Genes Nutr (2013) 8:241–252
DOI 10.1007/s12263-012-0320-8

RESEARCH PAPER

Genetic and environmental influences on nutrient intake

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Received: 3 April 2012 / Accepted: 6 September 2012 / Published online: 2 October 2012

Abstract The relationship between genetic and the environment represents a pathway to better understand individual variations in nutrition intake and food preferences. However, the present literature is weakened somewhat by methodological flaws (e.g., overreliance on self-report questionnaires), discrepancies in statistical approaches, and inconsistent findings. Little research on this topic to date has included examination of micronutrient intake. The purpose of this study is to improve the existing literature on genetic and environmental influences on energy and nutrient intake by addressing these gaps. Twin pairs ($N = 358$; age 11–13 years) provided 3-day food intake diaries, which were assessed for intake of total energy, macronutrients, and micronutrients. Structural equation modeling revealed that genetic influences accounted for a significant portion of the total variance in total energy (48 %), macronutrients (35–45 %), minerals (45 %), and vitamins (21 %). Consistent with previous studies, the shared environment appeared to contribute little to nutritional intake. Findings on vitamin and mineral intake are novel and are particularly beneficial for further research on the contribution of micronutrients to individual physical health status. Better understanding of the linkage between genes, environment, and nutritional intake and deficiencies can clarify behavioral and physical outcomes, potentially informing risk reduction, primary prevention, and intervention strategies.

Keywords Twin · Genes · Nutrient · Diet · Heritability · Environment

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Introduction

A growing body of twin studies has examined the role of genetic and environmental influences on individual differences in food preferences and intake. Evidence for heritable influences on food consumption (Breen et al. 2006; Keskitalo et al. 2008; Hasselbalch et al. 2008; van den Bree et al. 1999) and energy and macronutrient intake (de Castro 1993; Faith et al. 1999; Hasselbalch et al. 2008; Hur et al. 1998) is compelling. The magnitude of genetic effects is heterogeneous among studies; they generally explain about 20–40 % of the variance in energy and macronutrient intake (Rankinen and Bouchard 2006). For the most part, the remainder of variance is attributed to non-shared environmental influences, with the shared (familial) environment reportedly contributing minimal, if any, effects (de Castro 1993; Hasselbalch et al. 2008; Heller et al. 1988; Hur et al. 1998; Wade et al. 1981). An exception is reported by Faith et al. (1999), who measured ad libitum food intake under controlled conditions using buffet-style lunches during laboratory sessions. Genetic, shared environmental, and non-shared environmental factors were reported to have an influence on total caloric intake of 33, 48, and 19 %, respectively. Because these findings were based on single meals of limited food choice, they may be poor representations of overall energy and nutrient intake and instead reflect food intake phenotypes/behaviors.

Indeed, in children aged 4–5 years, strong shared environmental influences on preference and use-frequency has been reported for desserts (62 %), vegetables (46 %), and fruits (32 %), while non-shared environmental effects are low across all food groups (10–18 %) (Breen et al. 2006). This significance of the shared environment most likely reflects the limited exposure children have to important food encounters away from home at such a young age, since similar studies in adults aged 22–27 (Keskitalo et al. 2008) and over 50 (van den Bree et al. 1999) suggest a disappearance of this shared environmental effect after childhood. Indeed, Faith et al. (2008) have more recently supported the notion that the shared family environment can influence short-term eating patterns, and another recent study found that the shared environment can strongly influence the consumption of certain food groups, such as fruits and vegetables (40–46 %), without having any influence on total daily energy or macronutrient intake (Hasselbalch et al. 2008).

Hasselbalch et al. (2008) have recently provided convincing evidence, however, that some of these early family environmental effects in specific food groups such as fruits and vegetables (40–46 %) may remain throughout an individual's lifetime.

Several studies have also reported that heritability for protein, fat, and carbohydrates generally does not differ substantially from overall caloric intake. Rather than suggest that genetic influences act non-selectively on macronutrient consumption, this trend may reflect an overlap in genetic factors that account for specific macronutrients and overall energy requirements (Hur et al. 1998). Research in the field of nutrigenomics and nutrigenetics has already begun to expose important, bidirectional relationship between nutrients and genetics, such as a specific polymorphism effect on individual macronutrient intake (Timpson et al. 2008; Qi et al. 2008) and the consequences of single-vitamin deficiencies on DNA replication and long-term health, as in, for example, the study performed by Crott and others (Crott et al. 2001).

There is still, however, considerable variability among studies regarding the magnitude of genetic influence on energy and macronutrient intake. One reason may stem from the methodological differences used in the current literature. Most of the existing literature on food consumption and intake is based on self-report questionnaires about general food preferences and eating behaviors, which may introduce subject error or bias (Hebert et al. 1995). Food frequency questionnaires (FFQ) may also poorly represent actual overall nutrient intake, since a limited number of possible responses, use of broadly generalized food categories, and use of specific questions can lead to both under- and overestimation of nutrient intake (Cade et al. 2002). Of the six notable studies on energy and macronutrient intake summarized in Table 1, two studies have relied on FFQs (Hasselbalch et al. 2008; Hur et al. 1998). Faith and colleagues (Faith et al. 1999) utilized a laboratory setting, which can provide more accurate data collection under controlled conditions but can also complicate and prevent separation of genetic and shared environmental effects. Indeed, although significant measurement-specific genetic and shared environmental effects in their study were not found, these effects could not be dropped from the model, suggesting that familial (genetic and shared environment) influences are important for macronutrient intake.

Self-report food intake diaries may offer a more reliable and effective way to study nutrient intake (Block 1982). To our knowledge, this method has only been used in three early twin studies (Wade et al. 1981; Heller et al. 1988; de Castro 1993), although even these provided inconsistent findings. Heller et al. (Heller et al. 1988) reported that genetic influence accounted for 55 % of the total variance in complex carbohydrates, and from 3 to 31 % in macronutrients. Wade et al. (Wade et al. 1981) found that genetic influences accounted for 66 % of the variance in absolute intake of carbohydrates, 70 % in concentration of proteins, and 67 % in carbohydrates. However, the genetic effect was insignificant for absolute intake of energy (11 %), protein (20 %), and other macronutrients. Finally, de Castro (1993) reported strong heritability (65 % for energy intake) in both total caloric (65 %) and macronutrient consumption (50–61 %).

Three-day and seven-day nutritional records have yielded high (>0.9) correlations and intraclass correlations (0.74–0.91) between measures (Tremblay et al. 1983), and thus, time difference alone should not account for the discrepancies in these three studies' findings. A more likely explanation may be differences in sample characteristics and statistical approaches. For example, Wade et al. (Wade et al. 1981) included a very small sample (13 monozygotic and 10 dizygotic pairs) of only female twins, compared to the larger, mixed-sex samples in Heller et al. (Heller et al. 1988) (106 monozygotic and 94 like-sex dizygotic twin pairs) and de Castro et al. (1993) (220 individuals consisting of 53 male and 57 female pairs of identical twins). Both Wade et al. and Heller et al. used simple calculation approaches to estimate the heritability, rather than more modern model fitting methods. Wade et al. used the Holzingers' H equation [(Variance DZ−Variance MZ)/Variance DZ], and Heller et al. used the Falconer equation [2(rMZ−rDZ)], and the heritability was also estimated as the ratio of the genetic variance to the total variance. As such, neither of the two studies reported shared or non-shared environmental estimates. Only de Castro et al. used a more sophisticated approach of linear structural modeling, which provides constrained estimates of both genetic

Table 1 A summary of previous studies examining genetic and environmental influences on energy and macronutrient

Author	Sample	Age	Method	Gender	Energy intake			Macronutrient intake								
								Carbohydrate			Fat			Protein		
					A	C(D)	E	A	C(D)	E	A	C(D)	E	A	C(D)	E
Hasselbalch et al. (2008)	$n = 1{,}212$ Denmark	18–67 $\bar{x} = 38$	FFQ	Males	0.38	–	0.62	0.36[a]	–	0.64	0.01 ns	(0.36)	0.63	0.28[a]	–	0.72
				Females	0.32	–	0.68	0.49[a]	–	0.51	0.01 ns	(0.43)	0.56	0.01 ns	(0.55[a])	0.44
Hur et al. (1998)	$n = 335$ USA/UK	18–77 $\bar{x} = 42.4$	FFQ	Male + female	0.32	–	0.68	0.25	–	0.75	0.35	–	0.65	0.16 ns	–	0.84
								0.24 ns SC	–	0.76	0.37 Sat	–	0.63			
								0.18 ns CC	–	0.82	0.46 Poly	–	0.54			
Wade et al. (1981)[d]	$n = 46$ Canada	19–58 $\bar{x} = 39.2$	3-day food diary	Female	0.11 ns			0.66			–[b]			0.20		
								0.67[a]			0.48[a]			0.70[a]		
Heller et al. (1988)[d]	$n = 400$ Australia	17–66 $\bar{x} = 36$	4-day food diary	Male + female	0.38 ns (0.27)			0.31 ns (0.36)			0.24 ns (0.16)			0.08 ns (−0.13)		
								0.20 SC			0.10 SF					
								0.55 CC			0.33 MF					
											0.03 PF					
de Castro et al. (1993)	$n = 390$ USA	$\bar{x} = 38.8$	7-day food diary	Male + female	0.65	–	0.35	0.61	–	0.39	0.57	–	0.43	0.50	–	0.50
Faith et al. (1999)	$n = 108$ USA	>18	2 buffet lunches in lab session	Male + female	0.33	0.48[c] ns	0.19[c]									

Macronutrient intakes are reported as absolute daily intake, unless denoted with "a" for calculation as concentration

A additive genetic effect, *D* non-additive genetic effect, *C* shared environment effect, *E* non-shared environment effect, *FFQ* food frequency questionnaire, *SC* simple carbohydrate, *CC* complex carbohydrate, *SF* saturated fat, *MF* monounsaturated fat, *PF* polyunsaturated fat, *ns* nonsignificant

[b] Heritability of fat intake in grams was not calculated because MZ within-pair mean square exceeded DZ value

[c] Daily caloric intake only

[d] Heritability estimates were based on either Holzingers' *H* equation [(Variance DZ−Variance MZ)/Variance DZ] or Falconer equation [2(rMZ−rDZ)]; C and E estimates were not provided. Heller et al. also estimated the heritability as the ratio of the genetic variance to the total variance; estimates are presented in the parentheses

and environmental effects as well as their standard errors (de Castro 1993). It is noteworthy also that none of these prior twin studies examined sex differences in the relative magnitude of genetic and environmental components in nutrient intake.

The mixture of findings in past research underscores the importance of elucidating the relationship between genes, environment, and nutritional preferences. The purpose of this study is help clarify potential genetic and environmental influences on energy and nutrient intake in an effort to understand outcomes related to individual differences in food consumption, such as disease and overall health status. Further, in addressing methodological gaps in the literature, this twin study will utilize reliable measures and a varied sample size that is much larger than in the previous food intake studies, which have collected data by food diaries instead of FFQ. The present study also examines a greater range of micronutrients (i.e., vitamins) than those examined by previous researchers, which may ultimately lead to better understanding of specific interactions among micronutrients and genetic and cell processes (Farhud and Yeganeh 2010).

Methods

Participants

The subjects were participants in the University of Southern California (USC) Twin Study, which is a prospective, longitudinal study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial behavior from childhood to young adulthood. The participants were recruited from the greater Los Angeles area, and both the ethnic and socioeconomic status composition of the sample were representative of Southern California. To be eligible to participate in the study, twins had to be fluent in English and caregivers had to be fluent in either English or Spanish. To date, four waves of data have been collected. During Wave I (2001–2004) the twins were 9–10 years old (mean age = 9.60, SD = 0.59); during Wave II (2003–2006) the twins were 11–13 years old (mean age = 11.79, SD = 0.92); during Wave III (2006–2010) the twins were 14–16 years old (mean age = 14.87, SD = 0.87); and during Wave 4 (2008-2011) the twins were 17–18 years old (mean age = 17.28, SD = 0.77). The total study sample includes 1,562 subjects, including 169 monozygotic (MZ) male, 171 MZ female, 121 dizygotic (DZ) male, 120 DZ female, and 200 DZ opposite-sex twin pairs (Baker et al. 2006). The present study used data from Wave 2, as information on nutrients was not collected during Wave 1. Data on nutrition was collected from 358 of the participants. Lower number of participants during the second wave of assessment is due to the inclusion of 3-day nutrition diary only in the laboratory protocol, and not in the alternative mailed packet of surveys that many families chose to complete during this wave of assessment. Table 2 provides detailed information on number of participants broken down by sex and zygosity.

Zygosity was based on DNA microsatellite analysis [>7 concordant and zero discordant markers = monozygotic (MZ); one or more discordant markers = dizygotic (DZ)] for 87 % of the same-sex twin pairs. For the remaining

Table 2 Means, standard deviations, and number of participants (*n*) for energy and micronutrients, ages 11–13 years, by sex and zygosity

	Males [Means (SD)]		Females [Means (SD)]		DZ opposite sex [Means (SD)]	
	MZ	DZ	MZ	DZ	Males	Females
Protein	4.70 (3.21)	4.82 (4.81)	5.85 (9.93)	4.97 (5.73)	5.97 (5.32)	4.02 (2.20)
	$n = 93$	$n = 46$	$n = 95$	$n = 52$	$n = 35$	$n = 37$
Carbohydrate	50.94 (32.15)	60.85 (72.44)	52.20 (33.10)	55.65 (83.85)	59.32 (81.16)	47.43 (25.47)
	$n = 93$	$n = 46$	$n = 95$	$n = 52$	$n = 35$	$n = 37$
Mineral	448.37 (245.44)	461.49 (253.80)	470.49 (393.98)	439.71 (295.22)	483.68 (202.25)	423.25 (247.53)
	$n = 93$	$n = 46$	$n = 95$	$n = 52$	$n = 35$	$n = 37$
Vitamin	252.21 (208.07)	284.37 (214.31)	271.58 (424.12)	239.41 (215.15)	255.84 (152.47)	206.50 (196.44)
	$n = 93$	$n = 46$	$n = 95$	$n = 52$	$n = 35$	$n = 37$
Energy	2,258.14 (1,531.39)	2,487.82 (2,308.61)	2,283.75 (2,111.44)	2,325.84 (3,246.32)	2,594.79 (2,934.65)	2,122.75 (1,343.40)
	$n = 93$	$n = 46$	$n = 95$	$n = 52$	$n = 35$	$n = 37$

Fat, proteins, and carbohydrates were measured in grams. Energy was measured in kilocalories. Minerals and vitamins were measured in mg, mcg, and IU

MZ monozygotic, *DZ* dizygotic

same-sex twin pairs, zygosity was established by questionnaire items about the twins' physical similarity and the frequency with which people confuse them. For these remaining same-sex twins, the Lykken Twin Similarity Questionnaire (TSQ) was used to assess zygosity. The TSQ has proven to correctly identify MZ twins with almost complete certainty (Lichtenstein et al. 2002). The questionnaire was used only when DNA samples were insufficient for one or both twins. When both questionnaire and DNA results were available, there was a 90 % agreement between the two. Complete details on the procedures and measures can be found elsewhere (Baker et al. 2006).

Dietary assessment

Three-day food diary intake was assessed by 24-h recall on three different days (2 weekdays and 1 weekend day) by trained research assistants. The first 24-h recall was administered during a laboratory interview to familiarize the child with the procedures for food intakes recall, while other 2-day, 24-h recalls were collected by mail and followed by telephone interview. Respondents were instructed to recall and describe all the foods and beverages consumed at home and outside of the home over the past 24 h. Not all subjects were able to complete 3-day food diary. Our data indicate that about 61 % MZ subjects and 53 % DZ subjects completed 1 day, approximately 30 % MZ and 32 % DZ completed 2 days, and only about 9 % MZ and 14 % DZ completed 3 days. There was no statistical difference between MZ and DZ twins in terms of days completed. Among those collected, 80.7 % food diaries were obtained from weekdays, while 19.3 % were obtained from weekends. These diet records were entered into Food Processor Plus™ (ESHA Research, Inc., Salem, OR, USA) by the same research assistant who interviewed on the phone and carefully checked for errors by a trained registered nurse. This nutritional analysis software yielded estimates of various micronutrients and macronutrients, which were used in turn to create summary variables used in genetic analyses here. Each nutrient indicated in the following paragraph was derived from Food Processor Plus and was based on the average of food records available for the analysis. Variables were created to represent daily energy intake, fat, protein, carbohydrates, total energy, minerals, and vitamins, as described below.

Fat was calculated by summing the mean grams of fatty acids, including various omega-9 fatty acids, omega-3 fatty acids, omega-6 fatty acids, trans fatty acids, and cholesterol. *Protein* consisted of 20 common nonessential and essential amino acids, such as alanine, glycine, histidine, leucine, proline, serine, and tryptophan, in addition to manganese. *Carbohydrate* included dietary fiber, disaccharides, monosaccharides, starch, soluble fiber, alcohol, and sugar alcohols. *Mineral* intake consisted of macrominerals such as calcium and chloride, trace minerals such as boron and fluoride, and organic acids such as acetic acid, malic acid, chloride, citric acid, iodine, iron, sodium, potassium, and zinc. The *vitamin* category was composed of 30 micronutrients that included vitamins A, B, C, D, E, and K, as well as carotenoids such as lutein and alpha and beta carotenes. Finally, *Energy* included total calories, calories from fat, calories from saturated fat, and calories from trans fat. A complete and detailed list of the nutrients included in each group for analysis can be found from the Food Processor Plus™ software (ESHA Research, Inc., Salem, OR, USA).

Statistical analyses

Descriptive statistics and twin correlations

Descriptive statistics, including means and standard deviations, were first computed for nutrients. These statistics are outlined in Table 2.

The classical twin design is a natural experiment that relies on the different levels of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twins to estimate the relative contribution of genetic and environmental factors to individual differences in a phenotype of interest, in this case nutrient intake. The total phenotypic variance of a measured trait can be divided into additive genetic factors (A), shared environmental factors (C), and non-shared environmental factors (E). Since MZ twins are assumed to be genetically identical, additive genetic factors are correlated 1.0. For DZ twins the genetic factors are correlated 0.5 as they are assumed to on average share 50 % of their segregating genes. Shared environmental factors refer to non-genetic influences that contribute to similarity within pairs of twins (e.g., growing up in the same house hold). Shared environmental influences are assumed to contribute equally to similarity in MZ and DZ twins, and thus shared environmental factors correlate 1.0 in both MZ and DZ twins. Non-shared environmental factors are those experiences that make siblings dissimilar (e.g., different peer influences). There is no correlation for the unique environment by definition, and this parameter also includes measurement error. Heritability is the proportion of total phenotypic variance due to genetic variation. To obtain a first indication of the underlying sources of variance in nutrients, comparisons were made among twin correlations (Twin-1–Twin-2 correlations). For example, a DZ intraclass correlation approximately half the value of the MZ intraclass correlation would indicate the presence of additive genetic effects, whereas a DZ intraclass correlation more than half an MZ intraclass correlation indicates the presence of both genetic and shared environmental effects. However, this is a descriptive approach which does not specifically identify latent factors underlying covariance across

measures. Thus, formal genetic modeling is necessary to test the accuracy of the inferences made from these observations (Neale 1992).

Biometric analyses

Univariate models were fit to estimate the relative contributions of additive genetic factors (A), shared environmental factors (C), and non-shared environmental factors (E), to nutrients. To test for sex differences in the variance components, a model in which the genetic and environmental effects were allowed to differ between males and females was compared against a model in which the estimates were constrained to be equal. A saturated model, which estimates the variances, covariances, and means of nutrients, was first fit and used as a baseline model to which subsequent models were compared.

Models were fit with the structural equation program Mx (Neale et al. 2003), using a maximum likelihood estimation procedure for raw data. Raw maximum likelihood yields a goodness of fit index called log-likelihood. The adequacy of fit is assessed by computing twice the difference between the log-likelihood of a full model and that of a submodel, in which parameters are fixed to be zero or constrained to be equal. This difference follows a χ^2 distribution with the difference in the number of estimated parameters in the two models as the degrees of freedom. A significant χ^2 indicates that the model with fewer parameters to be estimated fits the data worse. The suitability of the models was also determined by comparing the model's Akaike's information criterion (AIC). The AIC represents the balance between model fit and the number of parameters (parsimony), with lower values indicating the most suitable model (Akaike 1987). The last model-selection statistic was the Bayesian information criterion (BIC), where increasingly negative values correspond to increasingly better fitting models (Raftery 1995).

Results

Table 2 presents the number of participants broken down by zygosity (in total there were 188 MZ twins and 170 DZ twin), means, and standard deviations for the raw (untransformed) nutrient variables along with the number of participants in the present study. No mean or variance differences were found between MZ and DZ twins for any of the variables (fat $t_{(355)} = 0.13, p = 0.90$; $F_{(186,\ 169)} = 1.19, p = 0.26$; protein $t_{(355)} = 0.07,\ p = 0.95$; $F_{(186,\ 169)} = 1.0,\ p = 0.77$; carbohydrate $t_{(355)} = 0.62, p = 0.54$; $F_{(186,\ 169)} = 1.08, p = 0.62$; mineral $t_{(355)} = -0.10,\ p = 0.92$; $F_{(186,\ 169)} = 1.12,\ p = 0.45$; vitamin $t_{(355)} = -0.73,\ p = 0.46$; $F_{(186,\ 169)} = 1.25, p = 0.14$; energy $t_{(355)} = 0.24, p = 0.81$; $F_{(186,\ 169)} = 1.03,\ p = 0.84$). There were no mean differences between males and females (fat $t_{(356)} = 0.84,\ p = 0.40$; protein $t_{(356)} = -0.52, p = 0.51$; carbohydrate $t_{(356)} = 0.54,\ p = 0.59$; mineral $t_{(356)} = 0.36,\ p = 0.71$; vitamin $t_{(356)} = 0.36, p = 0.72$; energy $t_{(356)} = 0.55, p = 0.58$).

Twin correlations

Intraclass twin correlations for the nutrient variables are presented in Table 3. The MZ intraclass correlations were higher than DZ correlations, suggesting genetic influences for all nutrients. All MZ intraclass correlations were less than one, which suggests influence of non-shared environment. The DZ twin correlations were nonsignificant, which is probably due to low power.

Univariate model fitting results are presented in Table 4. A full ACE model provided a better fit of the data for each variable based on BIC and AIC criteria (e.g., *fat* Model # 2, AIC = 286.64; BIC = −420.24) and did not significantly differ from the saturated model (e.g., *fat* Model # 2, $\chi^2 = 6.99$; $df = 10$; $p = 0.73$). No significant sex differences were seen in the ACE estimates for any of the variables, since male and female components could be constrained to be equal (e.g., *fat* Model # 3, AIC = 285.47; BIC = −426.58). The model could be further reduced for each variable by dropping the shared environmental component (C) (e.g., fat, Model # 4, AIC = 281.63, BIC = −429.19), except for *vitamin*. For *vitamin* it was difficult to distinguish between an AC and CE model based on the AIC and BIC criteria. Therefore, a full ACE model is presented (Model # 3).

Table 4 also displays the estimated variance components. Genetic influences (A) accounted for 44 % of the

Table 3 Intraclass correlations for energy and micronutrients at ages 11–13

	MZ male	DZ male	MZ female	DZ female	DZ−OS
Fat	0.46*	0.19	0.53*	0.12	0.09
Protein	0.29*	0.12	0.37*	0.16	−0.04
Carbohydrate	0.36	0.16	0.55*	0.33*	0.14
Mineral	0.39*	0.11	0.61*	0.01	0.15
Vitamin	0.35*	0.18	0.18	0.10	0.06
Energy	0.45*	0.06	0.60*	0.08	0.08

MZ monozygotic, *DZ* dizygotic, *DZ−OS* dizygotic opposite sex, * $p < 0.05$

Table 4 Univariate model fitting results and parameter estimates for energy and micronutrients at ages 11–13

	Overall fit							Parameter estimates (95 % CI)		
	−2LL	*df*	AIC	BIC	χ^2	*df*	*p*	A	C	E
Fat										
Saturated	979.654	340	299.654	−397.633						
ACE (males ≠ females)	986.641	350	286.641	−420.242	6.987	10	0.727			
ACE (males = females)	989.632	353	285.474	−426.577	9.978	13	0.696			
AE (males = females)	989.632	354	281.632	−429.187	9.978	14	0.764	0.44 (0.28–0.58)		0.56 (0.42–0.72)
CE (males = females)	996.080	354	288.080	−425.963	16.426	14	0.288			
E (males = females)	1,013.122	355	303.122	−420.052	33.468	15	<0.001			
Protein										
Saturated	991.609	340	311.609	−391.656						
ACE (males ≠ females)	997.572	350	297.572	−414.776	5.963	10	0.818			
ACE (males = females)	998.930	353	292.930	−421.928	7.321	13	0.885			
AE (males = females)	998.930	354	290.930	−424.538	7.321	14	0.922	0.31 (0.13–0.47)		0.69 (0.53–0.88)
CE (males = females)	1,001.701	354	293.701	−423.153	10.092	14	0.755			
E (males = females)	1,009.342	355	299.342	−421.942	17.733	15	0.277			
Carbohydrate										
Saturated	982.500	340	302.500	−396.211						
ACE (males ≠ females)	986.252	350	286.252	−420.436	3.752	10	0.958			
ACE (males = females)	993.391	353	287.391	−424.698	10.891	13	0.620			
AE (males = females)	993.391	354	285.391	−427.308	10.891	14	0.695	0.43 (0.25–0.58)		0.57 (0.42–0.75)
CE (males = females)	999.411	354	291.411	−424.297	16.911	14	0.261			
E (males = females)	1,013.081	355	303.081	−420.072	30.581	15	<0.001			
Mineral										
Saturated	974.539	340	294.539	−400.191						
ACE (males ≠ females)	985.265	350	285.265	−420.930	10.726	10	0.379			
ACE (males = females)	987.602	353	281.602	−427.592	13.063	13	0.443			
AE (males = females)	987.602	354	279.602	−430.202	13.063	14	0.522	0.45 (0.29–0.59)		0.55 (0.41–0.71)
CE (males = females)	994.549	354	286.549	−426.729	20.010	14	0.130			
E (males = females)	1,013.030	355	303.030	−420.098	38.491	15	<0.001			
Vitamin										
Saturated	989.174	340	309.174	−392.874						
ACE (males ≠ females)	1,000.733	350	300.733	−413.196	11.559	10	0.316			
ACE (males = females)	1,003.013	353	297.013	−419.887	13.839	13	0.385	0.21 (0.00–0.41)	0.04 (0.00–0.34)	0.75 (0.59–0.93)
AE (males = females)	1,003.040	354	295.040	−422.483	13.866	14	0.460			
CE (males = females)	1,003.492	354	295.492	−422.257	14.318	14	0.426			
E (males = females)	1,011.319	355	301.319	−420.954	22.145	15	0.104			
Energy										
Saturated	973.921	340	293.921	−400.500						
ACE (males ≠ females)	982.769	350	282.769	−422.178	8.848	10	0.547			
ACE (males = females)	984.514	353	278.514	−429.136	10.593	13	0.645			
AE (males = females)	984.514	354	276.514	−431.746	10.593	14	0.718	0.48 (0.31–0.61)		0.52 (0.39–0.69)
CE (males = females)	993.593	354	285.593	−427.206	19.672	14	0.141			
E (males = females)	1,010.441	355	300.441	−421.393	36.520	15	<0.001			

−2LL −2(log-likelihood), *AIC* Akaike's information criterion, *BIC* Bayesian information criterion, χ^2 = difference in log-likelihoods between nested models, *df* change in degrees of freedom

variance in *fat*, 31 % of the variance in *protein*, 43 % of the variance in *carbohydrate*, 45 % of the variance in *mineral*, and 48 % of the variance in *energy*, with the non-shared environment accounting for the remaining portion of the variance in these variables. For *vitamin intake*, genetic factors explained 21 % (ns) and the shared environment explained 4 % (ns) of the variance, and 75 % was explained by non-shared environmental influences.

Discussion

The results of the present study provide support for a heritable influence on food intake, with 21 to 48 % of the variance attributable to genetic factors. In addition to confirming previous reports that individual differences in total energy and macronutrient intake are influenced by genetic factors (e.g., de Castro 1993; Hasselbalch et al. 2008), our finding of a heritable basis (21–45 %) for micronutrient intake is novel. Consistent with the previous studies, the remaining variance in nutrient intake was due to the non-shared environment, with little to no significance of the shared environment (de Castro 1993; Hasselbalch et al. 2008; Heller et al. 1988; Hur et al. 1998; Wade et al. 1981). Heritable influences on specific macro- and micronutrients serve as an important and informative indicator of potential nutritional intake over the course of an individual's life. Of note, food recall in this study included nutritional intake both inside and outside the home. Children have relatively limited control over their access to food in general, but this is particularly true for the home setting, where food choice is typically determined by parents or caregivers. At school, children are generally limited to whatever food is provided by the school setting, although they may have a greater range of selection given factors such as school cafeterias usually offering various meal options; food and beverage vending machines; access to classmates' food (e.g., sharing food); for adolescents, access to fast food restaurants or gas stations in vicinity of the school; and the like. In fact, a study done by Contento reports that most adolescents interviewed for the study felt they had a high degree of control over their food choices (Contento et al. 2006). Therefore, it is important for future studies to consider environmental influences on nutritional intake in terms of those largely reflecting parental contributions (i.e., home environment influences) versus those in which the child has more control and selection (i.e., non-home environment influences).

Total energy intake

Our finding that 48 % of variance in total energy intake was due to genetic influences is higher than previous estimates (approximately 30 %). This may be due to greater measurement error for nutrient intake assessment in studies relying on self-reported food frequency questionnaires (Hasselbalch et al. 2008; Hur et al. 1998) and the combination of small and/or limited sample sizes and less sophisticated statistical analysis methods used by the food diary intake studies of Wade (Wade et al. 1981) and Heller (Heller et al. 1988). However, our estimate for genetic influence is less than the 65 % reported by de Castro (de Castro 1993), who used a 7-day diary and linear structural modeling analysis. Although different study duration and analyses may contribute to this difference, it is likely also reflective of differences in the characteristics of subjects. Furthermore, De Castro's exclusion criteria eliminated major factors known to affect energy and nutrient intake (e.g., subjects could not be living together, dieting, or alcoholic). Our sample is also younger (mean age = 11.79) than de Castro's (mean age = 38.8), as well as other studies specifically examining nutrient intake. Although an influence on age is usually seen in studies examining food consumption and phenotypes such as food preference (Breen et al. 2006; Faith et al. 2008; Keskitalo et al. 2008), the lack of nutrient intake studies in populations under the age of 17 years may compromise our ability to compare our findings to previous studies.

Macronutrient intake

Genetic effects accounted for approximately 30 to 45 % of variance in macronutrient intake. We found little difference among the heritabilities for overall energy intake and for individual macronutrient components. These findings are generally consistent with the previous studies using food diaries (Heller et al. 1988; de Castro 1993) as well as other methodologies, including questionnaires (Fabsitz et al. 1978; Hur et al. 1998). Our finding that the heritability of protein was the lowest among the macronutrients is also consistent with the previous findings, although the magnitude varies among studies. Heritability estimates have been previously reported around 25–60 % for carbohydrates and fats (de Castro 1993; Heller et al. 1988; Hur et al. 1998; Wade et al. 1981).

As suggested by Hur et al., the genetic factors accounting for overall energy intake likely overlap with those contributing to the individual macronutrient components, since total energy intake is the sum of the individual macronutrients (Hur et al. 1998). However, de Castro found a residual effect of genetics on daily intake of each macronutrient, even after accounting for the overall intake, demonstrating a significant ($p < 0.05$) genetic effect on individual macronutrient intake separate from the genetic effect on overall intake (de Castro 1993). Indeed, although genetic studies have been limited in their examination of

specific food intake and appetite factors, studies have begun to shed light on how polymorphism of specific genetic loci is associated with specific macronutrient intake. For instance, the gene *TUB* has been associated with fat and carbohydrate intake; particularly, in women (van Vliet-Ostaptchouk et al. 2008), *MC4R* has been associated with total energy, total fat, and protein intake (Qi et al. 2008), and a single-nucleotide polymorphism at the *FTO* locus has been found to be related to fat intake in children (Timpson et al. 2008).

Micronutrient intake

To our knowledge, this is the first study demonstrating a genetic influence on mineral and vitamin intake. In rare cases, genetic factors have been shown to affect the body's ability to extract nutrients from food, as with hemochromatosis (a genetic inability to metabolize iron), lysosomal storage disorders (genetic enzyme disorders that impair fat metabolism), and phenylketonuria (a genetic inability to break down the amino acid phenylalanine) (Elliott and Ong 2002; Farhud and Yeganeh 2010). However, their influence on the actual intake of these micronutrients has not been observed and remains unclear. Although minerals and vitamins do not contribute directly to total energy intake, they are critical for biological processes even at the level of the genome (Farhud and Yeganeh 2010) and may influence preferences, behaviors, and other phenotypes that can in turn affect energy intake.

Minerals

The present study found that genetic effects accounted for 45 % of variance in mineral intake, which is similar to what was found for macronutrient and total energy intake. This similarity may reflect a relationship between micronutrient intake and behavioral eating patterns and preferences. Indeed, eating patterns have been shown to affect micronutrient intake. For example, boys and girls who obtain high percentages of energy from snack food consumed between main meals have demonstrated significantly lower intakes of micronutrients (Sjöberg et al. 2003). Increased sugar consumption (Gibson and Neate 2007) and skipping breakfast (Deshmukh-Taskar et al. 2010) have also been negatively correlated with adequate micronutrient intake in children. Future twin studies which assess breakfast intake as well as nutrient intake could further assess whether heritability of specific behavioral patterns such as these can account for heritability of micronutrient intake (i.e., whether a genetic correlation between eating breakfast and nutrient intake exists).

Vitamins

At 21 %, the heritability for vitamin intake in our study was less than half of that found for the other measured variables. Although variation was predominantly attributable to the non-shared environment (75 %), there was a shared environment effect also present (4 %). It is possible that the low heritability of vitamin intake may reflect or underlie the similar pattern seen in food types that are usually the source of these micronutrients. Notably, Hasselbalch et al. (2008) recently reported that genetic effects were nonexistent for the consumption of fruits and low for vegetables in both men (24 %) and women (14 %). For these food types, the shared (40–46 %) and non-shared (37–59 %) environment exerted a much greater influence. These findings are somewhat at odds with previous research showing genetic factors to account for 42–46 % of the variation in fruit and vegetable consumption in young children (Breen et al. 2006) as well as approximately 40–50 % of the use of "healthy" foods including fruits and vegetables (Keskitalo et al. 2008; van den Bree et al. 1999). However, compared to these earlier studies, Hasselbalch et al. have the advantage of using a large, population-based sample with dietary information based on an extensive 247 item FFQ used to generate 20 food groups (Hasselbalch et al. 2008). This is in contrast to earlier FFQ of 24–99 items that generate only four broad categories (Breen et al. 2006; Keskitalo et al. 2008) and two eating patterns (van den Bree et al. 1999).

The consistently strong non-shared environmental influence, for nutrient intake, especially on vitamin intake, emphasizes the potential importance in helping individuals develop healthy eating habits in order to prevent nutritional and consequent health problems. In this regard, the low genetic and shared environmental effects—and very high non-shared environmental effect—for vitamin intake are particularly promising. Like minerals, vitamins play an important role in biological processes and have been shown to hold important implications in a wide range of conditions, such as neurocognitive deficits in children (Liu et al. 2003) and behavior problems across childhood (Liu et al. 2004; Liu and Raine 2006), as well as chronic diseases, including multiple sclerosis (Hayes 2000), cancer (Guyton et al. 2001), osteoarthritis (McAlindon et al. 1996), cardiovascular disease (Ryan-Harshman and Aldoori 2008), and cognitive impairment and dementias (Selhub et al. 2010). The emerging focus on nutritional genomics and genetics has provided increasing evidence for the importance of micronutrients in genome stability and health. Even small damages caused by micronutrient deficiencies in the genome can produce life-threatening consequences. The prevention, control, and treatment of chronic diseases in the future may utilize dietary interventions based on an

understanding of the interaction and dependencies between individual genotypes and nutritional requirement and status (Farhud and Yeganeh 2010). Thus, an improved understanding of micronutrient–genetic relationships may provide key data to shed light on the relationship between nutrition and human disease.

Furthermore, twin studies have shown that virtually all phenotypes are heritable, including personality traits (McGue et al. 1993), behaviors, disorders, and diseases (Plomin et al. 2001). Genome-wide association studies have provided important information about genetic variants and the genetic architecture of phenotypes (Dick et al. 2004; Viding et al. 2010). However, most variants identified so far explain only a small proportion of total genetic variance, while the remaining "missing" heritability may be attributed to a contribution of additional genetic, environmental, and epigenetic factors, such as DNA methylation (Bell and Saffery 2012; Bell and Spector 2011).

Monozygotic (MZ) twin concordance for several psychiatric conditions is seldom 100 % (Petronis et al. 2003), indicating that environmental and/or epigenetic factors modulate the phenotypes. Methylation differences have been found in MZ twin pairs discordant for environmental exposure (Kaminsky et al. 2008; Sutherland and Costa 2003). Also, epigenetic differences between MZ twins have been found to increase over time as the twins get older (Fraga et al. 2005). Given their young age (i.e., 11–13 years), twins in the present study likely purchased few meals outside the home, meaning they had less control over what was consumed. However, as children age, they become more autonomous and have greater independent access to food (e.g., having access to money; having the ability to drive). Differences in environmental exposure (i.e., consuming different types of food) are therefore likely to partly influence DNA methylation as these twins get older.

Study limitations

Despite the strengths of our design and analyses, such as the use of food diaries, and reliable statistical methods, our study is not without limitations. First, although the 3-day food diary has been shown to be a reliable and valid method for measuring nutrient intake (Tremblay et al. 1983), individual diets can vary greatly on a day-to-day basis (Block 1982) and our 3-day period of study may not have been long enough to capture several long-term genetic effects. Using a 7-day food diary, such as de Castro (de Castro 1993) did, might have revealed higher heritability estimates for our measured variables. However, a recent evaluation comparing measures from different 3-day intervals within a 7-day recording period found no significant difference in the mean energy intake, macronutrient intake, or micronutrient intake determined from possible 3-day periods and the total 7-day period (Fyfe et al. 2010). Nevertheless, this study was performed on adults, and it is possible the influence of weekly intervals may be different on adult eating behavior than on that of children. Another potential limitation is that the present study's sample, which is population based but also consists of only twins between the ages of 11 and 13 years old, may limit the generalizability of our findings. This narrow age does, however, allow insight into age-related patterns of intake (i.e., vitamin) not previously described in the literature. More research regarding micronutrient intake in other age-groups may shed light into patterns similar to those seen in the changing influence of genetic, shared, and non-shared environments in food preferences (Breen et al. 2006; van den Bree et al. 1999). Finally, there are several assumptions in classical twin design that may not have been met here (for a more detailed discussion, see Plomin et al. 2001). For example, it is generally assumed that genetic and environmental influences are uncorrelated and do not interact. Dominant genetic and shared environmental influences are confounded and cannot be estimated simultaneously in a study of MZ and DZ twins reared together.

Conclusion

Much work is needed before an adequate understanding of the mechanisms behind gene–environment interactions on food intake is achieved. The current study adds to the existing literature by confirming the strong effects of genetic and the non-shared environment on total energy and macronutrient intake. The study also extends these findings to micronutrients. Although effects on mineral intake were very similar to those on overall energy and macronutrient intakes, vitamin intake revealed a relatively low genetic effect and the presence of shared environment effects. That the large majority of variability in vitamin intake, however, was attributable to non-shared environmental effects in our sample population highlights the importance of encouraging good nutritional habits in adolescents, so as to prevent development of later nutritional and health-related problems. Our findings also encourage the continued momentum in the field of nutrigenomics and nutrigenetics, as the integration and expansion of literature in these subdisciplines in conjunction with food intake and use studies may inform further studies in obesity, hypertension, and other dietary-related health outcomes. Furthermore, the fact that we did not find any significant sex differences in the relative magnitude of genetic and environmental influences could be strength with the present study as none of the studies in Table 1 have examined the gender effect. Finally, findings on macronutrient and

micronutrient intake specifically may be useful for understanding the heritable aspects of nutritional deficiencies and their influence on physical and behavioral outcomes.

Acknowledgments This study was supported by grants from NIMH (RO1 MH58354) to Laura Baker, NIMH (Independent Scientist Award K02 MH01114-08) to Adrian Raine, and NIH/NIEHS (K01 ES015877-01 and K02 ES019878-01) to Jianghong Liu. The authors also wish to thank research assistants Xiaoyang Ma, for her assistance in data organization and initial data analysis, and Linda Li, for her literature compilation and analysis.

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