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Palatability and intake relationships in free-living humans: the influence of heredity

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Abstract

The heritability of the before and after meal self-ratings of palatability and their relationship to food intake was investigated with 86 identical and 78 fraternal same-sex and 51 fraternal mixed gender adult twin pairs who were paid to maintain 7-day food intake diaries. From the diary reports, the total and meal intakes of food energy and the amounts of the macronutrients ingested were estimated. Participants rated the meals for palatability on a 7-point (Bad - Good) scale both before and after eating. Linear structural modeling was applied to investigate the nature and degree of genetic and environmental influences and revealed significant genetic influences on subjective palatability both before and after the meals. In addition, the relationship between palatability and intake was influenced by the genes. Significant dominance genetic effects were found for the change in meal size between low-moderate and high palatability meals, indicating that palatability and it's relationship with intake are to some extent heritable. This suggests that the level of subjective experience and the individuals responses to them are influenced by the genes and become part of the total package of genetically determined physiological, socio/cultural, and psychological processes that regulate energy balance.

Keywords: Eating; Heredity; Twins; Hedonics; Carbohydrate; Protein; Fat; Meal Pattern

1. Introduction

It is becoming apparent that a wide range of correlates and influences of food and fluid intake are affected by heredity. Heredity is not only an influence on body size [4,5,6,25,35,

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39,42–44] but also on food and fluid intakes in a fashion that is independent of body size [8]. Heritabilities have also been shown for the microstructure of intake independent of the level of overall daily intake, including the number, timing, and composition of meals and drinks [7,9,45]. In addition, heritability has also been detected for very subtle influences on intake, including the levels of physiological and environmental variables that are present at meals and also the individuals' responsiveness to these stimuli [11,7,8]. Heredity appears to not only influence the degree of stomach filling that an individual tends to have before and after a meal but also the degree of restraint on the amount eaten in meals exerted by stomach filling [7]. These subtle influences of the genes on intake regulation appear to extend into the realm of social influences on eating. The genes have been shown to affect the overall number and type of eating companions that an individual chooses to eat with and also how responsive an individual is to social facilitation and how much their intake is increased by the presence of others [11].

Psychological/subjective influences on eating also appear to be affected by heredity. Significant genetic influences have been demonstrated on the levels of self-rated hunger both before and after the meals and the change in hunger over the meal. In addition, the relationship between hunger and intake was influenced by the genes, in that hunger's effect on intake and, in turn, intake's effects on hunger were found to be to some extent heritable [8]. This suggests that the level of subjective experience and the individuals responses to them are influenced by the genes.

This raises the question as to what other aspects of psychological/subjective influences on intake regulation might be affected by heredity. For the present study, the influences of the genes on the relationship between the perceived palatability of the food and the amount ingested was selected for study. Palatability is a hypothetical construct that stands for the stimulus qualities of a substance that affects its acceptability [28,36]. Palatability is influenced by both learned and innate factors [36] and it has been shown to have a potent effect on the desire to eat and the amounts ingested [3,21,26,40,37,46,47]. The self-reported level of palatability has been shown to have a positive relationship with the amount ingested in meals in free-living humans [16,17]. In the present study, whether and how the genes might influence the palatability-intake relationship was investigated by employing linear structural modeling techniques [23,24,34] to analyze the diet-diary reports of the eating and drinking behavior of adult twins [7–9,12–14].

2. Methods

2.1. Participants

The data were collected from 430 individuals consisting of 86 identical (40 male and 46 female pairs), 78 same-sex fraternal (46 male and 32 female pairs) and 51 mixed gender fraternal twin pairs. They were recruited through mailings to the members of twin associations and the Minnesota Twin Registry [32], through word of mouth, and through newspaper ads requesting volunteers for studies of nutrient intakes in twins. They were paid $30 to participate and also received a detailed nutritional analysis of their intake. The participants

averaged 41.9 ±10.2 (standard deviation) years, 72.9 ±15.5 kg and 1.69 ±.10 m and had an average body mass index of 25.3 ±4.2. In order to participate the twins could not be living together, actively dieting, pregnant or lactating, on chronic medication, or alcoholic.

2.2. Procedure

For a detailed review of the method, reliability, and validity of the diet-diary procedure see de Castro (1994; 1999c) [10,15]. The participants were given a small (8 × 18 cm) pocket sized diary and were instructed to record in as detailed a manner as possible every item that they either ate or drank, the time they ate it, the amount they consumed, how the food was prepared, and the number of other people eating with them. Self-ratings were obtained at the beginning and again at the end of the meal of the attractiveness of the food on a 7-point scale from 1 - Very Bad, 4 - Neutral, 7 - Very Good. The participants initially recorded this information for a day and were then contacted by the experimenter who reviewed the information, corrected any problems and answered any questions. The participants were then asked to record their intake for seven consecutive days. After this recording period the participants were again contacted by the experimenter who reviewed the diaries, clarifying any ambiguities or missing data. After the completed diaries were submitted, two individuals who ate with the participant during the recording period were contacted and asked to verify the participants reported intake. In some cases, difficulty was encountered in remembering exactly what the participant ate. However, in no case was the participant's diary report contradicted in either the nature or the amount [15].

2.3. Data Analysis

The foods reported in the diaries were assigned codes from a computer file of over 3500 food items, by an experienced registered dietitian. The coder was unaware of the experimental hypotheses and the participants' zygosity, and did not interact directly with the participants. Total daily intakes of food energy were calculated by summing the contributions of the individual items. Meals were identified and the compositions of the individual items composing the meal were summed. In order for a reported intake to be classified as an individual meal it had to contain at least 209 kilojoules (kJ) of food energy, or more stringently 418 kJ or 837kJ. It also had to be separated in time from the preceding and following ingestive behaviors by at least 15 minutes. More stringent definitions of 45 and 90 minutes were also employed. Five different definitions of a meal were used combining these minimum criteria, 15 min/209 kJ, 45 min/209 kJ, 45 min/418 kJ, 45 min/837 kJ, and 90 min/209 kJ.

To investigate genetic influences on palatability, heritability analysis was carried out on the before and after meal palatability ratings and the change in palatability over the meal; after meal rating minus the before meal rating. To assess genetic influences on the relationship of palatability with subsequent intake the average meal size for meals rated low-moderate in palatability (5 or less on the 7 point scale) and those rated high in palatability (6 and above on the 7-point scale) as well as their difference were calculated for each participant (see de Castro, Bellisle & Dalix, 1999 and de Castro, Bellisle, Dalix & Pearcey,

2000 for frequency distributions of these palatability ratings) [16,17]. These meal sizes and differences were also subjected to heritability analysis. Linear structural modeling was applied to the twin intake data according to the sex limitation model outlined by Heath et al., (1989; see also Neale & Cardon, 1992) [23,34]. Both the general and the scalar sex limitation models were fit to the data. However, in no case was there a significant advantage of the more complex general model over the simpler scalar model. Hence, only the results for the scalar model are reported. A series of path models were fit, using maximum likelihood estimation, to the MZ and DZ twin covariance matrices using the computer program LISREL-VII (Joreskog & Sorbom, 1989) [27]. This analysis yields an estimate of the effects of individual environment, additive genetic effects, and either common (familial) environment or dominance genetic effects. These are presented as standardized path coefficients which can be interpreted like correlations. The square of the path coefficient provides an estimate of the amount of the variance in the dependent variable that can be accounted for. It should be noted that individual environment factor contains all unexplained variance in the dependent measure. It, contains both the immediate environment effects, non-shared past environment effects, and error. Hence, if no other variable accounts for a statistically significant portion of the variance, then the immediate environment will capture 100% of the variance.

Common environment and dominance effects cannot be estimated simultaneously. Both models are assessed separately and the one that best fits the data as assessed with χ^2 is reported.

Prior to LISREL analysis the data were tested for univariate skewness and kurtosis [38] and for multivariate kurtosis [33]. If significant deviations from normality were detected, logarithmic transforms were performed to normalize the distribution.

3. Results

The various meal definitions did not produce significant qualitative differences in the pattern of results obtained. Hence, the analysis reported in the text are for the minimum 209 kJ, 45 min definition, which are presented as representative.

3.1. Self-rated before and after meal palatability

The heritability analysis results for the before meal palatability ratings is depicted on the left side of Figure 1.The means and heritability analysis of the before and after meal palatability ratings are presented in Table 1. The ratings are on a 7-point scale with 4 as a neutral rating. Before and after meal palatability is rated, on average, as good and there is very little change in the ratings over the meal, decreasing by only 3% of the full scale. The heritability analysis indicates that there are significant genetic contributions to both the before and after meal palatability ratings. These genetic effects account for 23% and 21% of the variance in the ratings respectively. In no case does common (familial) environment have a significant effect. Hence, it would appear that the genes influence the level of the perceived palatability of the meal.

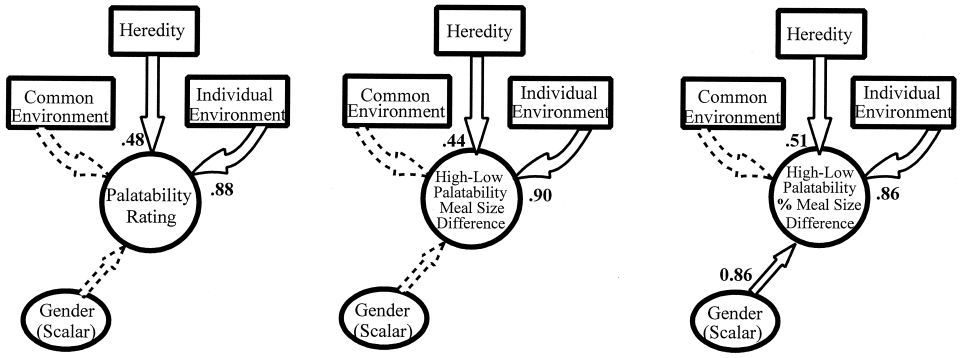

Fig. 1. The most parsimonious linear structural scalar sex limitation heritability models fitting the twin data for the before meal palatability ratings (left), the difference in meal sizes between high and low palatability meals (middle), and the percent difference in meal sizes, relative to the average meal size for the subject, between high and low palatability meals (right). The path coefficients for these estimated relationships are provided beside the arrows representing the paths. Dashed arrows represent non-significant paths. For all remaining parameters, removing any one leads to a statistically significant reduction in the model's account of the observations.

3.2. Palatability - meal size relationship

The correlations between the before meal palatability ratings and the size of the meal ingested are also presented in Table 1. These correlations, although statistically significant, are weak and only account for 4% to 5% of the variance. With such weak relationships it is

Table 1
Means ± standard error of the mean, intrapair correlations, and LISREL estimates for individual (Ind) and familial (Fam) environments, genetic (Gen) effects and the gender scalar for the ratings of meal palatability before and after meals and the correlations between palatability and meal size. Removal of any path coefficient presented in the table produces a significant degradation of the models fit as assessed with a χ^2 Test ($p < .05$). Blanks indicate non-significant paths. [d] Indicates that a dominance genetic estimate produces a best fit while no superscript indicates that the additive genetic estimate produces the best fit. Fit is the Goodness of Fit Index [48]. [‡] Indicates that the model does not significantly deviate from an adequate fit as assessed with a χ^2 Test.

	Means			Intrapair correlations			LISREL estimates standardized path coefficients				
	Twin pair types			Twin pair types			Environment			Gender	
	Ident	Fratl	♂♀	Ident	Fratl	♂♀	Ind	Fam	Gen	scalar	Fit
Palatability self-rating											
Before meal	5.11 ± .05	5.06 ± .06	5.21 ± .07	.191	.122	.054	.88		.48		.95[‡]
After meal	5.00 ± .06	4.81 ± .08	5.01 ± .10	.145	.140	.099	.89		.46	.92	.85
Change	−0.11 ± .05	−0.25 ± .07	−0.20 ± .09	.180	.024	.074	1.00				.78
Palatability correlations											
Meal size	.225 ± .020	.212 ± .022	.201 ± .020	.078	−.109	−.028	1.00				.96[‡]

Table 2
Means and LISREL estimates for individual (Ind) and familial (Fam) environments, genetic (Gen) effects, and gender for the meal sizes of low-moderate and high palatability meals and the difference between low-moderate and high palatability meals (See Table 1 legend).

	Means			LISREL estimates-path coefficients				
	Twin pair types			Environments			Gender	
	Ident	Fratl.	♂♀	Ind	Fam	Gen	scalar	Fit
Low palatability meals								
kJ	2043	2208	2005	.74		.68[d]	1.16	.95[‡]
Carbohydrate	1002	1054	992	.74		.68[d]	1.16	.98[‡]
Fat	715	795	695	.84		.54[d]	1.17	.99[‡]
Protein	326	359	318	.84		.54[d]	1.17	.99[‡]
Meal sizes								
kJ % average	90.4	92.2	89.5	1.00			.92	.94
Carb. % average	93.7	94.83	93.5	1.00				.83
Fat % average	87.3	88.7	86.3	1.00				.99
Prot. % average	87.6	90.9	86.8	1.00			.96	.69
High palatability meals								
kJ	2691	2840	2602	.87		.50[d]	1.14	.95
Carbohydrate	1188	1251	1159	.87		.49[d]		.90
Fat	1043	1095	983	.85		.52[d]	1.14	.78
Protein	460	494	460	.90		.44[d]	1.15	.78
Meal sizes								
kJ % average	120.5	117.3	117.8	.83		.55[d]	.83	.82
Carb. % average	111.9	111.9	109.4	.90		.44[d]	.76	.94
Fat % average	127.1	122.1	123.1	.85		.52[d]	.86	.79
Prot. % average	126.4	120.6	124.1	.85		.52[d]	.86	.79
High palatability								
kJ	648	632	597	.90		.44[d]		.83
Carbohydrate	186	197	167	.91		.41[d]		.97
Low palatability								
Fat	328	300	288	.89		.45[d]		.73
Protein	134	135	142	.88		.47[d]	1.08	.75
Meal difference								
kJ % average	30.6	26.3	29.8	.86		.51[d]	.86	.85
Carb. % average	18.4	17.2	17.0	.91		.42[d]	.81	.95
Fat % Average	40.4	35.5	38.7	.88		.48[d]	.89	.84[‡]
Prot. % average	39.8	31.1	39.6	.88		.48[d]	.89	.84

not surprising that the heritability analysis was unable to detect any significant effects. In order to provide a more sensitive measure to investigate palatability - intake relationships, the average size of the meals ingested that were rated high in palatability, those rated low to moderate in palatability, and the difference between the two were calculated and were analyzed for genetic and environmental influences. The results of this heritability analysis are depicted in the middle and right side of Figure 1, the more detailed and results are presented in Table 2.

Meals that were rated high in palatability were significantly larger than those rated low-moderate in palatability for all twin pair types (Table 2). This was true for overall meal size, and for carbohydrate, fat, and protein contents of the meals ($F(1,821) = 98.56$; 40.00; 93.60; 107.25; $p < .05$, respectively) and for the percent of the average meal size for overall

meal size, and for carbohydrate, fat, and protein contents of the meals ($F(1,821) = 195.61$; 92.65; 196.39; 183.69; $p < .05$, respectively).

Clear and significant dominance genetic influences were present for all of the absolute meal size measures for low-moderate and high palatability meals and their differences. These genetic influences accounted for between 17% to 46% of the variance. There were no significant influences of familial (shared) environment on any of these variables. When meal sizes were expressed for each participant as a percent of that participant's average overall meal size, significant genetic influences were apparent for high palatability meals and the difference between high and low-moderate palatability meals, accounting for between 17% and 30% of the variance. On the other hand, there were no significant genetic effects on the low-moderate palatability meals.

4. Discussion

An assumption of the heritability model used in the present analysis is that the identical twins and the fraternal twins had equivalent degrees of shared experiences. If, as has been postulated [18], identical twins have greater shared experiences than fraternal twins, then the model would produce a spurious effect and the variance due to the greater shared experiences would be apportioned to the genetic factor. To assess this we had the twins fill out an extensive questionnaire rating their degree of shared experiences during different phases of their lives. Surprisingly, the identical twins rated their shared experiences during childhood and adolescence as less similar than the fraternal twins. In addition, the shared experience ratings did not significantly correlate with either the intrapair differences in palatability, meal sizes, or high vs. low palatability meal size differences. This suggests that the alternative explanation is not correct and that the obtained heritabilities are not spurious.

The results of the present study indicate that inheritance affects the magnitude of the palatability ratings at both the beginning and end of a meal. These heritabilities were for average palatability ratings over all meals. In other words, identical twins tended to have a greater similarity in their overall levels of rated palatability than fraternal twins. This could reflect an inheritance of a similar appreciation of foods, or a similarity in taste sensitivity, or a similar labeling of palatability, or a similarity in the types of foods that tend to be chosen, or even a similarity in reactivity to the hedonic properties of foods. For the most part the present data do not contain any clues as to the likelihood of these various explanations, with the exception of the labeling explanation. Labeling is a learned behavior. The individual must learn how to assign various labels of palatability to their subjective responses to food. Since these are learned, the childhood (familial) environment would be expected to exert a major influence. However, this was not the case. There were no significant effect for familial environment in any of the analysis. Therefore, it would appear to be unlikely that the calculated inheritances were due to similarities in labeling.

The present results indicate that there are significant influences of heredity on the sizes of meals rated as low-moderate in palatability and also as high in palatability. However, these findings may simply result from the fact that the average size of meals is affected by the genes [7]. Expressing the meal sizes in terms of the percentage of each participant's overall

average meal size corrects for overall meal size. This resulted in a loss of significant heritability for the low-moderate palatability meals, suggesting that these calculated heritabilities were secondary to the effect of inheritance on overall meal size. On the other hand, the fact that significant heritability was present for the high palatability meals, even when expressed as a percent of the average meal size, suggests that the influence of the genes is on the effect of high palatability on meal size.

The relationship of palatability with intake can also be assessed by calculating the differences between the meal sizes that were rated low-moderate in palatability and those rated high in palatability. This provides a measure of the responsiveness of the individual to the perceived palatability of the meal and it was found to be heritable. Since the magnitude of the absolute differences is influenced by the overall level of intake, this heritability could be secondary to a heritability of overall intake levels. However, the fact that significant heritabilities were still present when the difference were adjusted for the average meal size suggests that the heritability of palatability responsiveness is independent of overall intake level. In addition, inaccuracy or bias in the labeling of palatability is unlikely to be responsible for the relationships. Since the proportionate differences are magnitude independent and calculated individually for each participant, individual differences in labeling could not affect the significant relationship of palatability with intake. Hence, it would appear that the relationship of palatability with intake is heritable.

A self-report methodology was used in the present study. Even though the diet-diary technique appears to be reliable and valid [1,19,22,29,41] (see de Castro; 1994; 1999c for review) [10,15], it is not without error. In particular it appears to underestimate intake [2,20,30,31]. However, underestimation is a constant influence on the magnitude of estimates of intake and the present analyses have suggested that the findings are magnitude independent. Hence, recording errors could not be responsible for the observed palatability - intake relationships or their heritability. There still are probably unsystematic, random, errors of measurement. But, these should obfuscate significant relationships not produce them. The fact that significant relationships and heritabilities were found with a technique that includes considerable error suggests that the effects reported may actually be underestimated and that the influence of the genes are, in fact, considerable stronger than indicated by the reported results.

The heritabilities reported presently account for 17% to 46% of the variance. However, these calculations may underestimate the importance of heredity. In the present study, the heritability of palatability influences on intake accounts for about 25% of the variance. But, the perceived level of palatability was also found to be affected by heredity. Hence, both the magnitude of the perceived palatability and individual's responsiveness to that palatability are heritable. Thus, a calculation based upon a single factor does not capture the actual magnitude of the influence of heredity on the palatability-intake relationship.

It was demonstrated previously that body weight, height, overall daily intake, independent of body size, and meal intakes, independent of overall daily intake, show significant heritability [7–9,12,45]. The genes have also been found to affect social facilitation of food intake [11], the effects of the stomach contents on intake [13], and subjective hunger influences on intake [14]. The present results, taken together with these prior findings, suggests that food intake regulation occurs as the result of the operation of a multilevel

system, each component of which is to some extent influenced by inheritance. The genes affect the determination of body structure and size, and the level of and responsiveness to internal variables, including subjective signals. In addition, the genes appear to affect the responsiveness to external signals and what kinds of external environments the individuals will select. The genes then strongly influence the individual's nutrient intakes and body weight. But, not by dictating the level of single factors. Rather, inheritance would appear to produce its effects as the integral of the combination of its influences on a large number of diverse factors.

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