sensory or nutritional qualities. The underlying genes might be affecting sensory functions or preferences, other aspects of food behavior (e.g. ability to restrain from eating [20]), or both. Moreover, expression of the genes can also vary at different ages, between sexes, and in different metabolic states, e.g. in acquired obesity [21].

Sex differences were observed in the magnitude of genetic influence and in the genes underlying food use. The heritability estimates of males and females differed significantly for the use of chocolate (ACE model provided better fit for females than AE model), other sweets (ACE model provided better fit for males than AE model), fried foods ($a^2=0.22$ for males and 0.43 for females), and meat ($a^2=0.22$ for males and 0.43 for females). Sex-specific genetic effects (i.e. different genes affecting males and females) were significant for three foods: fresh vegetables, fruits, and cheeses. These effects were, however, significant for three of the four patterns of food use. This may be due to the greater power of analysis when continuous variables instead of categorical variables were used to reveal such effects. The sex differences for the intake of macronutrients, fluids, and 13 food and six beverage item types obtained by 7-day food intake diaries of 530 twin individuals with average age of 40.2 years (SD 10.6) were investigated by de Castro [22]. Sex differences were only found in the variance components of the between-meal ingestion of fluids. Thus, while genetic and environmental effects on the amount of macronutrients or certain foods ingested appear to be similar in males and females, sex differences may exist in the variance components of the frequency of use of foods or food types. It is, however, noteworthy that the age distribution of the study of de Castro differs significantly from that of ours.

Age and sex differences have been also shown to exist in the genetic and environmental influences on eating disorders. Klump et al. [23] evaluated disordered eating attitudes in 11- and 17-year-old female twins. Univariate models showed that in 17-year-old adolescents the genetic effects (>50% of the variance) had a greater influence on eating attitudes and behavior than in 11-year-old children (3–9%). In a further study, the authors divided the group of 11-year-old females into prepubertal and pubertal twins and found that the genetic effects start contributing to eating pathology during puberty [24]. Disordered eating, measured on two subscales of the Eating Disorder Inventory, namely body dissatisfaction and drive for thinness, was studied in a sample of young Finnish adult twins by Keski-Rahkonen et al. [25], who found that in females additive genetic effects accounted for 59% and 51% of variance in body dissatisfaction and drive for thinness, respectively, whereas in males, they accounted for none of the variance.

4.2. Methodological considerations

While our study has several strengths, some limitations also exist. The study sample is large, and with a high response rate and a narrow age distribution, it provides good representativeness of the target population. The data are based on self-reported use-frequencies of foods. Thus, the method does not provide information about consumed quantities and may poorly represent the nutrient intake as many of the food items may include foods with different nutritional values and on the other hand, only a limited range of foods was included in the list of 24 foods. However, it has been shown that enquiry of individual portion size adds only limited information in large epidemiologic studies [26]. Furthermore, in a study of young women (n=87, mean age 25.4 years) use-frequency scores were correlated with self-reported liking for 75 foods (median r=0.40) and predicted dietary outcomes measured as intake of fat, fiber, and vitamin C [27]. Food preferences and consumption are measured by many, often correlated measures. In a recent study of Tuorila et al. [28], the pleasantness ("very unpleasant"–"very pleasant") and liking ("not at all"–"very much") ratings were correlated with the use-frequencies ("never"–"2–4 times a day") of ten foods (the food names used as stimuli), with mean correlations (r) of 0.52 and 0.53, respectively. Although commonly used affective and consumption scales are intercorrelated, they may reveal somewhat different aspects of food preferences, and poorly translate to consumed quantities.

Misreporting of food use may have occurred either consciously or unconsciously. Women and individuals with a low education or a high body mass index (BMI) have, for instance, been shown to underreport their diet [29]. Both underreporting and overreporting may also derive from the social desirability of an eating pattern. In our study, misreporting could also have been caused by variables, such as "creamy foods" and "sweet desserts", being unclear to the subjects, although in the latter case, examples were given to clarify the concept. In addition, some items in our questionnaire consisted of several foods, e.g. variables "meat (hot or cold)" and "fresh vegetables". It may have been difficult for the subjects to consider all foods of the given group when providing their estimates of use-frequencies. Furthermore, the food items were chosen to cover the diet as fully as possible, thus the rated foods are commonly used in the Finnish diet. In the homogenous Finnish food culture [30], analyzing variation in use-frequency ratings of common food items may have allowed a situation in which the variation shared by a twin pair merely represents genetic influences. If less common food items or subjects with different cultural backgrounds were included, more variation due to shared environmental effects might have been observed.

In a classical twin study, with no information on other family members, one cannot provide an estimate of common environmental (C) and nonadditive genetic (D) variation at the same time. Based on the within-pair correlation patterns, we used an ACE model as a general model, thus fixing the nonadditive genetic effects to 0. The classical twin design also expects no gene–environment interaction or random mating with respect to the traits. Coventry and Keller [31] compared parameter estimates of 17 phenotypes (e.g. BMI, stature, and measures of social attitudes and mental health) obtained from extended twin-family studies, which allow estimation of more than three parameters, with parameters obtained using classical twin design. They found that with the classical twin design shared environmental factors (C) were, depending on the phenotype, either lower or confounded with assortative mating, nonadditive genetic factors (D) were an average of 43% lower, and additive genetic factors (A) were 63% higher than those obtained using extended twin-family design. However, the estimates of the variation due to all genetic factors (broad-sense heritability) were only 18% higher using classical twin design. Thus, the parameter estimates of classical twin studies provide one set of estimates of heritability, which need to be replicated using other study designs, including the identification of the genes specifically responsible for the genetic variance.

4.3. Conclusions

Almost half of the variation in food use of young adults appears to be inherited. Environmental effects shared by siblings, e.g. childhood family environment and common friends, do not influence the variation in food use of young adults. Thus, the effect of the family environment on individual differences in food use apparently ceases after childhood. This highlights the importance of targeting dietary education at young adults, as this is a relevant and timely moment to support the development of healthy eating patterns, thus preventing nutritional problems later in life.

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