THE BODY-MASS INDEX OF TWINS WHO HAVE BEEN REARED APART

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Abstract To assess the relative importance of genetic and environmental effects on the body-mass index (weight in kilograms divided by the square of the height in meters), we studied samples of identical and fraternal twins, reared apart or reared together. The samples consisted of 93 pairs of identical twins reared apart, 154 pairs of identical twins reared together, 218 pairs of fraternal twins reared apart, and 208 pairs of fraternal twins reared together.

The intrapair correlation coefficients of the values for body-mass index of identical twins reared apart were 0.70 for men and 0.66 for women. These are the most direct estimates of the relative importance of genetic influences (heritability) on the body-mass index, and they were only slightly lower than those for twins reared together in this

DECENT studies have established the influence of Regenetic factors in human obesity.^{1,2} Adoption studies have revealed that the body-mass index (weight in kilograms divided by the square of the height in meters) of adoptees resembles that of their biologic parents^{3,4} and siblings,⁵ but not that of their adoptive parents. However, the extent of the genetic contribution is uncertain. In studies of twins reared together,^{6,7} the genetic contribution to the body-mass index has been estimated to be 64 to 84 percent, but these values may overestimate the contribution of heredity because similarities in twins reared together may result from shared environments as well as shared genes.

The study of identical twins reared apart is generally considered to be one of the most effective designs for distinguishing the importance of shared genes from that of shared environments. We studied a sample of 93 pairs of identical twins reared apart, 154 pairs of identical twins reared together, 218 pairs of fraternal twins reared apart, and 208 pairs of fraternal twins reared together. All were participants in the Swedish Adoption/Twin Study of Aging (SATSA).8,9 This unique sample makes possible an analysis that combines the adoption and twin designs, thus avoiding many of the limitations of the classic twin method and allowing a more accurate estimate of genetic contributions. We present here such an analysis of genetic contributions to the body-mass index.

and earlier studies. Similar estimates were derived from maximum-likelihood model-fitting analyses - 0.74 for men and 0.69 for women. Nonadditive genetic variance made a significant contribution to the estimates of heritability, particularly among men. Of the potential environmental influences, only those unique to the individual and not those shared by family members were important, contributing about 30 percent of the variance. Sharing the same childhood environment did not contribute to the similarity of the body-mass index of twins later in life.

We conclude that genetic influences on body-mass index are substantial, whereas the childhood environment has little or no influence. These findings corroborate and extend the results of earlier studies of twins and adoptees. (N Engl J Med 1990; 322:1483-7.)

Methods

The SATSA sample of twins separated early in life and matched twins reared together was identified through the Swedish Twin Registry, which includes nearly 25,000 pairs of twins of the same sex born in Sweden between 1886 and 1958.¹⁰ The zygosity of the twins was determined according to criteria of physical similarity, the accuracy of which was established by Cederlöf et al.¹¹ in a subsample from the twin registry and later confirmed by Sarna.¹² The mean age (\pm SD) of the twins we studied was 58.6 \pm 13.6 years, with an approximately normal distribution around the sixth decade of life; 2 percent of the pairs were more than 80 years old, 18 percent were 71 to 80, 31 percent were 61 to 70, 20 percent were 51 to 60, 18 percent were 41 to 50, 9 percent were 31 to 40, and 2 percent were 21 to 30. Sixty percent were women, which conforms to the sex ratio for the last half of the life span. The SATSA sample is representative of twins in the Swedish Twin Registry except in age. Those in the sample are older, because most of the twins reared apart were born during the first three decades of the century, when economic depression and epidemics increased the likelihood of separation.

The identification of the SATSA sample of twins reared apart and matched twins reared together has been described by Pedersen, McClearn, and their colleagues.^{8,9} The average age at separation was 2.8 years; 48 percent of the pairs were separated during the first year of life, and 82 percent by the age of 5. The reasons for separation varied, the most common being the illness or death of one or both parents and economic hardship. Age at separation and the degree of separation of the twins had no effect on their degree of similarity in either height or weight.9 Twins separated earlier were not less similar than twins separated later in childhood.

Heights and weights were reported by the twins themselves on questionnaires administered in the fall of 1984. Height and weight were also measured in a sample of 588 of the 1346 twins in this study. The correlation coefficient between measured values and those reported by the twins themselves was 0.97 for height and 0.95 for weight, and the mean differences (±SD) between reported and measured values were 1.2±2.4 cm for height and 0.8±4.0 kg for weight.

Analyses were performed to evaluate the roles of genetic and environmental determinants of total phenotypic variance. They consisted of intrapair correlations and maximum-likelihood modelfitting analyses.

Intrapair Correlations

The proportion of total variance attributable to genetic influences (heritability¹³) was estimated from intrapair correlations in four wavs.

In the classic twin method, the difference between the intrapair correlations of monozygotic and dizygotic twins reared together is doubled. This estimate of heritability includes both the additive and nonadditive components of genetic variance. The additive

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The sample we report on is from the Swedish Adoption/Twin Study of Aging, being conducted at the Department of Environmental Hygiene of the Karolinska Institute in Stockholm (coinvestigators, Drs. Nancy L. Pedersen, Lars Friberg, Ulf DeFaire, and Stig Berg) in collaboration with the Center for Developmental and Health Genetics at Pennsylvania State University (coinvestigators, Drs. Gerald E. McClearn, Robert Plomin, and J.R. Nesselroade).

component is the sum of the average effects of all the genes that influence a character and thus "breed true." Nonadditive effects result from interactions between alleles at a locus (dominance) or among genes at different loci (epistasis). Monozygotic twins share all genetic effects, additive and nonadditive, whereas dizygotic twins share 50 percent of the additive effects and 25 percent of the nonadditive effects.¹³ Thus, if nonadditive effects are important for a trait, the expected correlation for dizygotic twins is less than half that for monozygotic twins and procedures that compare monozygotic and dizygotic correlations will overestimate heritability.

The SATSA sample of twins reared apart made possible three additional estimates of heritability. In the first, the difference between the intrapair correlations for monozygotic and dizygotic twins reared apart was doubled. When nonadditive variance is present, this method also overestimates heritability, but it avoids other problems intrinsic to the classic twin method, as will be discussed below. The second estimate was obtained by doubling the intrapair correlation for dizygotic twins reared apart. This method underestimates heritability in the presence of nonadditive variance. Third, the intrapair correlation for monozygotic twins reared apart in uncorrelated environments constitutes an unbiased estimate of heritability, even if nonadditive variance is present.

The separated-twin method makes it possible to estimate not only genetic effects but also three different environmental effects -- the shared rearing environment, other correlated environments, and nonshared environments that are unique to the individual.

In studies of twins reared together, estimates of the influence of a shared environment are derived from resemblances that cannot be explained by genetic effects. Such studies cannot separate the relative contributions of two different environments --- that in which the twins were raised (the shared rearing environment) and that in which they lived after they left home (the correlated environment). The study of twins reared apart makes it possible to estimate the effects of the rearing environment; the resemblances between twins reared together are compared with those between twins reared apart.

Even twins reared apart could resemble one another for environmental reasons if their environments were similar - that is, if the environments were correlated. All resemblances between twins that are not attributable to either heredity or the shared rearing environment may be attributed to correlated environments. For example, a correlated environment may be the result of "selective placement" placing adopted children in homes that resemble those of their biologic parents - or of contact between the twins in adulthood. The effects of correlated environments can be estimated by subtracting the estimate of heritability from the correlation for monozygotic twins. When applied to twins reared apart, this calculation includes the effects of selective placement.

Residual variance that cannot be explained by heredity, the shared rearing environment, or correlated environments is attributed to the effects of a nonshared environment that is unique to the individual.

Model-Fitting Analyses

Correlations for the four groups of twins permitted us to estimate the contribution to variability in the body-mass index of genetic variance, variance due to a shared rearing environment, and variance due to correlated environments. These estimates are not independent; they should be regarded as providing the range of possible values that can be obtained from this sample. Model fitting represents a more powerful approach, for it is based on a simultaneous solution of all the information used in the other four methods, makes assumptions explicit, and permits testing of the relative fit of different models. The results of model fitting complement the intrapair correlations, confirming estimates that were based on comparisons of correlations

Our model was derived from the standard biometric model of Jinks and Fulker for a twin design that fits three parameters: heritability, shared environment, and nonshared environment.¹⁴ It was extended to partition heritability into additive and nonadditive components and to distinguish between the presence of shared rearing and correlated environments. Expected and observed mean squares were used to derive the estimates of interest. Maximumlikelihood estimates and standard errors were obtained with use of the LISREL-VI program for linear structural-equation modeling.¹ The model as it is applied to the SATSA sample has been described more fully elsewhere.^{16,17}

Description of the Sample

The mean $(\pm SD)$ values for body-mass index are listed in Table 1. Univariate analyses for sex and age (in five-year groups) yielded small but statistically significant differences for three effects: rearing, age, and sex. Twins reared apart were slightly heavier ($F_{1,136} = 14.52$, P<0.01), as were older twins ($F_{10,1441} = 8.91$, P<0.01) and men ($F_{1,1441} = 4.40$, P<0.05).

In addition to these mean differences, the variance in body-mass index was greater (P<0.001) for women (SD, 3.7) than men (SD, 2.9). For this reason and because adoption studies had shown differing heritability according to sex,3,4 analyses were performed separately for men and women. We adjusted for the effect of age on body-mass index by using the residuals from a regression of body-mass index on age.¹⁸

RESULTS

Intrapair Correlations

The heritability of the body-mass index was estimated by each of the four correlational methods outlined above as well as by model-fitting approaches. The intrapair correlations according to zygosity and sex are listed in Table 1, and the estimates of heritability derived from them in Table 2. These estimates indicate a substantial genetic contribution to the body-mass index.

The first estimate of heritability, based on the classic twin design, was 0.82 for men and 0.78 for women. The intrapair correlation of both male and female monozygotic twins was more than twice that of the dizygotic twins, implying the presence of nonadditive genetic effects and thus an overestimate of heritability.

The second estimate of heritability, derived from correlations of monozygotic and dizygotic twins reared apart, was also high. It too probably represents an overestimation because of the presence of nonadditive variance. The third estimate of heritability, twice the correlation for dizygotic twins reared apart, was the smallest of the four. In the presence of nonadditive variance it represents an underestimate and sets a lower limit for the heritability of the body-mass index.

The fourth estimate of heritability, the intrapair correlation for the monozygotic twins reared apart, was the most direct and perhaps the best estimate of the heritability of the body-mass index. It was 0.70 for men and 0.66 for women. Note that these values are approximately halfway between those estimated by the classic twin method, which in the presence of nonadditive genetic variance probably overestimates heritability, and the separated-dizygotic-twin method, which in these circumstances probably underestimates it.

The four estimates of heritability were similar for men and women (Table 2). The pattern of correlations shown in Table 1, however, suggests a larger role for nonadditive genetic effects among men.

Three types of environmental effects were assessed. We estimated the effect of a shared rearing environment by comparing the intrapair correlations for twins

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Table 1. Body-Mass Index and Intrapair Correlations in Monozygotic and Dizygotic Pairs of Twins Reared Apart or Together.*

Түре	Men			Women			
	NO. OF PAIRS	BODY- MASS INDEX	INTRAPAIR CORRELATION	NO. OF PAIRS	BODY- MASS INDEX	INTRAPAIR CORRELATION	
Monozygotic							
Reared apart	49	24.8 ± 2.4	0.70	44	24.2 ± 3.4	0.66	
Reared together	66	24.2 ± 2.9	0.74	88	23.7±3.5	0.66	
Dizygotic							
Reared apart	75	25.1 ± 3.0	0.15	143	24.9 ± 4.1	0.25	
Reared together	89	24.6±2.7	0.33	119	23.9±3.5	0.27	

*Plus-minus values are means ±SD.

reared together with those for twins reared apart. Table 1 shows that twins reared together were no more similar than those reared apart, thus ruling out any effect of a shared rearing environment on the bodymass index.

We estimated the effect of similar (correlated) environments by subtracting the estimate of heritability from the correlation for monozygotic twins. Heritability estimates were actually larger than the correlations for monozygotic twins, both for those reared together and those reared apart. Correlated environments evidently had no effect on the body-mass index, providing further confirmation that sharing similar environments had no effect and ruling out any effect of selective placement.

Residual variance that cannot be explained by either genetic or shared environmental influences is attributable to nonshared variance unique to the individual person. In respect to the body-mass index, all environmental variance was of this type, accounting for about 30 percent of total variance.

Model-Fitting Analyses

We tested differences between the sexes in genetic and environmental influences by comparing the relative fits of two models. The first model allowed us to derive estimates separately for each sex, whereas the second model constrained the estimates to be equal across the two groups. The difference between the models was highly significant ($\chi^2 = 47.48$, 3 df; P<0.001), indicating that the estimates of genetic and environmental influences were different for men and women. Accordingly, the remaining analyses were performed separately for men and women.

The full model, which included additive and nonadditive genetic variance, shared rearing environment, and nonshared environment, provided a satisfactory fit to the data for men ($\chi^2 = 6.60$, 4 df; P = 0.159) but not women ($x^2 = 11.35$, 4 df; P = 0.023). The poor fit of the model to the data for women may reflect the fact that the variances among women were not homogeneous across rearing and zygosity groups; those among dizygotic twins reared apart were greater. Consistent with the information from the intrapair correlations, the estimates for shared rearing environment and correlated environments accounted for less than 1 percent of the variance.

Estimates for a reduced model from which shared rearing environment and correlated environments were excluded are shown in Table 3. We calculated the proportion of the total variance explained by each parameter by dividing the square of the estimate of that parameter by the sum of the squares of the other estimates. The resulting estimates of heritability were very similar to those yielded by the correlational analyses — 74 percent for men and 69 percent for women. Furthermore, they permitted us to estimate the extent of nonadditive variance — 57 percent for men and 37 percent for women (Fig. 1 and Table 3). Additive genetic variance was 17 percent for men and 32 percent for women. The remaining variance (26 percent for men and 31 percent for women) can be attributed to nonshared environmental influences unique to the individual.

The relative importance of additive and nonadditive genetic variance was further assessed by procedures that tested whether dropping either parameter significantly reduced the fit of the model. Dropping additive genetic variance did not significantly change the χ^2 for either men or women. Dropping nonadditive variance, however, resulted in a significant change for men (change in χ^2 , 4.29, 1 df; P = 0.04) but not women. This more rigorous test confirmed the significant contribution of nonadditive genetic variance among men.

Despite this difference in variance between the sexes, it is important to note that the relative contributions of genes and environment to the body-mass index of men and women were quite similar. Although body-mass index appeared to be more variable among women, this greater variance did not result from either shared rearing environments or correlated environments.

DISCUSSION

Three principal findings from this study have advanced our understanding of the determinants of the body-mass index.

The first was the strong evidence of the influence of heredity on the body-mass index. The intrapair correlations of the 93 pairs of monozygotic twins reared apart provided estimates of genetic influences that were independent of environmental contributions. These values — 0.70 for men and 0.66 for women may be the best available estimates of total genetic

Table 2.	Heritability Accord	ling to Sex, as	Estimated
	by Four N	lethods.	

Method*	CALCULATION [†]	ESTIMATE OF HERITABILITY		
		MEN	WOMEN	
1	$2(\mathbf{r}_{MZT} - \mathbf{r}_{DZT})$	0.82	0.78	
2	$2(r_{MZA} - r_{DZA})$	1.10	0.82	
3	$2(r_{DZA})$	0.30	0.50	
4	MZA	0.70	0.66	

*See text for an explanation of the methods.

 $^\dagger\text{MZ}$ denotes monozygotic, DZ dizygotic, A reared apart, and T reared together.

Table 3. Parameter Estimates for Maximum-Likelihood Model-Fitting Analyses.*

	Additive Genetic Variance	Nonadditive Genetic Variance	Nonshared Environment	Chi- Square	Degrees of Freedom	P Value
Men						
Body-mass index	0.34 ± 0.30	0.62±0.16	0.42 ± 0.03	6.64	5	0.249
Variance (%)	17	57	26			
Women						
Body-mass index	0.61±0.22	0.65±0.21	0.60 ± 0.04	11.35	5	0.045
Variance (%)	32	37	31			

*Plus-minus values are means \pm SE. Estimates were squared before the percentage of total variance was calculated. A nonsignificant P value indicates that the model adequately fits the data.

influence on the body-mass index. They are very similar to those obtained from the more powerful modelfitting analyses that combined data from twins reared together and twins reared apart --- 0.74 and 0.69 for men and women, respectively.

Reassurance as to the representativeness of the SATSA sample is provided by its remarkable similarity to other large samples. In the sample of middle-aged American men referred to above (4071 pairs of twins),⁷ the mean (\pm SD) body-mass index was 24.9 \pm 2.7 among the monozygotic twins and 24.8 \pm 2.7 among the dizygotic twins. The estimates of heritability yielded by the classic twin method in the two samples were also quite similar — 0.84 for the American men and 0.81 for the Swedish men. The values for body-mass index in both the SATSA and the American samples were very similar to those obtained in Waaler's population-based survey of 1.8 million Norwegians — 24.9 \pm 2.8 for men 40 to 44 years old and 25.3 \pm 3.2 for men 60 to 64 years old.¹⁹

The second finding of this study was the indication of significant nonadditive genetic variance. The intrapair correlations of monozygotic twins were more than twice those of dizygotic twins, as was the case among the older American twins.⁶ The model-fitting

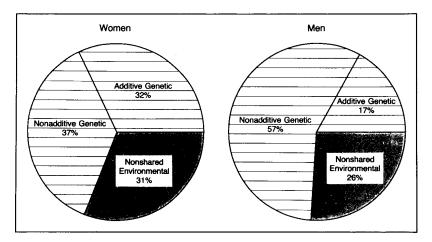


Figure 1. Genetic and Environmental Components of Variance in Body-Mass Index. The contributions of shared and correlated environments each account for less than 1 percent of the variance among men and women.

analyses further supported the importance of nonadditive genetic effects, particularly in men.

The fact that there was significant nonadditive variance in a sample of older Swedish twins raises the question of whether such variance can be attributed to ethnicity or age. The marked similarity of the Swedish twins to the American twins should allay concern about the effects of ethnicity. The cross-sectional nature of this study made it impossible to assess the effects of age. Again the study of the American twins is useful, for they were measured twice — once at the age of 20 and again at 45. At 20 there was no evidence of nonadditive variance, but at 45 there was. Nonadditive variance apparently exerts its effects only later in life.

The third finding of this study is that neither the shared rearing environment nor correlated environments contributed to variation in the body-mass index. This finding is supported by two lines of evidence. First, among the SATSA twins the intrapair similarity in height and weight was not influenced by either age at separation or degree of separation.⁹ Second, adoption studies have not found any effect of the childhood rearing environment.^{3,4} Estimates of the heritability of the body-mass index that are based on data from family members who have shared the same environments (twins reared together or parents and offspring) do not appear to have been biased by the effects of shared or correlated environments. The significant environmental influences on body-mass index are not attributable to shared family influences but are unique to the individual person.

Like the studies described above, our study assessed genetic influences on the body-mass index. Unlike those studies, however, ours involved few frankly obese persons. The relevance of these results to obesity thus depends on how the middle range of values for body-mass index relates to the extreme that characterizes obesity. In this regard it is reassuring that three

> adoption studies have found that genetic influences extend across the range of weight from thin to very obese.³⁻⁵

> The values for height and weight that we used were based on selfreports rather than direct measurements; the latter are clearly preferable. Self-reported heights and weights, however, are often quite accurate and are increasingly used in epidemiologic studies if direct measurements are not available.²⁰⁻²⁵ The self-reported values used in our study corresponded closely to measured values. In a sample of more than one third of the twins, the correlations between self-reported and measured height and weight were very high. It

should be noted that estimates of genetic influence in twin studies are not affected by over- or underestimates of height and weight unless there is a differential bias in reporting between monozygotic and dizygotic twins. There was no evidence of such bias.

The results of this study should be interpreted in the light of the concept of heritability. Heritability does not imply an invariant, immutable genetic influence such as occurs in the case of hair or eye color. It describes instead the genetic influences found among persons living in a particular range of environmental conditions. Under different environmental conditions, different estimates of heritability might be obtained. Nevertheless, the conditions in which the subjects of our study live are those of much of Western society, and our results should apply to persons in that society. Similar results have been obtained by other studies in Western society, whether of twins in America^{6,7} or of adoptees in Denmark^{3,5} or America.⁴ Genetic factors appear to be major determinants of the body-mass index in Western society, and they may account for as much as 70 percent of the variance.

References

- Stunkard AJ. Some perspectives on human obesity: its causes. Bull N Y Acad Med 1988; 64:902-23.
- 2. Price RA. Genetics of human obesity. Ann Behav Med 1987; 9:9-14.
- Stunkard AJ, Sørensen TIA, Hanis C, et al. An adoption study of human obesity. N Engl J Med 1986; 314:193-8.
- Price RA, Cadoret RJ, Stunkard AJ, Troughton E. Genetic contribution to human obesity: an adoption study. Am J Psychiatry 1987; 144:1003-8.
- Sørensen TIA, Price RA, Stunkard AJ, Schulsinger F. Genetics of obesity in adult adoptees and their biological siblings. BMJ 1989; 298:87-90.
- Feinleib M, Garrison RJ, Fabsitz R, et al. The NHLB1 twin study of cardiovascular disease risk factors: methodology and summary of results. Am J Epidemiol 1977; 106:284-95.
- Stunkard AJ, Foch TT, Hrubec Z. A twin study of human obesity. JAMA 1986; 256:51-4.

- Pedersen NL, Friberg L, Floderus-Myrhed B, McClearn GE, Plomin R. Swedish early separated twins: identification and characterization. Acta Genet Med Gemellol (Roma) 1984; 33:243-50.
- McClearn GE, Pedersen NL, Plomin R, Nesselroade JR, Berg S, DeFaire U. The Swedish Adoption/Twin Study of Aging: an update. Acta Genet Med Gemellol (Roma) (in press).
- Cederlöf R, Lorich U. The Swedish twin registry. In: Nance WE, Allen G, Parisi P, eds. Twin research: proceedings of the 2nd International Congress on Twin Studies, August 29–September 1, 1977, Washington, D.C. Part B. Biology and epidemiology. New York: Alan R. Liss, 1978:188-95.
- Cederlöf R, Friberg L, Jonsson E, Kaij L. Studies on similarity diagnosis in twins with the aid of mailed questionnaires. Acta Genet Stat Med 1961; 11:338-62.
- Sarna S. Zygosity diagnosis in epidemiological twin studies: by blood markers and by questionnaires. Helsinki, Finland: University of Helsinki Press, 1977.
- Falconer DS. Introduction to quantitative genetics. 2nd ed. New York: Longman, 1981:148-69.
- Jinks JL, Fulker DW. Comparison of the biometrical, genetic, MAVA, and classical approaches to the analysis of human behavior. Psychol Bull 1970; 73:311-49.
- Jöreskog KG, Sörböm D. Lisrel-VI user's guide. Morrisville, Ind.: Scientific Software, 1986.
- Pedersen NL, Gatz M, Plomin R, Nesselroade JR, McClearn GE. Individual differences in locus of control during the second half of the life span for identical and fraternal twins reared apart and reared together. J Gerontol 1989; 44:100-5.
- Pedersen NL, Lichtenstein P, Plomin R, DeFaire U, McClearn GE, Matthews KA. Genetic and environmental influences for type A-like and related traits: a study of twins reared apart and twins reared together. Psychosom Med 1989; 51:428-40.
- McGue M, Bouchard TJ Jr. Adjustment of twin data for the effects of age and sex. Behav Genet 1984; 14:325-43.
- Waaler HT. Height, weight and mortality: the Norwegian experience. Acta Med Scand Suppl 1984; 679:1-56.
- Stunkard AJ, Albaum JM. The accuracy of self-reported weights. Am J Clin Nutr 1981; 34:1593-9.
- Wing RR, Epstein LH, Ossip DJ, LaPorte RE. Reliability and validity of self-report and observers' estimates of relative weight. Addict Behav 1979; 4:133-40.
- Charney E, Goodman HC, McBride M, Lyon B, Pratt R. Childhood antecedents of adult obesity: do chubby infants become obese adults? N Engl J Med 1976; 295:6-9.
- 23. Coates TJ, Jeffrey RW, Wing RR. The relationship between persons' relative body weights and the quality and quantity of food stored in their homes. Addict Behav 1978; 3:179-84.
- Schlichting P, Høilund-Carlsen PF, Quaade F. Comparison of self-reported height and weight with controlled height and weight in women and men. Int J Obes 1981; 5:67-76.
- Palta M, Prineas RJ, Berman R, Hannan P. Comparison of self-reported and measured height and weight. Am J Epidemiol 1982; 115:223-30.

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