
Genetic and Environmental Influences on Body Size in Early Childhood: A Twin Birth-Cohort Study

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Genetic and environmental contributions to body size from birth to 5 years in a population-based twin cohort were studied. Sex differences in gene–environment etiology were also explored. Analyses used data from the Quebec Newborn Twin Study (QNTS), a population-based birth cohort of 672 twin pairs. The final sample consisted of 177 complete twin pairs. Heritability of weight was moderate at birth while common environmental factors accounted for almost half of the variance. Influence of family environment disappeared by 5 months and genetic effects were high (approximately 90%) for both sexes at 5 months and 5 years. Adjustment of weight for height yielded similar results as for weight alone. Slight but significant sex-limitation of genetic effects was observed at 5 months. Overall, genetic factors accounted for 40% of birthweight variance, with intrauterine environment influences explaining almost half. However, genetic factors accounted for most of the variance in weight. These results do not imply a lack of environmental effects on body weight, but rather a lack of: (1) environmental effects that are *independent* from genetic liability, and/or (2) a lack of significant environmental *variation* in the population (e.g., uniform nutritional habits) that leaves genetic differences between children to generate most of the variance in weight.

Developed countries are facing a new health threat: the epidemic of childhood obesity (Dubois, 2006; Lobstein et al., 2004; WHO, 2000; U.S. Department of Health and Human Services, 2001). Overweight and obesity in some countries now affect between 5% and 15% of children and adolescents (Chinn & Rona, 2001; Danish Ministry of Health, 1999; Russell et al., 1999; U.S. Department of Health and Human Services, 2001; UK Department of Health, 1999). The origins of obesity may be traced to circumstances experienced in utero and in the first months of life. Birthweight is

associated with body mass index (BMI) in childhood and in adulthood (Dietz & Gortmaker, 2001; Lobstein et al., 2004; Ong et al., 2000; Parsons et al., 1999; Stettler et al., 2002; Tanaka et al., 2001). Current treatment for obesity is not very effective at the population level. It is therefore important to get a better understanding of the effects of genes and environment in relation to body measures in the first years of life.

The existence of genetic effects on obesity has been well researched in adults, but little is known about the extent of the genetic contribution to childhood obesity and whether it differs from etiologic findings in adults (Parsons et al., 1999). Twin studies are among the most powerful tools for resolving the respective contributions of genetic and environmental effects on physical and behavioral phenotypes (Evans & Martin, 2000). The majority of twin studies, however, have been conducted with adults and have generally been cross-sectional. Results show a strong heritability for adult BMI (Maes et al., 1997). Sex differences in heritability for body weight are inconsistent (Herskind et al., 1996). It is not clear either to what extent genes act in complement to common and unique environmental factors. Few studies on body size of adolescent or children twins are available (Maes et al., 1997). Even fewer studies have been performed on large samples of twins within a longitudinal design. For their study of 1818 pairs of twins born in the United Kingdom (UK) in 1994, Koeppen-Schomerus et al. (2001) used height and weight reported by the parents and weight corrected for height for 4-year-olds, and concluded that unlike in adults, weight and overweight among 4-year-olds displayed

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substantial environmental family influence. In a smaller sample of 66 pairs of twins aged 3 to 17 years, Faith et al. (1999) estimated that genes contributed to between 75% and 80% of the variance in fat mass, the rest being related to nonshared environmental influences.

The aim of this research was to study genetic and environmental contributions to body size variables from birth to 5 years in a population-based twin cohort, using laboratory measures of weight and height for weight, height, and weight-adjusted-for-height. Sex differences in gene-environment etiology were also explored.

Methods

The analysis was done using data from the Quebec Newborn Twin Study (QNTS; Pérusse, 1995). The QNTS is a population-based birth cohort generated from all twin births occurring in the seven health districts of the greater Montreal area between April 1st, 1995 and December 31st, 1998. When the twins were aged between 59 and 61 weeks from conception, or 5 months corrected for gestational age, families were contacted to participate in the study. Twins with chronic diseases and those who died before the age of 5 months were not part of the cohort. The QNTS is based on a total of 672 twin pairs, or 1344 twins. Participants were followed longitudinally at 18, 30, 48 and 60 months, using a series of questionnaires and laboratory measures.

Potential biases related to the perceived zygosity of subjects were eliminated by using a procedure whereby all measures were taken on each twin of a pair by different research assistants for all pairs. Opposite-sex dizygotic (DZ) pairs were included, enabling the exploration of potentially important sex differences in etiology. Zygosity was diagnosed through aggregation of independent tester ratings based on the live assessment of physical similarity of twins at 5 and 18 months of age. Mouth swabs were collected by mothers at 5 months for all same-sex twins; the DNA was extracted from the cells, amplified by polymerase chain reaction, and typed using 8 to 10 highly polymorphic micro-satellite markers, yielding a zygosity diagnosis of above 99.8% accuracy. A comparison of physical similarity and DNA diagnoses was performed in a random subsample of same-sex pairs and suggested above 96% agreement between the two methods (Forget-Dubois et al., 2003).

Children's height (in centimeters — cm) and weight (in grams — g) were measured at birth (collected from medical records) and in the laboratory at 5 and 60 months. Half of the pairs have height and/or weight values at 5 months and two thirds at 60 months; therefore, data from 210 pairs of twins are available for the analyses. Weight *z*-scores (adjusted for height) based on age–sex CDC Growth Curves (National Center for Health Statistics and the National Center for Chronic Disease Prevention and Health Promotion, 2000) were calculated. Outlier values, found in 36 children (i.e., 36 children had one outlier value for height or weight or weight-adjusted-for-height — all of them were part of the same twin pair, therefore 18 pairs of twins had at

least one outlier value), were not included in the analysis to assess normality in distribution; they were removed only when their values were found to be nonrealistic as compared to other databases of height and weight in same-aged children. Outlier identification was done separately for height and weight and amongst each child of the pair. Missing values ($n = 15$ children) were also removed. For these children, height is missing for one twin, or weight and height are missing at 5 months for both twins. All children with weight and height at 60 months have birthweight and height data. Of a potential 210 pairs of twins, the final sample for the analysis was 177 (85%) complete twin pairs (85 monozygotic [MZ] pairs: 45 female, 40 male; 92 DZ pairs: 21 female, 25 male, and 46 opposite-sex), comprised of twins with complete measures of weight and height at birth, 5 months, and 60 months. This sample size is still sufficient to provide powerful measures of genetic estimates since the method of estimation is based on clustered groups analyses as described below. Dependent variables (i.e., weight, height, and weight-adjusted-for-height) have been standardized with a mean of 0 and variance of 1 to ensure normality distribution of data, a critical assumption of the statistical technique used in the genetic analyses.

Intraclass correlations were first calculated for the five zygosity-sex groups (MZ-boys, MZ-girls, DZ-boys, DZ-girls, DZ-opposite-sex) for each variable (weight, height, weight-adjusted-for-height) at each age (birth, 5 months, 5 years) to obtain a general overview of genetic and environmental influences. Model-fitting (Neale & Cardon, 1992) was performed to formally test for different etiologic models and to estimate the magnitude of genetic and environmental effects.

Since twins are clustered within pairs, they form a natural two-level hierarchy (level 1 for individual twins and level 2 for twin pairs). A hierarchical random-effect multilevel model of twin data (Guo & Wang, 2002; Pérusse, 1999) was developed. Such a model considers simultaneously the variation at each level by estimating two residual variance terms, one for the within-twin pair variance (level 1) and one for the between-twin pair variance (level 2). A multilevel model has two parts: the fixed part represents the average relation for all individuals regardless of grouping, and the random part accounts for the variation at each level. Random-effect multilevel models allow for a full likelihood estimation of all parameters (Goldstein, 1995).

We first built a model that completely specifies the means, between-pair and within-pair variances separately for MZ and DZ twins. Thus, the predicted means, variances and covariances of this model are equal to their observed values in both twin groups. This can be written as a random-effect multilevel model with six parameters:

Model A

$$P_{i,j} = \alpha_{MZ}MZ_j + \alpha_{DZ}DZ_j + \beta1_j^{MZ}MZ_j + \beta1_j^{DZ}DZ_j + \beta2_{ij}^{MZ}MZ_j + \beta2_{ij}^{DZ}DZ_j$$

where P_{ij} is the phenotype of individual i (level 1) in the j^{th} pair (level 2), and MZ_j and DZ_j are observed indicator variables denoting zygosity. Thus, α_{MZ} is a fixed parameter that represents the mean phenotypic value across MZ twins while α_{DZ} models the mean across DZ twins. The random parameters $\beta_{1_j}^{MZ}$ and $\beta_{1_j}^{DZ}$ vary only between twin pairs and their variance represents the between-pair variances for MZ and DZ twins, respectively. In the same way, the random parameters $\beta_{2_{i,j}}^{MZ}$ and $\beta_{2_{i,j}}^{DZ}$ vary only between individuals and their variance represents the within-pair variances for MZ and DZ twins, respectively. Since MZ and DZ are mutually exclusive, they are included in the model as dummy variable where 1 is MZ and 0 not MZ, and 1 is DZ and 0 not DZ, for MZ and DZ respectively. Therefore the model for MZ will include only the terms with MZ while the term with the DZ besides them will be included in the model for DZ.

In a twin design, the random part of the model can be specified to reflect additive genetic (A), common environmental (C) and individual environmental (E) components of phenotypic variance as follows:

Model B

$$P_{ij} = \mu_p + g_j^{MZ}MZ_j + g_{1_j}^{DZ}DZ_j + g_{2_{i,j}}^{DZ}DZ_j + C_j + \epsilon_{i,j}$$

where P_{ij} is the measured phenotype (e.g., body weight) of the i^{th} individual in the j^{th} twin pair, μ_p is a constant representing the mean phenotypic value for both MZ and DZ twins, C_j and $\epsilon_{i,j}$ are pair-level and individual-level random parameters for which the variances represent the effects of common and individual environmental influences respectively for all twins, MZ_j and DZ_j are indicator variables denoting zygosity of the j^{th} pair and are thus indices of genetic similarity, g_j^{MZ} and $g_{1_j}^{DZ}$ represent the contribution to the between-pair variance of the effects of genetic similarity on the phenotype and $g_{2_{i,j}}^{DZ}$ represents the contribution to the within-pair variance of the effects of genetic dissimilarity on the phenotype in DZ twins. Since MZ twins are identical genetically, there are only common genetic effects in these twins; however, there are two genetic components equally contributing to phenotypic variance in DZ twins, one common and one unique, corresponding to the shared and unshared genetic effects respectively. Thus, we have the following constraint on the genetic variances:

$$\text{Var}(g_{1_j}^{DZ}) = \text{Var}(g_{2_{i,j}}^{DZ}) = \text{Var}(g_j^{MZ})/2$$

The resulting ACE model estimates four parameters (phenotypic mean, additive genetic variance, common environmental variance and unique environmental variance) and has two degrees of freedom. Specifications are similarly formulated for all submodels. For example, a CE model (no genetic effect) is specified from the ACE model by removing all genetic components at both levels, thus leaving three parameters to estimate and three degrees of freedom. An AE model (no effect of the family

environment) can be similarly specified. These models described above are referred as being the univariate models.

Models can also be derived to test for sex-limitation in genetic and/or common environmental effects. This is done by having two models, one for each sex, tested simultaneously (Model C) and controlling for the covariance between opposite-sex DZ twins for A and C components. In fact, this is the same model as shown previously (Model B) but with the addition of dummy variables for each sex as MZ in Model B. Thus, ACE, CE, and AE models, all with common effects, correlated effects, and uncorrelated effects can be tested.

Model C

$$P_{ij} = \mu_{pb} + \mu_{pg} + g_j^{MZ}BMZ_j + g_{1_j}^{DZ}BDZ_j + g_{2_{i,j}}^{DZ}BDZ_j + g_j^{MZ}GMZ_j + g_{1_j}^{DZ}GDZ_j + g_{2_{i,j}}^{DZ}GDZ_j + C_j + \epsilon_{i,j}$$

where G is a dummy variable for girls and B is a dummy variable for boys.

The following analysis strategy was applied for each dependent variable and each age. The sex-limited saturated model and the univariate saturated model were first fitted to examine the sex effect in the genetic component. The two saturated models were compared using the likelihood ratio test (-2Log) and when this test was significant at the .05 level, the sex-limitation models were used in the analyses; whenever the sex-limited saturated model did not reveal any sex effect, the univariate ACE models were used in the analyses. Tabulated results reflect the type of analyses that were used by presenting the estimates by sex if the sex-limited models were used, and estimates for all children without indication for sex otherwise. Successive modeling from saturated to reduced models (ACE, AE, CE, E) was performed. The reduced models were compared to the full (saturated) model by means of a likelihood ratio test (-2Log) and the Akaike's Information Criterion (AIC: $\text{chi-square}-2df$), which takes into account both goodness-of-fit and parsimony in the assessment of a model's explanatory value. This selection of models involving AIC tend to have more power and require a smaller sample size. Statistical analysis and model-fitting were done using SAS/NLMIXED 8.2 and the level of statistical significance was set at .05.

Results

The average birthweight for all children irrespective to zygosity and sex was 2.5 kg ($SEM = 0.02$). Average weight at 5 months was 7.4 kg ($SEM = 0.04$) and at 60 months was 19.1 kg ($SEM = 0.10$). The average height for all children irrespective to zygosity at birth was 46 cm ($SEM = 0.1$), at 5 months was 64 cm ($SEM = 0.1$) and at 60 months was 110 cm ($SEM = 0.2$). The differences between sex/zygosity were captured through the intraclass correlation analyses. The intraclass correlations shown in Table 1 display several important features: (1) For all variables at all

Table 1

Intraclass Correlations for Body Measures at Birth, 5 Months, and 5 Years

Variable	Age	MZ Twins			DZ Twins			
		Girls (<i>n</i> = 90)	Boys (<i>n</i> = 80)	Total (<i>n</i> = 170)	Girls (<i>n</i> = 42)	Boys (<i>n</i> = 50)	Mixed (<i>n</i> = 92)	Total (<i>n</i> = 184)
Weight	Birth	.840	.878	.866 ^c	.689	.736	.601	.672 ^c
	5 mo	.890 ^a	.887	.900 ^c	.531 ^a	.704	.394	.548 ^c
	60 mo	.834	.902 ^b	.869 ^c	.669	.188 ^b	.603	.536 ^c
Height	Birth	.763	.889	.841 ^c	.414	.768	.557	.596 ^c
	5 mo	.807	.748	.799 ^c	.566	.583	.637	.623 ^c
	60 mo	.890 ^a	.862 ^b	.879 ^c	.538 ^a	.412 ^b	.724	.631 ^c
Weight-adjusted-for-height	Birth	.808	.851	.837 ^c	.709	.697	.610	.673 ^c
	5 mo	.878 ^a	.879	.887 ^c	.485 ^a	.668	.307	.493 ^c
	60 mo	.756	.896 ^b	.826 ^c	.660	.181 ^b	.565	.514 ^c

Note: ^a intraclass correlation for MZ significantly ($p \leq .05$) different from DZ (girls only)^b intraclass correlation for MZ significantly ($p \leq .05$) different from DZ (boys only)^c intraclass correlation for MZ significantly ($p \leq .05$) different from DZ (total)

ages, MZ correlations are greater than DZ correlations, which suggests pervasive genetic effects on body measures in early childhood; (2) for height at all ages and for all variables at birth, however, MZ correlations are less than twice DZ correlations, indicating the presence of common environmental factors on these measures; (3) for all variables (except height) at all ages (except birth) MZ correlations were both high (close to or greater than .8) and approximately twice greater than DZ correlations, which suggest very strong heritability for these variables; and (4) different correlations by sex were significant in some cases, indicating potential sex-limitation for those measures.

Best fitting and most parsimonious models, as determined by nonsignificant chi-square deterioration from the full model and lowest AIC, are presented in Tables 2, 3, and 4. For body weight (Table 2), the best models were ACE for birthweight, AE with genetic sex-limitation at 5 months, and AE at 60 months. Heritability was moderate at birth while common environmental factors accounted for close to half of the variance. This influence of the family environment disappeared as

early as 5 months and genetic effects were then very high (approximately 90%) for both sexes at 5 months and 5 years. Unique environmental influence, which includes measurement error, was minor at all ages.

Height (Table 3) displayed a very different etiology. ACE models were retained at all ages, with genetic effects fluctuating from low (22%) at 5 months to moderate (54%) at 5 years, and common environmental factors explaining approximately two thirds of the variance in height at 5 months, with a slight but significant sex-limitation. Unique environmental factors, once again, were negligible.

When weight was adjusted for height (Table 4), results were similar to the ones for weight only. Common environment exerted the main influence at birth but no influence thereafter, genetic factors explaining between 84% to 88% of weight-for-height variance at 5 months and 5 years.

Discussion

This study sheds light on the respective contributions of genes and family environmental factors on body size

Table 2Best Fitting Model^a for Body Weight and Proportion (%) of Variance Explained by Additive Genetic (a^2), Common Environmental (c^2), and Unique Environmental (e^2) Effects

Age	Model ^a	χ^2	<i>df</i>	<i>p</i> value	AIC	Sex	a^2	c^2	e^2
Birth	ACE	2.135	2	.3439	774.798	Both	39.9 (19.0–60.8)	47.1 (26.7–67.6)	13.0 (8.4–17.5)
5 months	AE (with sex)	5.741	5	.3322	842.772	Girls	90.0 (85.3–94.7)	—	10.0 (5.3–14.7)
						Boys	87.1 (81.2–93.1)	—	12.9 (6.9–18.8)
60 months	AE	2.105	3	.5509	886.179	Both	87.7 (83.6–91.9)	—	12.3 (8.1–16.4)

Note: ^a Selected among all possible models (ACE, AE, CE, E — not all shown) based on lowest AIC and nonsignificant chi-square likelihood ratio test of model against saturated model ($p > .05$)

Table 3

Best Fitting Model^a for Height and Proportion (%) of Variance Explained by Additive Genetic (a^2), Common Environmental (c^2), and Unique Environmental (e^2) Effects

Age	Model ^a	χ^2	df	p value	AIC	Sex	a^2	c^2	e^2
Birth	ACE	2.703	2	.2589	821.370	Both	44.5 (19.7–69.4)	39.0 (14.9–63.1)	16.5 (10.9–22.1)
5 months	ACE (with sex)	2.284	4	.6837	385.885	Girls	24.1 (4.6–43.6)	65.8 (45.4–86.1)	10.1 (5.1–15.0)
						Boys	22.3 (4.6–39.9)	61.9 (41.9–81.8)	15.8 (8.8–22.9)
60 months	ACE	0.841	2	.6567	842.871	Both	54.0 (30.8–77.1)	34.8 (11.8–57.8)	11.2 (7.3–15.2)

Note: ^a Selected among all possible models (ACE, AE, CE, E - not all shown) based on lowest AIC and nonsignificant chi-square likelihood ratio test of model against saturated model ($p > .05$)

measures in the first half of childhood. Results indicate that genetic factors accounted for 40% of birthweight variance, with the influence of the intrauterine environment explaining almost half of the variance. These results are in striking agreement with those of other studies such as Vlietinck et al. (1989), who also found a heritability of 40% for birthweight. It should be noted, however, that the apparent environmental effect of the intrauterine environment on birthweight may be related to the special situation of twinning, with monozygotic MZ twins competing more intensely for prenatal resources than dizygotic DZ twins because of their shared placental membranes (Forget-Dubois et al., 2003; Vlietinck et al., 1989). Further research taking chorionicity into account is needed for body measures at birth.

In any case, genes swiftly exert their influence in the postnatal period, accounting for as much as 90% of the variance in body weight at 5 months and only slightly less (87%) 5 years later. Considering that the very low variance related to the unique environment (around 10%) includes measurement error, it appears safe to conclude that genetic factors account for nearly all of the reliable variance in body weight in the first half of childhood. Similar results were observed previously, but only in school-age children. In a review of studies from different countries, Maes et al. (1997) reported high heritability estimates for BMI from 8 to 16 years of age, ranging from .67 to .93 (Hebebrand et al., 2001). Our study strongly suggests that genes basically account for body weight variation in children from birth to 5 years.

These results should not be construed as implying a lack of environmental effects on body weight, but rather a lack of environmental effects on body weight that are *independent* from genetic liability. The very strong heritability we observe does not reveal *how* genes influence body weight, only that they do so in a decisive way. Basically, genes may act on body weight through influence on caloric intake, metabolism, and caloric expenditure (Marti et al., 2004). This implies that genes, for instance, may impact the *environmental* input of food both in terms of quantity (through control of appetite) and quality (food preferences). A strong genetic influence does not preclude environmental intervention and treatment of a condition when gene-by-environment

interaction is at work, as the classic example of the disorder phenylketonuria (PKU) attests to; PKU is a monogenic condition whose devastating phenotypic effects can be largely negated with a phenylalanine-free diet (National Institutes of Health Consensus Development Panel, 2001). Moreover, a heritability of 80% to 90% for body weight, as found here, does not mean that 80% to 90% of weight is determined by genes, but rather that genes determine 80% to 90% of *variation* in body weight between individuals in a specific population. It may very well be that nutritional and physical activity habits, for example, are mostly invariant across children in this population — which would leave individual genetic differences to account for most of the phenotypic variance. There is data showing that this is indeed the case for nutrition among Quebec children (Desrosiers et al., 2005) as in other Western countries (Dubois, 2006).

In contrast to body weight, we found a varying but substantial influence of common environmental factors on height throughout early childhood. After what may be the special situation of birth, however, the heritability of height appears to be increasing with age, although more data points are needed to confirm this pattern. This information is important as BMI varies in short and tall young children (Buchan et al., 2007). Genetic influence was already estimated at 54% at 5 years, more than double that at 5 months (23%), and large-scale twin studies have repeatedly produced heritability estimates of approximately 70% for adult height (e.g., Eaves et al., 1989).

Despite the fact that height enters in the computation of weight-adjusted-for-height, individual differences in these composite variables appeared to be driven essentially by genetic effects, as was the case for weight alone. The magnitude of genetic influences hovered around 85%. Again discounting measurement error, it appears that genes determine most of the reliable variance of weight-adjusted-for-height in the first half of childhood.

Genetic sex-limitation observed in this study has been reported elsewhere for BMI, with heritability found to be higher for men than for women (Maes et al., 1997; Schousboe et al., 2003). Sex differences in heritability for weight-adjusted-for-height seem to develop in the

Table 4Best Fitting Model^a for Weight-Adjusted-for-Height and Proportion (%) of Variance Explained by Additive Genetic (a^2), Common Environmental (c^2), and Unique Environmental (e^2) Effects

Age	Model ^a	χ^2	df	p value	AIC	Sex	a^2	c^2	e^2
Birth	ACE	1.646	2	.4391	796.427	Both	33.7 (12.3-55.1)	50.4 (30.0-70.8)	15.9 (10.4-21.4)
5 months	AE (with sex)	3.034	5	.6947	864.073	Girls	87.6 (81.9-93.4)	—	12.4 (6.6-18.1)
						Boys	88.0 (82.2-93.7)	—	12.0 (6.3-17.8)
60 months	AE	1.929	3	.5873	926.238	Both	84.0 (78.6-89.3)	—	16.0 (10.7-21.4)

Note: ^a Selected among all possible models (ACE, AE, CE, E – not all shown) based on lowest AIC and nonsignificant chi-square likelihood ratio test of model against saturated model ($p > .05$)

preschool years, even if no sex difference is seen at this age for height, contrary to adult data indicating that heritability in height is lower in women than in men (Silventoinen et al., 2003).

Overweight and obesity are complex diseases for which the multifactorial polygenic threshold model of heredity is generally considered as the appropriate etiologic model. It is therefore important to study the complete distribution in body weight to gain full knowledge of the causes of individual variation and of distributional extremes such as obesity. Longitudinal twin studies illustrate that heritability for BMI decreases with age for men and women, and that heritability of BMI is higher in adolescents than in adults (Maes et al., 1997). We observed in our study that heritability for weight-adjusted-for-height is stronger at 5 months than at 5 years, and may continue to decline over years. Genetic effects on weight-adjusted-for-height in our study were very strong. The variation reported in heritability for BMI in school-age children and adolescents (60% to 90%; Maes et al., 1997) may be due to the fact that sex-limitation analysis was not always performed, or to differences in populations. For example, a study of adolescent twins showed higher heritability for Caucasian pairs (87%; Maes et al., 1997), which is what we find in our sample of mostly Caucasian twins.

As discussed above, strong genetic influence does not preclude powerful environmental modification of phenotypic outcome. In fact, the design of intervention programs for overweight and obesity would benefit from increased knowledge about genetic liability, its onset, and its influence on particular components of the overweight-obesity complex. More research is especially needed on potential gene-by-environment interaction processes that may be involved in caloric intake, baseline metabolism, and caloric expenditure.

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