Same genetic components underlie different measures of sweet taste preference1–3

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ABSTRACT

Background: Sweet taste preferences are measured by several often correlated measures.

Objective: We examined the relative proportions of genetic and environmental effects on sweet taste preference indicators and their mutual correlations.

Design: A total of 663 female twins (324 complete pairs, 149 monozygous and 175 dizygous pairs) aged 17–80 y rated the liking and intensity of a 20% (wt/vol) sucrose solution, reported the liking and the use-frequency of 6 sweet foods (sweet desserts, sweets, sweet pastry, ice cream, hard candy, and chocolate), and completed a questionnaire on cravings of sweet foods. The estimated contributions of genetic factors, environmental factors shared by a twin pair, and environmental factors unique to each twin individual to the variance and covariance of the traits were obtained with the use of linear structural equation modeling.

Results: Approximately half of the variation in liking for sweet solution and liking and use-frequency of sweet foods (49–53%) was explained by genetic factors, whereas the rest of the variation was due to environmental factors unique to each twin individual. Sweet taste preference–related traits were correlated. Tetravariate model showed that the correlation between liking for the sweet solution and liking for sweet foods was due to genetic factors (genetic r = 0.27). Correlations between liking, use-frequency, and craving for sweet foods were due to both genetic and unshared environmental factors.

Conclusion: Detailed information on the associations between preference measures is an important intermediate goal in the determination of the genetic components affecting sweet taste preferences. Am J Clin Nutr 2007;86:1663–9.

KEY WORDS Twin study, sweet taste, genetic effects, heritability, taste preferences

INTRODUCTION

Humans have innate preference for a sweet taste (1), but the degree of liking for sweetness varies greatly among individuals (2). This variation is likely to have environmental roots, but it may also have a genetic component (3). Although individual differences exist, most people find sweet foods palatable, which has led to an extensive supply and consumption of sugar-containing products. Nutritionally beneficial foods, such as fruit, often naturally contain sugars, but foods with added sugars are disadvantageous to health because of extra calories and an increased risk of dental caries. Dietary guidelines worldwide discourage the consumption of added sugar (4).

Several methods for measuring sweet taste preferences have been developed. In chemosensory tests, aqueous solutions of sucrose have often been used as the taste stimulus. However, the preference for sugar in water may poorly represent the liking for sweet foods, not to mention their actual use. In a cross-cultural study of 122 students, Holt et al (5) found that liking for sweetness in an aqueous solution did not predict the degree of liking for sweetness in orange juice, custard, or shortbread biscuits.

An easier and less expensive way to collect data on sweetness preferences is to use postal or electronic questionnaires. Usually, a list of foods is presented to a subject and he or she is requested to evaluate liking or use-frequency of the foods. In the case of sweet foods, the liking and the use-frequency of a food item are often correlated (6, 7). In addition, behavioral questionnaires measuring the tendency to crave sweet foods (8, 9) or attitudes toward them (6, 10) have been developed.

The outcomes of different sweet taste preference–related measures are often correlated (11), but it is not known whether this correlation is due to an underlying genetically determined preference for sweet taste or environmental factors. Our earlier family study showed that sweet taste preference–related traits were inherited, but we were unable to separate the effects of shared genes and family environment (3). In this study, our aims were 1) to test whether the variation shared by family members is due to genetic or shared environmental factors and 2) to examine whether the correlations between different sweet taste preference measures are due to genetic or environmental factors in a genetically informative sample of monozygotic and dizygotic female twins.

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SUBJECTS AND METHODS

Subjects

The subjects were recruited from the UK Adult Twin Registry (Twins UK) in 2005 (12). The registry consists predominantly of same-sex female twin pairs; thus, no males were included in this study. Only subjects who had both participated in the taste test and completed the questionnaires were included in the analyses, which yielded a sample of 663 females with a mean (±SD) age of 55.6 ± 12.4 (range: 17.3–80.7 y). This sample comprised 303 monozygotic (149 complete pairs) and 363 dizygotic (175 complete pairs) twins. Zygosity was determined by using the “Peas in the Pod” questionnaire (13, 14); if zygosity was still uncertain, it was checked by genotyping. The study was approved by Guy’s and St. Thomas’ Hospital Ethics Committee, and all participants gave informed consent. Subjects were recruited without selecting for a particular trait or disease. The average clinic visit lasted between 3 and 6 h, during which the subjects participated in several clinical tests.

Chemosensory test

In our previous family study (3), we determined the heritability of 3 suprathreshold sucrose concentrations: 3%, 7.5%, and 18.75% (wt:vol) sucrose in water. We found that the highest heritability (41% of variation) was obtained for the liking for the 18.75% solution. In the present study, we wanted to have a sweet taste preference test that would be easy and fast to prepare and to administer; therefore, only one very sweet solution was used. The sample was a 20% (wt:vol) sucrose solution that was prepared by pouring a 4-g prepackaged sugar sachet (Finnsugar, Kantvik, Finland) into a standard size plastic cup marked for 4 mL and 2 mL (Polarcup, Hämeenlinna, Finland). The cup was filled with water until the mark of 2 mL was reached, and the solution was stirred gently until the sugar had dissolved completely. The test administrator prepared the samples, which were stored overnight in the refrigerator (7 °C) and brought to room temperature before serving. Pilot testing indicated that the presentation of this intensely sweet stimulus in a single stimulus condition resulted in ratings similar to those given to the stimulus as part of the sample series used in the earlier study. The ratings for the 20% sucrose solution were not different from those of the 18.75% sucrose solution—a fact predicted by the Weber ratio (i.e., just noticeable difference from a reference concentration) of sucrose in water that varies between 0.08 and 0.20 (15).

Subjects visited the clinic after fasting overnight. The instructions for the taste test were given both orally and in written form, and the test administrator was present throughout the testing procedure. Before tasting, the subjects were not told that the solution was sweet. If there were more than one subject present at the time, they were told to refrain from communicating during the test. Subjects were requested to first rinse their mouths with water and then put the entire 20-mL solution into their mouths, to swirl it around for a short while (5–10 s), and to expectorate. They then rated the degree of liking or disliking and the intensity of the taste per a 120-mm vertical Labeled Affective Magnitude Scale (LAM; 16) and Labeled Magnitude Scale (LMS; 17), respectively. LAM and LMS are relatively new instruments, but they have been validated against more conventional scales (16–19). With an extreme stimulus, i.e., a very high sweetness, we wanted to use scales that allow ratings without the risk of the ceiling effect. Our pilot testing with 31 subjects indicated that the LAM and LMS resulted in better discrimination than did the conventional 9-point category scales. The verbal labels and their positions on the line from the bottom of the scale were as follows for the LAM: “the greatest imaginable dislike” (0 mm), “dislike extremely” (13 mm), “dislike very much” (26 mm), “dislike moderately” (39 mm), “dislike slightly” (54 mm), “neither like nor dislike” (60 mm), “like slightly” (66 mm), “like moderately” (81 mm), “like very much” (94 mm), “like extremely” (107 mm), and “the greatest imaginable like” (120 mm). In the LMS, the verbal labels were “barely detectable” (2 mm), “weak” (7 mm), “moderate” (20 mm), “strong” (42 mm), “very strong” (59 mm), and “the strongest imaginable sensation” (120 mm).

In addition, the intensity rating of a 6-n-propylthiouracil (PROP) filter paper (20) was included in the study as a positive control for heritability: the contribution of genetic factors on the variance of the intensity rating of PROP is known to be >50% (21). The preparation of PROP filter papers was made as described earlier (3). Subjects first tasted pure filter paper to be later able to distinguish the taste of PROP from that of paper. After rinsing their mouths, they set the PROP filter paper on their tongues for ~10 s. After waiting a short while (the strongest sensation of PROP intensity often comes with a delay), they rated the intensity using a similar 120-mm vertical LMS as for the intensity rating of the sucrose solution.

Questionnaire data

Before the clinic visit, the twins were sent postal questionnaires, which were completed at home and brought with them to the visit. The questionnaire included the ratings of like and dislike and use-frequency for 34 foods. The response alternatives for liking and disliking were 1 = dislike very much, 2 = dislike moderately, 3 = dislike slightly, 4 = neither like nor dislike, 5 = like slightly, 6 = like moderately, and 7 = like very much. For use-frequency, the ratings were 1 = never, 2 = a couple times of a year or more rarely, 3 = a couple times a month, 4 = a couple times a week, 5 = once a day, and 6 = several times a day. The foods were categorized by using factor analysis with maximum likelihood extraction and orthogonal Varimax rotation. A group of 6 sweet foods (sweet desserts, sweets, sweet pastry, ice cream, hard candy, and chocolate) was identified, and composite measures for liking and use-frequency of sweet foods were calculated as the mean of ratings given to 6 sweet foods items. Thus, the theoretical range was 1–7 for liking and 1–6 for use-frequency of sweet foods. The reliability of the scales was further studied by Cronbach’s α values; the values for liking and use-frequency of sweet foods were 0.84 and 0.71, respectively.

The questionnaire also included a Craving for Sweet Foods scale, which is a subscale of the Health and Taste Attitude Scales (9, 22). This validated scale measures the tendency to crave sweet foods with 6 statements, each evaluated according to a 7-point Likert scale (1 = strongly disagree, 2 = moderately disagree, 3 = slightly disagree, 4 = neither agree nor disagree, 5 = slightly agree, 6 = moderately agree, and 7 = strongly agree). The score for craving for sweet foods is calculated as the mean of ratings given to the 6 items; thus, the theoretical range was 1–7. The Cronbach’s α value for the scale was 0.70.

Quantitative genetic analysis

Classic twin modeling relies on the assumption that monozygotic twins are genetically identical, whereas dizygotic twins
share, on average, half of their segregating genes (23). 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). The epistatic effect, ie, interaction between alleles in different loci, is assumed to be absent. The correlations of both additive and nonadditive genetic effects are 1 within monozygotic pairs. Within dizygotic pairs, the correlations are 0.5 for additive and 0.25 for nonadditive genetic effects.

Environmental variation can be divided into environmental factors shared and unshared by cotwins. The shared environment, having a similar effect on monozygotic and dizygotic pairs, includes all environmental factors that make the twin pair similar for the trait, such as maternal nonheritable factors, shared childhood experiences, parental socioeconomic status, and the same friends. The 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 monozygotic and dizygotic twin pairs. Random mating with respect to the traits in question and the absence of gene-environment interactions is also assumed in the model. Assortative mating of parents may increase dizygotic correlations and thus inflate the estimates of shared environmental variance and reduce the genetic variance. The possible effect of gene-environment interaction is estimated as part of additive genetic component to the extent that the environmental factors interacting with the genes are shared within the pair, which thus may also reflect genetically based differences in susceptibility to environmental factors. To the extent that such environmental factors are not shared between twin pairs, the unique environmental component will absorb the effect (24).

On the basis of these assumptions, the phenotypic variance of a trait can be decomposed into additive genetic effects (A), dominant (nonadditive) genetic effects (D), shared (common) environmental effects (C), and unshared environmental effects (E). 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. The variance components explaining the total observed phenotypic variance can be calculated by squaring the path coefficients (regression coefficients) in the model. Because we had only twins reared together, but not adopted twins or other relatives in these data, we were unable to estimate shared environment (C) and dominant genetic (D) variance components simultaneously (24).

Genetic modeling was carried out with the Mx statistical package, version 1.7 (23). We first built univariate models estimating relative proportions of additive genetic (A), shared environmental (C), and unshared environmental effects (E) on the variation of each trait separately. The assumptions of the twin model were tested by comparing the chi-square change (Δχ²) between the twin model and the saturated model, which did not make any of the assumptions of the twin model. Relying on the final models and on the correlations between the phenotypes, we hypothesized which traits may have common underlying genetic or environmental effects and included these in a multivariate model. Cholesky decomposition was chosen as the general overall multivariate model. Cholesky decomposition assumes that specific genetic and environmental factors affect each phenotype, but these factors can also affect other phenotypes. Thus, we could study whether a correlation between phenotypes was due to shared genetic or environmental factors. Starting with the full model, we first tested whether all 3 variance components—A, C, and E—were necessary to explain the variance of and covariance between the traits. Second, we tested whether the specific variance components affecting a trait were unique to that trait or also affected the other traits.

The fit of the model was estimated by using chi-square goodness-of-fit statistics. If the change in chi-square values compared with the change in the df measured by a P value was >0.05 between 2 nested models, the more parsimonious model was assumed to provide a better fit to the data.

RESULTS

The mean ratings, SDs, and within-pair intraclass correlations of the traits for monozygotic and dizygotic twins are presented in Table 1. The differences in means and variances between monozygotic and dizygotic twins were negligible and were not statistically significant. Age did not significantly correlate with any of the phenotypes (Pearson’s r values between −0.16 and 0.09).

For most of the traits, the within-pair correlations of the monozygotic twins were higher than those of the dizygotic twins, which implies that genetic effects probably underlie the traits. Because the within-pair correlations of monozygotic twins were

<p>| TABLE 1 |</p>
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<thead>
<tr>
<th>Ratings and within-pair intraclass correlations of monozygotic and dizygotic pairs (n = 663)</th>
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<tr>
<td>Monozygotic twins (n = 303)</td>
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<tr>
<td>149 complete pairs</td>
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<tr>
<td>Variable</td>
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<tr>
<td>Liking for sweet solution</td>
</tr>
<tr>
<td>Intensity of sweet solution</td>
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<tr>
<td>Intensity of PROP†</td>
</tr>
<tr>
<td>Liking for sweet foods</td>
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<tr>
<td>Use-frequency of sweet foods</td>
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<tr>
<td>Craving for sweet foods</td>
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† Pearson correlation coefficient.

Significance level for the difference in correlation coefficients between the groups (Fisher Z transform): 1 P < 0.001, 2 P < 0.05.

PROP, 6-α-propylthiouracil.
less than twice those of the dizygotic correlations, the genetic effects were assumed to be additive and the ACE model was chosen as the starting point for quantitative genetic analysis. Pearson’s correlation coefficients between the phenotypes are shown in Table 2. Many significant correlations were observed between the phenotypes, which suggests that common factors underlie the traits. However, the intensity rating of the sweet solution and PROP were not strongly correlated with any of the other traits.

Quantitative genetic analysis

On the basis of the within-pair correlation patterns, an ACE model was chosen as the starting point of genetic modeling. A comparison of the fits of the twin models with the saturated model showed that the additive genetic and specific environmental (AE) model was found to fit all the variables (poorest change in fit for the use-frequency of sweet foods: $\Delta \chi^2 = 12, P = 0.11$), and a more complex additive genetic, shared environment, and specific environment (ACE) model did not offer a better fit, which suggests a lack of shared environmental effects. However, for 3 variables (the intensity rating of the sweet solution, intensity rating of PROP, and craving for sweet foods), the CE model provided an equally good fit. The intensity rating of PROP, here used as a positive control, was clearly under genetic influence. The proportional effects of the additive genetic and specific environmental components with their 95% CIs are presented in Figure 1.

In the multivariate modeling, we left out the intensity ratings of the sweet solution and the PROP filter paper because they did not correlate with the other measures. The multivariate model built was thus a tetravariate model. Because the variation of all the variables in the model could be explained by additive genetic and unshared environmental factors, we first tested whether the shared environmental factors could be excluded from the model. The removal of these variance components did not significantly worsen the fit of the model compared with the full model ($\Delta \chi^2 = 9, P = 0.44$); thus, we could only consider additive genetic and unshared environmental effects. The additive genetic and unshared environmental correlations that were very low (lower CI = 0) were then dropped from the model if their removal did not significantly worsen the model fit. The final model ($\Delta \chi^2 = 20, P = 0.14$ compared with the full model) calculated using the unstandardized variances of the variables is presented in Figure 2, and the additive genetic and unshared environmental correlations between the traits are shown in Table 3.

The correlation ($r = 0.23$) between liking for the sweet solution and liking for sweet foods was explained solely by genetic factors. The correlations between self-reported measures of sweet taste preference were, in turn, due to both genetic and unshared environmental factors.

DISCUSSION

Genetic and environmental effects on different sweet taste preference measures

Genetic effects clearly contributed to the variation in the sweet taste preference–related traits, ie, liking for sweet solution, liking for sweet foods, and use-frequency of sweet foods. Approximately half of the variation in the latter traits ($a^2 = 49\%, 54\%$, and $53\%$, respectively) was explained by additive genetic effects, and shared environmental effects did not contribute to the variation. In our earlier family study (3), we found similar heritability estimates for these traits, but were unable to separate the effects of shared genes and shared environment in the population consisting of families with no members reared apart. The present results thus provide evidence that the heritability of sweet taste preference–related traits is mediated by genetic effects and not by shared environment, eg, by a family’s common dietary habits.

Although liking for sweet taste appears to be partly inherited, the intensity perception of the sweetness is only weakly, if at all, inherited. The source of the within-pair correlation of the twins in the intensity ratings of the sweet solution ($r = 0.32$ for monozygotic and $r = 0.23$ for dizygotic) could not be determined. When the common variation was modeled to derive from shared genes (additive genetic effects), the heritability estimate was lower than that for the sweet taste preference–related traits, in line with the family study results (3). Although the liking for and the intensity rating of the sweet solution were weakly correlated ($r = -0.20$), this study further supports the view that different mechanisms underlie these perceptions.

Correlations among the measures of sweet taste preference

Liking for sweet solution and for sweet foods was measured with different methods and in different situations, the former by tasting an extremely sweet aqueous sucrose solution at the clinic and the latter by evaluating liking and disliking for listed food

<table>
<thead>
<tr>
<th></th>
<th>Liking for sweet solution</th>
<th>Intensity of sweet solution</th>
<th>Intensity of PROP</th>
<th>Liking for sweet foods</th>
<th>Use-frequency of sweet foods</th>
<th>Craving for sweet foods</th>
</tr>
</thead>
<tbody>
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<td>Liking for sweet solution</td>
<td>1</td>
<td>-0.20^2</td>
<td>0.05^3</td>
<td>1</td>
<td>0.40^2</td>
<td>0.40^2</td>
</tr>
<tr>
<td>Intensity of sweet solution</td>
<td>-0.20^2</td>
<td>1</td>
<td>0.05^3</td>
<td>1</td>
<td>0.40^2</td>
<td>0.40^2</td>
</tr>
<tr>
<td>Intensity of PROP</td>
<td>0.09^4</td>
<td>0.17^2</td>
<td>0.05</td>
<td>1</td>
<td>0.40^2</td>
<td>0.30^2</td>
</tr>
<tr>
<td>Liking for sweet foods</td>
<td>0.23^2</td>
<td>0.00</td>
<td>0.05</td>
<td>1</td>
<td>0.40^2</td>
<td>0.30^2</td>
</tr>
<tr>
<td>Use-frequency of sweet foods</td>
<td>0.13^2</td>
<td>0.03</td>
<td>0.06</td>
<td>1</td>
<td>0.40^2</td>
<td>0.30^2</td>
</tr>
<tr>
<td>Craving for sweet foods</td>
<td>0.06</td>
<td>0.05</td>
<td>0.05</td>
<td>1</td>
<td>0.40^2</td>
<td>0.30^2</td>
</tr>
</tbody>
</table>

^1 PROP, 6-O-propylthiouracil.

^2 P < 0.001.

^3 P < 0.05.
names at home. The significant additive genetic correlation ($r_A = 0.27, 95\% \text{ CI}: 0.15, 0.40$) shows that these traits are partly affected by the same genes. Although our study did not reveal which genes are involved, we assume that they are affecting the preference for sweet taste because liking for sweetness is the only obvious explanation for the correlation. Thus, this result suggests that the solution test at least partially reflects the underlying sweet taste preference. In addition, there are probably other factors (eg, cultural factors) that influence the affection for preferred sweetness in specific foods (5, 25).

The instruments used here appear to measure different aspects of sweet taste preference. Liking of the sweet aqueous solution did not correlate significantly with use-frequency or craving for sweet foods. Thus, instruments focusing on sweet preferences may be so different from each other that they do not measure the same issue. In addition, the predictive value of separate sweet taste preference measures on dietary intake has been shown to be limited (11); thus, multiple measures are probably needed to track the best predictor until the best predictors or the role of each instrument is clarified.

**FIGURE 1.** Proportion of the variation of traits explained by additive genetic and unshared environmental effects; 95\% CIs are shown in parentheses. The variation of the traits in 324 complete twin pairs (149 monozygotic and 175 dizygotic pairs; $n = 648$) was decomposed with the use of linear structural equation modeling.

**FIGURE 2.** Path diagram of the tetravariate Cholesky model for the variance in sweet taste preference–related traits. The unstandardized variance of 149 monozygotic and 175 dizygotic pairs ($n = 648$) was decomposed to additive genetic (A1–A4) and unshared environmental (E1–E4) effects. Each latent (unmeasured) variable, A or E, represents a set of genetic or environmental factors, respectively. An arrow pointing from any given latent variable to 2 traits (ie, observed variables) means that this set of genetic or environmental factors underlies both of these traits. If 2 variables do not correlate significantly, they are not influenced by any common latent variables. The variance and covariance components can be obtained by squaring the path coefficients.
Correlations among the questionnaire variables

Correlations among the traits related to self-reported sweet taste preference were all moderate and positive (r = 0.30–0.55, P < 0.001). The correlation among affective ratings and use of sweet foods is in line with earlier studies (6, 7, 26). The scores for craving for sweet foods were correlated with liking and use-frequency of sweet foods, which further validates the use of this scale. Earlier validation studies have shown that the craving scores are associated with pleasantness ratings of chocolate bars and soft drinks (9, 22).

The tetravariate Cholesky model showed that the correlations among these traits are due to both additive genetic and unshared environmental effects. The genetic effects explained, on average, 62% of the phenotypic correlation. Shared family environment does not appear to contribute to the covariance of these traits. In addition, 2 separate sets of genes and environmental factors influence the traits, whereas the craving for sweet foods is also affected by genes and environmental factors specific to this trait. Again, our study did not determine which genes or environmental factors are involved. The specific environmental factors, ie, factors making a twin pair dissimilar, may include individual differences in the scale usage or attitudes. The subjects may have misreported either consciously or unconsciously. This misreporting may have resulted from an avoidance of the use of the ends of a scale (27) or from reporting according to expected social desirability (28). Attitudes toward sweetness, eg, concerning the healthiness of sweet foods, have been shown to affect the liking and use-frequency of sweet foods (6).

Study limitations

As noted earlier, we decomposed the variation to genetic and environmental factors, but were not able to determine the underlying genes or environmental factors. The genetic factors affecting the traits remain to be identified and localized by gene-mapping experiments and the environmental factors by epidemiologic studies. Another limitation is that the data consist solely of females. Males may prefer higher sweetness (29) and may have more positive attitudes toward sugar (10). In addition, sex differences exist in the variance components of food use (30, 31). Thus, the results of this study may not allow extrapolation to males.

Correlations among the questionnaire variables

The classic twin design assumes 1) random mating with respect to the traits (in this case, mating of individuals regardless of their sweet taste preferences), 2) that the shared environment affects equally monozygotic and dizygotic pairs, and 3) the absence of gene-environment interactions. The first 2 can be assumed to be true in the case of sweet taste preferences, but gene-environment interactions may occur, which means that individuals with different genotypes respond differently to the environment. This might be expressed as the craving versus the avoidance of sweet foods in stress by individuals with different genetic makeup (32).

Conclusions

This is the first time that the covariance of different sweet taste preference–related measures has been separated between genetic and environmental factors. The multivariate modeling showed that some of the same genes underlie the liking for a sweet aqueous solution, measured by chemosensory test and the self-reported liking for sweet foods. Thus, an underlying genetic inclination to like sweetness exists and is expressed in both measures. The covariance among scores of the questionnaire phenotypes derives from both genetic and shared environmental factors. Two separate sets of genetic and environmental factors underlie liking for, use-frequency of, and craving for sweet foods. In addition, craving for sweet foods is affected by specific genetic and environmental factors. However, the shared family environment does not appear to influence the variance of separate measures of sweet taste preference or the covariance between them. This suggests that, in adults, liking for sweet foods inherited from the childhood family is mediated through genes rather than through the food habits of the family. However, approximately half of the variation is due to environmental factors and thus modifiable by dietary education.

These results broaden our understanding of the background of sweet taste preference. The sweetness preference, which may lead to adverse health effects through the excess use of sugar, derives from multiple separate genetic and environmental factors. Studies of the effect of high carbohydrate intakes on the development of obesity have produced controversial results; although several studies have shown that the consumption of sugar-sweetened drinks is associated with weight gain (for review, see 33), it has also been suggested that a high intake of

<table>
<thead>
<tr>
<th>Trait 1</th>
<th>Trait 2</th>
<th>Trait correlation</th>
<th>Additive genetic correlation(^2)</th>
<th>Specific environmental correlation(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liking for sweet solution</td>
<td>Liking for sweet foods</td>
<td>0.23</td>
<td>0.27 (0.15, 0.40)</td>
<td>100% (0)</td>
</tr>
<tr>
<td>Liking for sweet foods</td>
<td>Use-frequency of sweet foods</td>
<td>0.55</td>
<td>0.66 (0.54, 0.77)</td>
<td>72% (0.36, 0.48)</td>
</tr>
<tr>
<td>Liking for sweet foods</td>
<td>Craving for sweet foods</td>
<td>0.40</td>
<td>0.48 (0.31, 0.64)</td>
<td>63% (0.28, 0.40)</td>
</tr>
<tr>
<td>Use-frequency of sweet foods</td>
<td>Craving for sweet foods</td>
<td>0.30</td>
<td>0.31 (0.11, 0.50)</td>
<td>52% (0.27, 0.40)</td>
</tr>
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</table>

\(^1\) Phenotypic Pearson correlation coefficient.  
\(^2\) Additive genetic and specific environmental correlations obtained with the use of linear structural equation modeling.

TABLE 3
Correlations between additive genetic and unshared environmental factors explaining correlations between different sweet taste preference–related traits in a tetravariate Cholesky model for 149 monozygotic and 175 dizygotic complete twin pairs (n = 648)
sugar may be negatively associated with the indexes of obesity (34). The high use of sugar also increases the risk of dental caries and may increase blood insulin concentrations and the risk of diabetes (35).

We showed that commonly used instruments reflecting sweet taste preference may measure different hedonic or behavioral aspects. It is not clear which of the measures best reflect the high intake of refined sugar. The results encourage selection or development of a (set of) sweet taste preference measures that would reveal the most important aspects of the preference and could be universally used to study the effect of taste preferences on the excess use of sugar. Understanding the genetic elements underlying sweetness preference would help to cope with a problem that has major nutritional implications.

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