

Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment^{1–3}

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ABSTRACT

Background: Body mass index (BMI) has been shown to be highly heritable, but most studies were carried out in cohorts born before the onset of the “obesity epidemic.”

Objective: We aimed to quantify genetic and environmental influences on BMI and central adiposity in children growing up during a time of dramatic rises in pediatric obesity.

Design: We carried out twin analyses of BMI and waist circumference (WC) in a UK sample of 5092 twin pairs aged 8–11 y. Quantitative genetic model-fitting was used for the univariate analyses, and bivariate quantitative genetic model-fitting was used for the analysis of covariance between BMI and WC.

Results: Quantitative genetic model-fitting confirmed substantial heritability for BMI and WC (77% for both). Bivariate genetic analyses showed that, although the genetic influence on WC was largely common to BMI (60%), there was also a significant independent genetic effect (40%). For both BMI and WC, there was a very modest shared-environment effect, and the remaining environmental variance was unshared.

Conclusions: Genetic influences on BMI and abdominal adiposity are high in children born since the onset of the pediatric obesity epidemic. Most of the genetic effect on abdominal adiposity is common to BMI, but 40% is attributable to independent genetic influences. Environmental effects are small and are divided approximately equally between shared and nonshared effects. Targeting the family may be vital for obesity prevention in the earliest years, but longer-term weight control will require a combination of individual engagement and society-wide efforts to modify the environment, especially for children at high genetic risk. *Am J Clin Nutr* 2008;87:398–404.

KEY WORDS Heritability, waist circumference, BMI, childhood obesity, twins

INTRODUCTION

The dramatic rise in childhood obesity in the past 15 y (1) is clearly due to changes in the environment, because genes have not altered. However, not all children are obese. This difference could be due to inherited genetic differences between children or to differences in their rearing environments. Twin studies provide a unique method for disentangling nature and nurture by taking advantage of the fact that monozygotic twins share all of their genes, whereas dizygotic twins on average share half of their segregating genes (2). If genetic influence is important, monozygotic twins must be more similar than dizygotic twins.

Twin studies can also estimate the extent to which the family environment makes family members more similar than would be expected from their genetic relatedness (the shared-environment effect). This is important in the field of childhood obesity because there is considerable interest in the role of the family. Finally, twin studies can go beyond pitting nature against nurture to consider interactions between genes and environment. A novel type of gene-environment interaction is a change in the relative influences of genes and environment after major changes in the environment.

A 1997 review of published adult twin and adoption studies found that variation in body mass index (BMI; in kg/m²) was largely due to heritable genetic differences (3). Studies published since 1997 have reached the same conclusion, with heritability estimates in adults ranging from 55% to 85% (4–7). Twin studies also show that most of the nongenetic effect comes from environmental factors that are unique to each person (nonshared-environment effects) and not from the shared family context; this observation has been confirmed by results from adoption studies (8,9). Contrary to widespread assumptions about the influence of the family environment, living in the same home in childhood appears to confer little similarity in adult BMI beyond that expected from the degree of genetic resemblance.

One limitation of existing twin studies is that many were carried out in adults, for whom the family home is not a contemporary environment. Shared-environment effects may be stronger in pediatric samples, as has been observed in 2 studies of very young twins (10,11). Most existing studies were also carried out in cohorts born before the onset of the current “obesity epidemic.” Obesogenic environments may either overshadow the observable effect of genetic differences or boost it by providing a permissive substrate for the expression of susceptibility.

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Abdominal obesity has increased even faster than BMI in pediatric populations (12–14); this increase has serious health implications, because visceral fat appears to be the primary cause of obesity-related health risks (15, 16). Twin designs make it possible to assess the heritability of abdominal fatness and also to discover whether genetic influences are unique or common to BMI. High heritability of other adiposity phenotypes [eg, truncal skinfold thickness, percentage body fat, and waist circumference (WC)] has been reported in adults (17), and associations with BMI have implicated both common and unique genetic determinants (18). No large twin study has examined the heritability of abdominal adiposity in children since the prevalence of that condition began to spiral upward. We quantified the genetic and environmental influences on BMI and WC and assessed the genetic and environmental overlap between the 2 variables in a population-based sample of 5092 twin pairs born between 1994 and 1996.

SUBJECTS AND METHODS

Participants

The sampling frame was the Twins' Early Development Study (TEDS), a population-based cohort of twins born in the United Kingdom in 1994, 1995, and 1996. The TEDS cohort is reasonably representative of population demographics and is described in more detail elsewhere (19). Zygosity was assessed through a parent questionnaire of physical similarity, which has been shown to be >95% as accurate as DNA testing (20). When zygosity was unclear from the questionnaire, DNA testing was conducted. The results of Koeppen-Schomerus et al (10), described above, were from a subsample of TEDS families when the twins were 4 y old.

Procedures

For the present study, carried out in 2005, parents were invited to weigh and measure their children. They were sent a tape measure for WC and height and were instructed to take the waist measurement directly over the skin at a point 4 cm above the navel (bellybutton) while the child was relaxed and after a slight exhalation (21). Weights, heights, and waists were also measured by researchers visiting the homes in a subsample of 228 children within a year of the parents' return of the questionnaire (mean: 5 mo). Correlations between researcher-measured and parent-measured heights, weights, and waists were 0.90, 0.83, and 0.92, respectively. On average, the measures taken by researchers showed children to be 1.7 cm taller and 2.6 kg heavier than the parental measurements found, and those values gave a BMI that was 0.9 higher. WCs measured by the researchers were on average 0.78 cm larger than parental measures. These differences were probably the result of a time lag between the 2 measures.

BMI and WC SD scores (SDSs) were calculated by using the EXCEL GROWTH MACRO software (version 2.12; Microsoft Corp, Redmond, WA) for the 1990 British growth reference curves, which have a mean of 0 and an SD of 1 at each age (22, 23). Overweight and obese status was determined by using the International Obesity Task Force criteria, which identify BMI values for each age associated with predicted BMIs of 25 and 30 at age 18 y (24).

The request for information on weights and heights was sent to 8978 families who were active participants in TEDS at the time

of data collection; of this group, 5543 (62%) returned completed questionnaires. The remaining 3234 families were not currently active, and only 359 of them returned completed questionnaires. Thus, the total sample comprised 5902 families. Excluded from the analyses were families in which either twin had a specific medical condition or was an extreme outlier for perinatal problems (eg, very low birth weight) or for whom zygosity information was unavailable. Criteria for raw data cleaning were based on the ranges of measured heights and weights from the Health Survey for England 2003 (Internet: www.archive2.official-documents.co.uk/document/deps/doh/survey03/hse_03.htm). Children with height <1.10 m or with weight <13 kg or >84 kg were excluded. When BMI was calculated from the cleaned data, we also excluded children with a BMI < 12. After exclusions, complete BMI and WC data were available for 5092 pairs of twins: 1813 monozygotic (845 M, 968 F) and 3279 dizygotic (818 M, 840 F; $n = 1621$ opposite-sex) pairs.

Each child's parents provided written informed consent. The study was approved by the ethics committees of King's College London and University College London.

Statistical analysis

The twin method depends on comparing the phenotypic similarity of genetically identical (monozygotic) and fraternal (dizygotic) twin pairs. Differences in within-pair correlations between monozygotic and dizygotic twin pairs give an estimate of the contribution of inherited genetic differences to phenotypic variation; the remaining variation is attributed to environmental differences. To the extent that within-pair correlations are higher than would be predicted from the heritability of the trait, shared-environment effects are implicated. The remaining environmental variance is therefore a nonshared-environment effect plus errors of measurement.

Quantitative genetic model fitting is standard in twin studies; it has been described elsewhere (25). Observable variation is decomposed into dominant and additive genetic components and shared and nonshared environmental components. Removal of each component in turn and testing of the deterioration in the fit of the model relative to the full model allows the identification of the best-fitting and most parsimonious model. We used MX software for structural equation modeling (26) to test the fit of the models to the data and to obtain CIs for estimates of genetic and environmental effects (25, 27). A sex-limitation model was used that examined quantitative differences in variables estimates between boys and girls and qualitative differences in parameter between same-sex and opposite-sex twin pairs (26). For the analysis of covariance between BMI and WC, standard bivariate quantitative genetic model-fitting techniques were used to decompose the phenotypic covariance into genetic and environmental components of covariance (25).

RESULTS

The mean age of the twins at the time of measurement was 9.9 y (range: 8.3–11.6 y). Mean heights, weights, BMI, and WC and the respective SDs, which showed the comparison with the 1990 norms, are given in **Table 1**. Heights were 0.22 SDSs higher and weights 0.14 SDSs higher than 1990 norms, but BMI was close to the 1990 levels (0.02 SDSs). WCs were substantially higher than 1990 norms (0.80 SDSs), and more so in girls (0.87 SDSs) than boys (0.74 SDSs). Overweight and obesity rates

TABLE 1
Anthropometric measures¹

	All (n = 10 184)	MZall (n = 3626)	DZall (n = 6558)	DZss (n = 3316)	DZos (n = 3242)	MZM (n = 1690)	MZF (n = 1936)	DZM (n = 1636)	DZF (n = 1680)
Height (cm)	139.44 ± 8.24 ²	139.05 ± 8.21	139.65 ± 8.25	139.61 ± 8.31	139.71 ± 8.19	139.14 ± 7.79	138.97 ± 8.56	140.06 ± 7.99	139.18 ± 8.59
Height SD score	0.22 ± 1.04	0.15 ± 1.03	0.25 ± 1.05	0.23 ± 1.05	0.27 ± 1.05	0.19 ± 1.00	0.11 ± 1.05	0.28 ± 1.02	0.19 ± 1.08
Weight (kg)	33.63 ± 7.89	33.24 ± 7.49	33.85 ± 8.09	33.89 ± 8.14	33.81 ± 8.03	32.83 ± 6.96	33.60 ± 7.92	33.41 ± 7.49	34.35 ± 8.71
Weight SD score	0.14 ± 1.06	0.08 ± 1.03	0.17 ± 1.07	0.16 ± 1.09	0.17 ± 1.06	0.12 ± 1.02	0.05 ± 1.05	0.19 ± 1.04	0.13 ± 1.13
BMI (kg/m ²)	17.17 ± 2.93	17.08 ± 2.81	17.23 ± 2.99	17.25 ± 2.98	17.21 ± 3.01	16.85 ± 2.45	17.28 ± 3.07	16.97 ± 2.65	17.52 ± 3.24
BMI SD score	0.02 ± 1.17	-0.02 ± 1.14	0.04 ± 1.18	0.04 ± 1.19	0.03 ± 1.17	0.01 ± 1.13	-0.04 ± 1.14	0.05 ± 1.15	0.03 ± 1.23
Waist circumference	62.40 ± 7.08	62.15 ± 6.77	62.55 ± 7.25	62.70 ± 7.45	62.40 ± 7.03	62.28 ± 6.44	62.03 ± 7.04	62.68 ± 6.81	62.71 ± 8.03
Waist SD score	0.80 ± 0.99	0.78 ± 0.98	0.82 ± 1.00	0.83 ± 1.02	0.81 ± 0.98	0.71 ± 0.94	0.83 ± 1.01	0.76 ± 0.92	0.90 ± 1.10
Overweight (%)	11.4	10.3	12.0	12.3	11.7	8.9	11.6	9.2	15.4
Obese (%)	2.9	2.4	3.1	3.3	2.9	1.5	3.2	2.6	4.0

¹ MZall, monozygotic twins; DZall, dizygotic same-sex and opposite-sex twins; DZss, same-sex dizygotic twins; DZos, opposite-sex dizygotic twins; MZM, monozygotic male twins; MZF, monozygotic female twins; DZM, dizygotic male twins; DZF, dizygotic female twins.
² $\bar{x} \pm$ SD (all such values).

(Internal Obesity Task Force criteria) were similar in boys and girls: $\approx 11\%$ of the subjects were overweight, and an additional 3% were obese (Table 1). Twins from dizygotic pairs were significantly taller than those from monozygotic pairs, but the difference was very small (0.58 cm; $t = 3.40$, $P = 0.001$). Dizygotic twins had significantly higher BMIs (difference = 0.15; $t = 2.46$, $P = 0.014$) and WCs (difference = 0.40, $t = 2.72$, $P = 0.007$) than did monozygotic twins.

Twin correlations for BMI SDSs and WC SDSs are shown separately by sex in **Table 2**. The monozygotic correlations were similar in boys and girls and greatly exceeded those of the dizygotic twins, which suggested a strong genetic influence. Doubling the difference between the monozygotic and the dizygotic correlations to estimate heritability indicated substantial genetic influence on BMI scores (74%) and WC (74%).

Model-fitting results (**Table 3**) confirmed these findings. The null model that combines estimates across sexes yielded heritability estimates of 77% for BMI and 76% for WC. Shared-environment effects were 10% and 10%; nonshared-environment effects were 13% and 14%. Sex-limitation model-fitting results shown in Table 3 indicate no major quantitative or qualitative differences between boys and girls. Variable estimates were similar for boys and girls and showed overlapping confidence limits, which indicated that the differences were not significant. The sex-limitation model's comparison of results for same-sex and opposite-sex twin pairs yields a genetic correlation (r_G) and a shared-environment correlation (r_C) between the sexes that are close to their expected values of 0.50 and 1.0, respectively. The best-fitting model is the common-effects model that allows for quantitative but not qualitative sex differences, but this latter effect is due to the power of the large sample to detect a significant overall difference in the pattern of results for boys and girls, with slightly lower r_C estimates for opposite-sex twins that are due to the slightly smaller correlations for opposite-sex dizygotic twins (0.47 and 0.45) than for same-sex dizygotic twins (0.51 and 0.51), as seen in Table 2.

WC had a correlation of 0.76 with BMI, and the results were similar in girls (0.72) and boys (0.78). Bivariate genetic analyses make it possible to assess the extent to which the phenotypic association is due to common genetic effects acting on both WC and BMI or to additional unique genetic influences on abdominal adiposity. Because the univariate analyses of BMI and WC showed similar results for boys and girls, bivariate analyses are presented for the total sample; the bivariate results were similar for boys and girls (data not shown). The cross-trait correlations between WC SDSs and BMI SDSs were 0.66 for monozygotic twins and 0.36 for dizygotic twins, which yielded a bivariate heritability estimate of 60% for the relation between WC and BMI. Model-fitting results (**Figure 1**) showed that BMI and WC are highly correlated genetically (0.77), which means that the same genes largely affected BMI and WC. The results in Figure 1 show that genetic mediation accounted for three-quarters (0.74; 95% CI: 0.70, 0.78) of the phenotypic correlation between BMI and WC. Nonetheless, 0.30 (0.27, 0.32) of the genetic influence on WC (ie, 40% of its heritability) was independent of genetic influence on BMI. Shared- and nonshared-environment correlations between BMI and WC also were high (0.76 and 0.69, respectively), although they contributed only modestly to the phenotypic correlation between BMI and WC because their contributions to the variance of BMI and WC were small (0.13; 0.09, 0.17 and 0.12; 0.11, 14, respectively).

TABLE 2
Intraclass twin correlations (and 95% CIs) for BMI and waist circumference SD scores¹

Measure	MZall (n = 1813)	DZall (n = 3279)	DZss (n = 1658)	DZos (n = 1621)	MZM (n = 845)	MZF (n = 968)	DZM (n = 818)	DZF (n = 840)
BMI	0.86 (0.85, 0.87)	0.49 (0.47, 0.51)	0.51 (0.48, 0.53)	0.47 (0.45, 0.50)	0.84 (0.83, 0.86)	0.87 (0.86, 0.88)	0.45 (0.41, 0.49)	0.55 (0.52, 0.59)
Waist	0.85 (0.84, 0.86)	0.48 (0.46, 0.50)	0.51 (0.49, 0.54)	0.45 (0.42, 0.48)	0.85 (0.83, 0.86)	0.86 (0.85, 0.87)	0.42 (0.38, 0.46)	0.57 (0.54, 0.60)

¹MZall, monozygotic twins; DZall, dizygotic same-sex and opposite-sex twins; DZss, same-sex dizygotic twins; DZos, opposite-sex dizygotic twins; MZM, monozygotic male twins; MZF, monozygotic female twins; DZM, dizygotic male twins; DZF, dizygotic female twins. All values were significant, $P < 0.001$.

DISCUSSION

These results indicate that adiposity in preadolescent children born since the onset of the obesity epidemic is highly heritable. The heritability of BMI in this sample (77%) is at the higher end of results obtained with large adult samples (4, 7). Heritability of BMI is also slightly higher than was found in a subsample of the same cohort at age 4 y (10). This could be due to the use of weight-for-height as the index of adiposity in the earlier analysis, but it is more likely that genetic effects on BMI increase during early childhood, as has been shown for other traits (28). In a study of twins born in the Netherlands in the 1980s and 1990s (29), heritability at birth was 24%; heritability increased to 55% at age 1 y and to 59% at age 2 y. The present results may indicate a further increase in the genetic effect, but longitudinal studies across the full span of childhood and adolescence are needed before definite conclusions can be reached.

The results in the present study are broadly comparable to findings from earlier cohorts of young adults, which indicates that the balance of genetic and environmental effects is much the same as that before the external environment became so obesogenic. Therefore, although contemporary environments have made today's children fatter than were children 20 y ago, the primary explanation for variations within the population, then and now, is genetic differences between individual children.

This is the first study to assess the heritability of WC in children, an increasingly important issue in the light of evidence that fat in the visceral region is the major cause of metabolic syndrome (30) and is an important contributor to cardiovascular disease (31) and some cancers (32). We found that WC was as heritable as BMI, with comparable contributions of shared- and nonshared- environment effects. The results of the bivariate analysis indicated that $\approx 60\%$ of the heritability of WC was common to BMI, but 40% was due to different genetic factors. The etiologic significance of visceral fat stores, as compared with other fat stores, may therefore be related to different underlying genetic factors.

BMI tends to be lower and obesity tends to be less prevalent in twins than in singletons (11, 33), a difference that may be related to the intrauterine environment or to the effect of growing up as a twin. Heights, weights, and the prevalence of obesity also were lower in the present sample than in 10-y-olds in the Health Survey for England (2003), but it was interesting that WCs in the present sample were as high as those in the surveyed 10-y-olds. However, there is no evidence that these effects differ significantly between monozygotic and dizygotic twins, and therefore the validity of the twin design and of any conclusions related to genetic and environmental effects should be secure.

Probably the most controversial finding from twin studies is the relatively low shared-environment effect, a finding that has been observed for behavioral traits. Discussions about the obesity epidemic almost invariably ascribe a key role to the family, but, in the present study, as in other twin and adoption studies, siblings from the same family were only slightly more similar in adiposity than would be expected from their genetic similarity, and the shared-environment effect was estimated at just over 10%. The fact that siblings' experience of being served similar food, being given the same options for television viewing and active outdoor play, seeing the same behaviors modeled by parents, and going to the same school does not make siblings more similar is a challenge for etiologic models that highlight the home

TABLE 3
Genetic and environmental parameter estimates (and 95% CIs) and fit indexes from a full-sex limitation model and nested submodels¹

Model	-211	$\Delta\chi^2$	Δdf	P	Male			Female		
					a^2	c^2	e^2	a^2	c^2	e^2
BMI										
Full (r_G free)	29381.62	—	—	—	0.78 (0.68, 0.84)	0.08 (0.02, 0.18)	0.14 (0.13, 0.16)	0.72 (0.63, 0.81)	0.16 (0.07, 0.24)	0.12 (0.11, 0.13)
Full (r_C free)	29381.62	—	—	—	0.78 (0.68, 0.84)	0.08 (0.02, 0.18)	0.14 (0.13, 0.16)	0.72 (0.63, 0.81)	0.16 (0.07, 0.24)	0.12 (0.11, 0.13)
Common effects	29381.85	0.22	1	0.64	0.80 (0.72, 0.84)	0.06 (0.01, 0.13)	0.14 (0.13, 0.16)	0.72 (0.63, 0.81)	0.16 (0.07, 0.24)	0.12 (0.11, 0.13)
Scalar	29390.51	8.88	3	0.03						
Null model	29392.53	10.9	4	0.03						
Waist										
Full (r_G free)	26344.19	—	—	—	0.79 (0.69, 0.85)	0.06 (0.01, 0.16)	0.14 (0.13, 0.16)	0.67 (0.59, 0.76)	0.20 (0.11, 0.27)	0.14 (0.12, 0.15)
Full (r_C free)	26344.19	—	—	—	0.79 (0.69, 0.85)	0.06 (0.01, 0.16)	0.14 (0.13, 0.16)	0.67 (0.59, 0.76)	0.20 (0.11, 0.27)	0.14 (0.12, 0.15)
Common effects	26344.49	0.30	1	0.59	0.82 (0.77, 0.86)	0.04 (0.01, 0.09)	0.14 (0.13, 0.16)	0.67 (0.59, 0.75)	0.20 (0.11, 0.28)	0.14 (0.12, 0.15)
Scalar	26352.06	7.87	3	0.05						
Null model	26370.50	26.3	4	0.00						

(Continued; additional data columns shown below)

Model	Both sexes					r_G	r_C	s^2_m	s^2_f
	a^2	c^2	e^2						
BMI									
Full (r_G free)						0.48	1.00	1.18	1.18
Full (r_C free)						0.50	0.86	1.18	1.18
Common effects						0.50	1.00	1.18	1.18
Scalar	0.77 (0.72, 0.82)	0.10 (0.05, 0.15)	0.13 (0.12, 0.14)			0.50	1.00	1.19	1.17
Null model	0.77 (0.72, 0.82)	0.10 (0.05, 0.15)	0.13 (0.12, 0.14)			0.50	1.00	1.18	1.18
Waist									
Full (r_G free)						0.47	1.00	0.96	1.03
Full (r_C free)						0.50	0.79	0.96	1.03
Common effects						0.50	1.00	0.96	1.03
Scalar	0.77 (0.72, 0.82)	0.10 (0.04, 0.14)	0.14 (0.13, 0.15)			0.50	1.00	0.97	1.03
Null model	0.76 (0.71, 0.81)	0.10 (0.05, 0.15)	0.14 (0.13, 0.15)			0.50	1.00	1.00	1.00

¹ -211, twice the log likelihood ratio chi-square; $\Delta\chi^2$, change in chi-square test from the full sex-limitation model; Δdf , change in degrees of freedom between comparison models; P , significance of reduced models compared with the full model; a^2 , c^2 , and e^2 , additive genetic, shared-environment, and nonshared-environment estimates; r_G , genetic correlation for opposite-sex dizygotic twins; r_C , shared-environment correlation for opposite-sex twins; s^2_m , predicted variance in males, s^2_f , predicted variance in females. The full sex-limitation model allows for different estimates of a^2 , c^2 , and e^2 for boys and girls (quantitative sex differences); the results show that the parameter estimates for boys and girls are well within their CIs. The full model also allows qualitative sex differences in that the genetic correlation (r_G) for opposite-sex twins can be less than the expected 0.50 for siblings if genetic effects differ qualitatively for boys and girls. Similarly, the shared-environment correlation (r_C) for opposite-sex twins can be less than the expected 1.0 for siblings reared together if shared-environmental effects differ for boys and girls. The results for 2 full models are shown, one with r_G free and the other with r_C free, because only one of these parameters can be freed at a time in the full model. For BMI, when r_G is free, the estimate is 0.48, very close to the expected 0.50. When r_C is free, the estimate is 0.86, somewhat less than the expected 1.0, which largely derives from opposite-sex DZ correlations (0.47 and 0.45) that are slightly lower than same-sex DZ correlations (0.51 and 0.51). The common-effects sex-limitation model allows quantitative sex differences but not qualitative differences; by constraining r_G to 0.5 in opposite-sex dizygotic twins, this model tests whether qualitative sex differences are significantly different from those in the full model. This is the best-fitting model in that the change in chi-square ($\Delta\chi^2$) with 1 df (Δdf) yields the largest P value ($P = 0.586$ for BMI), which indicates that the data differ least from this model and that qualitative sex differences are significant. The second nested model, the scalar sex-limitation model, tests for quantitative sex differences by constraining a^2 , c^2 , and e^2 to be equal for boys and girls and also constrains r_G to 0.5 but allows variances to differ. This model could be rejected by BMI and 0.05 for waist circumference), as could the null model, which equates all parameters including variances and thus tests for variance differences between boys and girls.

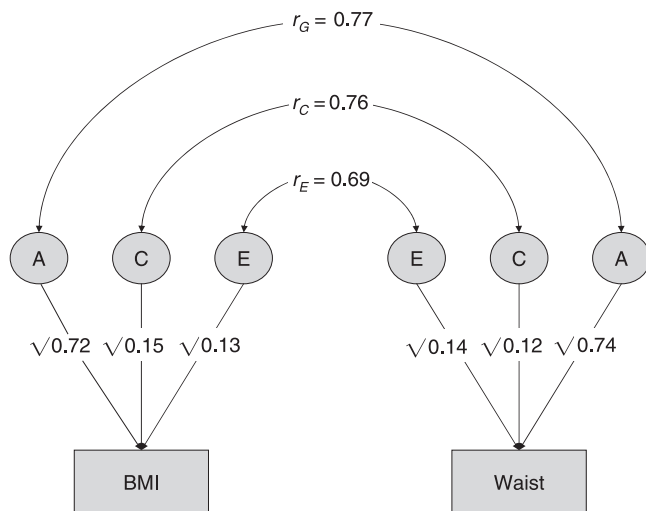


FIGURE 1. Estimates of genetic and environmental correlations between BMI and waist circumference. The 95% CIs for the variable estimates from the correlated-factors model-fitting solution are $r_G = 0.77$ (0.75, 0.79); $r_C = 0.76$ (0.64, 0.85); $r_E = 0.69$ (0.66, 0.71). The A, C, and E estimates are similar to those from the univariate model-fitting (Table 3), but they are not identical because the bivariate analysis includes the variance of BMI and waist circumference as well as the covariance between them.

environment as the root cause of obesity. This finding will, however, come as no surprise to parents, who are well aware that their children come in different shapes and sizes despite having a similar upbringing. What is important is this finding means that “blaming” parents is wrong. Findings from twin studies were influential in persuading clinicians that the “schizophrenogenic” mother was a myth. Results from the present study highlight the fact that excessive weight gain in a child is unlikely to be the fault of the parents and is more likely to be due to the child’s genetic susceptibility to the obesogenic features of the modern environment.

Does the fact that the shared-environment effects were comparatively small have implications for the potential effect of interventions that target the home? It certainly counsels caution against assumptions that, if all parents followed current child-feeding recommendations, the obesity problem would be solved. But etiologic processes do not always have simple indications for interventions. Strongly genetic conditions—notably, phenylketonuria—have proved to be entirely treatable by environmental interventions, eg, a phenylalanine-free diet in the case of phenylketonuria. It is therefore possible that aspects of family life could be modified enough to achieve a protective environment for children who are vulnerable to obesity. It is also worth remembering that control over 10% of the variance in an important health risk factor is not insignificant. Population-attributable risks for many of the risk factors for coronary heart disease are <10%, yet recommendations for behavior change are a central part of prevention and management (34). Nevertheless, these results signify that achieving major shifts in population weights—as proposed in recent public health targets—will require at least as much emphasis on creating healthier external environments and teaching vulnerable persons to adopt life-long prudent habits as on encouraging parents to modify home settings.

The present study had limitations. The twin method makes several assumptions, such as that monozygotic and dizygotic

twins have similar environments. These assumptions have been discussed in detail elsewhere (25, 35, 36). To the extent that the environments (uterine or familial) of monozygotic twin pairs are more similar than those of dizygotic pairs, heritability estimates from twin studies will be inflated. However, existing evidence suggests that this effect is likely to be small and that it would not materially change the conclusion that phenotypic variation in adiposity is significantly determined by heritable genetic differences between persons. There is also the potential for bias in volunteer samples, despite a population-based sampling frame, although this potential is common to all epidemiologic studies that depend on voluntary participation. If the participation bias is unrelated to the trait, it may not matter, but overweight families may be reluctant to participate in a study requiring weight reports. However, so long as the volunteer bias is the same in families with monozygotic and dizygotic twins, the twin comparisons remain valid. In common with many large-scale anthropometric studies, the present study used parental reports of the height, weight, and WC of the children. However, we gave careful guidance on how to take the measurements and showed high correlations between parental reports and all 3 measures in a subsample of families visited at home, which provides confidence in the results.

Quantitative genetic studies indicate how much of the variation in weight is due to genetic differences between persons, but they neither identify the genes nor address their mechanisms. It appears increasingly likely that weight variation is due to large numbers of genes, each exerting small effects, because no major genes for common obesity have been identified (37). Part of the genetic effect may well be due to variations in appetite and satiety and not just to the biology of fat storage (38). A quantitative, behavioral, genetic model helps make sense of the paradox that obesity is both predominantly environmental (as in the rapid secular increases) and predominantly genetic (as in quantitative genetic studies). In such a model, the epidemic of obesity is attributed squarely to changes in the environment, whereas individual differences are attributed to genetic differences between individual persons.

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