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Effects of Body Size and Body Fatness on Left Ventricular Mass in Children and Adolescents:

Project HeartBeat!

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Abstract

Background—Left ventricular mass (LVM) is a strong predictor of cardiovascular disease in adults. Available study findings on effects of body fatness on LVM in children are inconsistent. Understanding the impact of body fat on LVM in children may help prevent excessive LVM through measures to reduce overweight and obesity.

Methods—Healthy children (*n*=678) aged 8, 11, and 14 years at baseline were examined at 4-month intervals for up to 4 years (1991–1995); 4608 valid measurements of LVM were obtained with M-mode echocardiography. A multilevel linear model was used for analysis. The impact of body size was examined by adding separately nine body-size indicators to a basic LVM–gender–age model. The impact of body fatness was tested by introducing four body-fatness indicators into the nine models, yielding 36 models.

Results—All body-size indicators showed strong, positive effects on LVM. In models containing weight or body surface area (measuring both fat-free and fat contributions to body size), additional effects of body fatness were negative; in models containing fat-free mass (FFM) or height (both measuring body size independent of body fat), increased body fatness was related to a significant increase in LVM. For example, in models with FFM as a body-size indicator, a 1-SD increase in percent body fat or fat mass was related to a 5.4- or 7.2-g increase in LVM, respectively.

Conclusions—Effects of body size on LVM attributable to fat-free body mass can be distinguished from those attributable to fat body mass; both are independent, positive predictors, but the former is the stronger determinant. When a body-size indicator not independent of body fat is used as a predictor, effects of FFM and fat mass are forced to relate to the same indicator; because their magnitudes are estimated to be equal, the effect of fat body mass is overestimated. Thus, when an additional body-fatness indicator is included in the prediction of LVM, the additional estimated effect related to the indicator appears to be negative.

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Introduction

Left ventricular mass (LVM) is a major independent predictor of cardiovascular disease (CVD) morbidity and mortality in adults,[1–3] and body size has been recognized[4–8] as the most important determinant of LVM. Recent studies[9–12] showed that among various measures of body size, lean body mass is most closely related to LVM. Obesity is believed to be associated with an increase in LVM, whether hypertension is present or absent, and weight reduction has therefore been suggested as a strategy for preventing or reducing left ventricular hypertrophy.[13–17]

In children, however, studies[7,8,18,19] of the impact of body fatness on LVM have yielded inconsistent results, ranging from a positive independent association to a negative relationship. In most such studies[14–17,20] relating LVM to obesity, body fatness was characterized using the standard formula for BMI (kg/m^2). But because fat mass is included in total body mass in BMI, as defined, use of BMI precludes discriminating any independent effects of body fat on LVM from those of overall body size. Moreover, problems remain when trying to adjust LVM for differences in body size to derive reference standards for normality and to quantify severity of disease status, especially in obese subjects. Criteria for left ventricular hypertrophy based on LVM indexed by body weight or body surface area identified lower LVM index values in overweight than in non-overweight subjects, whereas criteria based on LVM indexed by height[2,7] identified increased LVM index values in overweight subjects.[6,21]

Based on data from Project HeartBeat!, a longitudinal study of CVD risk factors in children and adolescents, the current analysis aimed to (1) explore the independent effect of body fatness on LVM, adjusting for physical growth; (2) assess the influence of various body-size indicators on the estimated effect of body fatness on LVM; and (3) explore different combinations of body-size and body-fatness indicators to determine the most plausible predictors of LVM in children and adolescents.

Methods

Project HeartBeat! was designed to investigate the development of major CVD risk factors during this period of growth and maturation and to examine the determinants of the development of these risk factors.[22,23] The study was approved by the IRBs of the University of Texas Health Science Center at Houston and of Baylor College of Medicine. Design and methods of the study, described elsewhere,[22] are presented briefly below.

Project HeartBeat! included 678 children—314, 197, and 167, respectively, in three cohorts aged 8, 11, and 14 years at the time of enrollment from 1991 to 1993. The participants were recruited from schools in The Woodlands and Conroe TX. Informed written consent was obtained from the parent or guardian, and assent or consent was obtained from each study participant. Overall, 49.1% of the participants were female, 74.6% were white, 20.1% were black, and 5.3% were of other race/ethnicity. The study participants were examined every 4 months for up to 4 years (mean of 8.3 examinations per participant). When data collection ended in August 1995, 525 participants (77.4%) remained in the project.

M-mode, two-dimensional, and Doppler echocardiography was performed in resting subjects with the Interspec Apogee Annular Phased Array Doppler system and a 5- or 3.5-MHz transducer, as appropriate for the participant's size. Measurements were made at the time of the study, or offline from VCR videotape within 24 hours of the study using the Interspec Apogee calculation package. M-mode measurements were performed according to the standards of the American Society of Echocardiography.[24] End-diastolic measurements of left ventricular internal dimension (LVIDd), interventricular septal thickness (IVSd), and left ventricular posterior wall thickness (PWTd) were obtained at the level of the tips of the mitral

papillary muscles. Values for LVM were calculated according to the anatomically validated formula[25,26]

$$LVM=0.8 \times 1.04[(IVSd+LVIDd+PWTd)^3 - (LVIDd)^3]+0.6.$$

The quality of these echocardiograms and the accuracy of the measurements were continuously monitored at Texas Children's Hospital in Houston. In addition, a quality assessment substudy[27] was designed and implemented to assess overall accuracy and reproducibility of echocardiographic measurements. That study concluded that the echo measurements taken in Project HeartBeat! were made with acceptable precision and compare favorably with those taken in a clinical setting.

Ethnicity was categorized as nonblack or black. Exact age was calculated for the day of data collection. Anthropometric variables were measured using standard methods recommended for assessing physical growth and physique in childhood.[28] Participants were barefoot and wore surgical scrub suits for all measurements. Weight was measured to the nearest 0.1 kg with a beam-balance scale, and height was measured to the nearest 0.1 cm with a wall-mounted stadiometer. Skinfold thicknesses of triceps, subscapular, midaxillary, abdominal, distal thigh, and lateral calf areas were taken three times at each follow-up examination on the right side of the body with a Tanner–Whitehouse skinfold dial caliper. BMI was calculated by standard formula (kg/m^2). Body surface area (BSA) was estimated based on weight and height by the following method[29]:

$BSA\ (m^2) = (0.0001)\ (71.84)\ (Weight)^{0.425}\ (Height)^{0.725}$, where weight is in kg, height is in cm, and BSA is in m^2. The sum of skinfold thicknesses (SSF) was calculated as the total of the six skinfold thicknesses listed above.

Bioelectric resistance and reactance were measured using the RJL Systems bioelectric impedance analyzer BIA 101-A with participants supine and holding their breath. Both measurements were taken twice and averaged. Fat-free mass (FFM), fat mass, and percent body fat (PBF) were calculated from anthropometric and bioelectric impedance data, using the methods of Guo and colleagues.[30] Thus, the nine body-size indicators used in data analysis for predicting LVM were weight, BSA, $BSA^{1.5}$, FFM, $height^3$, $height^{2.7}$, $height^{2.3}$, $height^2$, and height; and the four body-fatness indicators used were fat mass, PBF, SSF, and BMI.

Descriptive statistical analyses were performed with SPSS, version 9.0. The effects of body size, body fatness, and covariates were estimated using MLwiN software, version 2.1. Because of the inherent hierarchic structure of the Project HeartBeat! data, a multilevel linear model was used as the statistical method for the analyses.[31]

The multivariate regression models of LVM were fitted in gender- and race-pooled data with gender (male=0, female=1) and race (nonblack=0, black=1) as covariates. Two levels of random variation were distinguished and estimated, one representing inter-individual variation and one representing variation nested within individuals. LVM was first regressed on gender and race. Age, higher-order age variables (age^2 and age^3), and interaction terms for gender by age and for race by age were then entered into the model one by one and their significance evaluated. Based on preliminary results indicating that race, unlike age and gender, was not a significant independent predictor of LVM, a basic LVM–gender–age model was derived, omitting race.

This basic model was used to generate nine unique models, each resulting from inclusion of one of the body-size indicator variables. To each of these nine models, one of the four indicators

of body fatness was added individually, in turn. This procedure resulted in 36 unique models in which LVM was regressed on age, gender, one of the nine body-size indicators, and one of the four body-fatness indicators. Change in the −2 log (likelihood) statistic was used to indicate whether the improvement or reduction in the goodness of fit of the model was significant after inclusion or exclusion of a predictor variable. Each parameter estimate divided by its SE assumes approximately the N(0,1) distribution and suggests whether the parameter is significantly different from 0 (if the result is >1.96 or <−1.96, then $p<0.05$). These LVM models were intended to be descriptive, and no corrections were made for multiple statistical tests. A p-value of 0.05 was used as the criterion for all statistical testing of regression coefficients.

Age was centered by subtracting 12.0 years (approximately the overall M of age) from the original values before deriving higher-order age terms for use in LVM models. Weight, height, their derivative terms (BSA, $BSA^{1,5}$, FFM, fat mass, PBF, BMI), and SSF were also centered before the multilevel modeling.

Results

Among the total of 5637 examinations, 4608 had valid determinations of LVM. Missing values of LVM mainly resulted from the observer being unavailable for echocardiography at the time of examinations (810), participants being authorized to miss this measurement (191), and participants' refusal (113). Age at data collection ranged from 8.1 to 17.98 years (M 12.06 years). The gender-specific Ms and SDs of LVM and the concurrent measures of age, weight, height, BSA, FFM, fat mass, PBF, BMI, and SSF are shown in Table 1. Mean value of LVM was 96.67 g overall, and it was approximately 15 g greater in boys than in girls. Weight, height, and BSA were slightly greater, and FFM was much greater, in boys than in girls. BMI was similar in both genders, and fat mass, PBF, and SSF were much greater in girls than in boys. Race and race-by-age interaction terms were not significant in any steps of the multilevel modeling and therefore were excluded from the LVM models.

The basic LVM–gender–age model is presented in Table 2. Gender, age, and gender-by-age interaction terms were significant determinants of LVM. Mean LVM according to the fixed part of the model is estimated as

$$Y=103.0-11.26(\text{Gender})+6.706(\text{Age})+0.0784(\text{Age}^2)-2.426(\text{Gender}^*\text{Age})-0.630(\text{Gender}^*\text{Age}^2),$$

where Age is the centered age, which equals chronologic age minus 12.0 years. At age 12, LVM was greater in boys than in girls by 11.26 g, whereas at age 9 or 16 years, it was about 10 g or 31 g greater in boys than in girls, respectively. Total variance of LVM was also age-dependent.

Considered individually, each of the nine body-size indicators significantly improved the model compared with the basic LVM–gender–age model. In each case, the p-value associated with the addition of a single body-size indicator was <0.0001 (Table 3), showing that each of these variables contained important additional information about the outcome. To assess the importance of including a body-fatness indicator in addition to each body-size indicator, fat mass, PBF, SSF, and BMI were added individually to the nine models, each containing a single body-size indicator. The results for the 36 models incorporating a body-fatness indicator in addition to a body-size indicator are shown in Table 3. The added body-fatness indicator is significant except when SSF or BMI are added to the model with BSA, or when PBF, SSF, or BMI are added to the model with $BSA^{1.5}$.

One of the resulting 36 models, containing the combination of FFM and PBF, among other variables, is presented in Table 4. Compared with the basic LVM–gender–age model in Table 2, the gender-by-age and gender-by-age^2 interaction terms were no longer significant. Significant between-subject variance of FFM indicated that the measurement variance of LVM varied with FFM, whereas the between-subject variance of age, observed in the LVM–gender–age model, was no longer significant. Average LVM was 8.959 g lower in girls aged 8–18 years than in boys of the same age. Age remained a significant predictor of LVM. FFM was the strongest determinant of LVM, as shown by the estimated regression coefficient. An increase of 1 kg in FFM was associated with a 2.425-g increase in LVM, equivalent to the change of 27.8 g in LVM with a change of 1 SD in FFM (see Table 1; SD of FFM=11.48 kg). PBF was also positively associated with LVM. A 1% increase in PBF was related to a 0.6759-g increase in LVM, which was a change of ~5.4 g in LVM with every 1-SD change in PBF.

Similarly, four multilevel LVM models, containing the combinations of FFM, weight, BSA, or height with FM, in addition to gender and age terms, are shown in Table 5. The gender effect was consistent across these models, with girls having lower LVM ranging from 8.6 to 12.3 g estimated from different models, when other predictors in each model were held at their M levels. Age continued to be a significant predictor of LVM in all four models after adjustment for gender, body size, and body fatness. In Models B–D, where weight, BSA, or height^3 were used instead of FFM as the body-size indicator, gender-by-age interaction terms remained significant, indicating an additional negative relationship between age and LVM in girls compared with boys.

The estimated effects of body size and body fatness on LVM from Model A in Table 5 were very similar to those estimated from the model presented in Table 4. Compared with the model in Table 4, the only different predictor in Model A was fat mass instead of PBF. In this model, every 1-kg change in FFM was associated with a change of 2.09 g in LVM (24.0-g change in LVM per 1-SD change in FFM), and every 1-kg change in fat mass was associated with a change of 1.121 g in LVM (7.2-g change in LVM per 1-SD change in fat mass).

Use of weight, BSA, or height^3 as body-size indicators in Models B–D revealed similar strong, positive effects of body size on LVM; however, results differed with respect to the effect of fat mass. A 1-kg change in body weight was estimated to be associated with a change of 2.022 g in LVM (33.3-g change in LVM per 1-SD change in weight); a 1-m^2 change in BSA was associated with a 110.0-g change in LVM (31.9-g change in LVM per 1-SD change in BSA); and a 1-m^3 change in height^3 was associated with a 24.51-g change in LVM (25.2-g change in LVM per 1-SD change in height^3). The regression coefficients of fat mass were negative, being −0.8069 g/kg (−5.3 g/SD) and −0.3153 g/m^2 (−2.03 g/SD), respectively, in Models B and C, when weight or BSA was used as the body-size indicator, respectively. In model D, where height^3 was used, the regression coefficient of fat mass remained positive (1.295 g/m^3 [8.3 g/SD]).

Further examination of the regression coefficients of body-fatness indicators among the 36 LVM models revealed a consistent pattern of the effect of body fatness: in models with a non–fat free body-size indicator (weight, BSA, or BSA$^{1.5}$) for prediction of LVM, effects of body-fatness indicators were all estimated to be negative (significant or not), whereas in those models containing a fat-free body-size indicator (FFM, height^3, height$^{2.7}$, height$^{2.3}$, height^2, or height), effects of body-fatness indicators were all estimated to be significantly positive (data not shown).

Discussion

The findings of the present analysis using longitudinal data from Project HeartBeat! confirmed previous results[6–8,19] showing that body size, especially when measured as FFM, is the strongest determinant of LVM in children and adolescents, and that both age and gender exert independent effects on LVM. The present results further indicated that the estimated effect of body fatness on LVM depends largely on the particular combination of body-size and body-fatness predictors chosen for the LVM models.

Earlier studies[6–8,19] in children and adolescents consistently revealed that body size was the most important determinant of LVM. Its importance was usually indicated by the percentage of variance of LVM explained by body-size indicators.[7,11] The magnitude of change in LVM related to each unit change in body-size indicators has only rarely been reported. A recent cross-sectional evaluation[10] in 201 healthy children aged 6–17 years using multiple regression analysis revealed that a 10-kg increase in lean body mass would result in a 20.2-g increase in LVM. The current findings regarding the LVM–body size relationship were in general agreement with existing results. Body size was again demonstrated as the most striking determinant of LVM among the explanatory variables evaluated. The mean change in LVM related to a 10-kg change in FFM was estimated in the current study to be 21–24 g when PBF or fat mass and other covariates were held constant (Tables 4 and 5). The magnitude of change in LVM per SD change in height^3 was estimated to be similar to that per SD change in FFM.

The impact of obesity on LVM has been of great interest because it may provide an approach to prevention or regression of left ventricular hypertrophy through weight control or weight reduction in overweight individuals. In adult populations, a positive association between obesity and LVM has been reported previously.[14–16] In these studies, the extent of obesity was frequently measured by the BMI. Although adults with higher BMI values usually tend to be more obese, BMI per se does not provide information on relative amounts of FFM and fat mass, nor does it estimate the absolute value of body fat. Thus, BMI alone does not permit direct estimation of the effect of body fat on LVM.

In childhood populations, available results on the independent effect of body fatness on LVM have been inconsistent. It has been reported[10] that a 10-kg increase in fat mass would result in a 5-g increase in LVM in healthy children and adolescents aged 6–17 years, after adjusting for effects of lean body mass and systolic blood pressure.[10] A study[18] conducted in patients aged 6–23 years with essential hypertension revealed an independent positive relationship between BMI and the index of LVM/height. A positive relationship between the index of LVM adjusted for height and total adipose weight was also found in Japanese children aged 12 years.[32]

It was reported from the Bogalusa Heart Study[8] that the ponderosity index (weight/height^3) and the triceps skinfold thickness were positive correlates of LVM and LVM/height$^{2.7}$ in most univariate cross-sectional and longitudinal analyses. However, in multivariate analyses, where weight and other covariates were also included in the regression models, the ponderosity index and the triceps skinfold thickness were no longer significant.[8] In the Medical College of Virginia Twin Study,[7] data from a group of 243 children aged 11 years showed that LVM was inversely related to suprailiac skinfold thickness in a multivariate regression model including weight, gender, systolic and diastolic blood pressures, and heart rate. Findings from the Quebec Family Study[33] demonstrated that, among healthy adolescents aged 9–18 years, BMI was positively and significantly correlated with LVM in all of the six age–gender subgroups, and the sum of six skinfolds was significantly correlated with LVM in only three of the six subgroups. Details of the correlations after adjustment for body size were not described in the report.

In view of the strong effect of body size in general, the recent recognition[10–12] of lean body mass as the closest correlate of LVM among various body-size indicators, and the inconsistent results on effects of body fatness, it was anticipated at the planning of the present study that it would be possible to distinguish, from among the total impact of body size on LVM, those effects resulting from lean body mass and those resulting from fat body mass. By comparing the effects of various combinations of body-size and body-fatness indicators as predictors of LVM, this study revealed a consistent pattern of the effect of body fatness: in those LVM models with weight or BSA as body-size indicators for prediction of LVM, the effects of body-fatness indicators were all estimated to be negative (significant or not), whereas in those models containing FFM or height, the effects of body-fatness indicators were all estimated to be significantly positive. This observation is also true for all 36 models for which the *p*-values were shown in Table 3, and it is consistent with existing results.[7,8,10,11] The CARDIA Study, [5] conducted in young adults, yielded similar results: subscapular skinfold thickness was not significant in the multivariate LVM regression model containing body weight, yet it became positively related to LVM when height was substituted for weight.

Obviously, a biologically plausible interpretation of the observation would be that both the size of the fat-free part of the human body and the size of the fat-containing part exert an impact on LVM; the former is the stronger determinant of LVM, and the latter, although weaker, is an additional positive determinant of LVM. In those LVM models with weight, BSA, or $BSA^{1.5}$, the effects of fat-free body size and fat body size were not distinguished, and as a result, the effect of fat body size was overestimated. Because of the overestimation, when a body-fatness indicator was further included in the LVM models, the residual effect of the fat body size was estimated to be negative.

This interpretation is further supported by a detail in the study's results: among those multilevel LVM models shown in Tables 4 and 5, the effects of body size on LVM were estimated to be larger in Models B and C by weight and BSA compared with those estimated by FFM and $height^3$, when changes of body-size indicators were considered in SD units. This finding means that in Models B and C, the total effects of body size on LVM, including those of fat-free body mass and those of fat body mass, were represented by either weight or BSA. By contrast, the results in Models A and D in Table 5 and the model in Table 4 show that only the effects of fat-free body size were represented, either by FFM or by $height^3$. Obviously, total body size effects should be larger than those of fat-free body size, if fat mass exerts an additional positive effect on LVM.

With an estimate of a 5-g change in LVM related to every 10-kg change in fat mass, it has been suggested[10] that fat mass played an important biological role in determining LVM but would be expected to be of only minor clinical importance. The current study's estimates of change in LVM with every 10-kg change in fat mass were about 11 g or 12 g (Models A and D, Table 5). Accordingly, an increase of 1 SD of fat mass would result in an average increase in LVM of 7.2–8.3 g in the current study population of healthy children and adolescents. Although the observed relationship between LVM and fat mass cannot be directly applied to obese individuals, it is reasonable to assume that the total amount of change in LVM related to possible change in fat mass among obese individuals could be much higher and thus have clinical significance.

It was reported earlier that criteria based on LVM indexed by body weight or BSA resulted in lower LVM index values in overweight subjects than in non-overweight subjects, whereas criteria based on LVM indexed by height identified the LVM index values as higher in these overweight subjects.[6,21] According to the results of the present study, overweight people on average have a higher LVM. However, the extra LVM related to excessive body fat is less than that related to additional FFM of the same amount. If body fat is adjusted in the same way as

FFM, as in the case of the criteria of LVM indexed by weight or BSA, it will be overadjusted in overweight people, who will then have a lower LVM index.

The current study has some potential limitations. First, body composition was determined by gender-specific formulas[30] on the basis of bioelectric impedance and body measurements rather than the more reliable methods of underwater weighing or dual-energy x-ray absorptiometry (DXA). However, underwater weighing and DXA were considered impractical for Project HeartBeat! because of the size of the study and frequency and logistics of examinations. Moreover, the formulas used had been cross-validated in several studies[30,34] that included children and young adults, and they had the smallest SEs among the various formulas for bioimpedance. Second, the study was conducted in healthy children and adolescents. The inclusion of apparently "normal" subjects precludes discrimination between physiologic increase of LVM and pathologic forms of left ventricular hypertrophy. Additionally, the observed quantitative relationship between LVM and fat mass or PBF could not be readily applied to morbidly obese individuals.

Conclusion

The current study confirmed that body size is a strong determinant of LVM and that body fatness is also an independent, positive correlate of LVM. Distinctions among body-size effects on LVM can be made between fat-free body mass and fat body mass, the former being the stronger predictor. When a non–fat free body-size indicator is used as a predictor of LVM, the effects of both fat-free body mass and fat body mass are forced to relate to the same body-size indicator, and their magnitudes are estimated to be equal, resulting in overestimation of the effect of fat body mass. Thus, when a further indicator for body fatness is included in prediction of LVM, the additional estimated effect related to the body fatness indicator appears to be negative. Based on this conclusion and biological plausibility, FFM and fat mass (or PBF) may be the best combination of body-size indicators and body fatness indicators for predicting LVM; height (e.g., $height^3$ or $height^{2.7}$) could be the substitute when FFM is not available. Non–fat free body-size indicators should be avoided in LVM models when a body-fatness indicator is included as a predictor. The additional positive effect of body fatness on LVM beyond that of the fat-free body size suggests that control of body fat may contribute to reducing the risk of left ventricular hypertrophy.

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Table 1

Left ventricular mass, and body-size and body-fat measurements by gender, Project HeartBeat!, 1991–1995

	Boys			Girls			Total		
	Mean	**SD**	***n***	**Mean**	**SD**	***n***	**Mean**	**SD**	***n***
Age (years)	12.04	2.55	2314	12.08	2.56	2294	12.06	2.56	4608
LVM (g)	104.37	39.53	2314	88.90	30.00	2294	96.67	35.94	4608
Weight (kg)	46.81	17.22	2307	45.50	15.70	2287	46.16	16.49	4594
Height (cm)	151.87	15.88	2310	149.52	13.46	2292	150.70	14.77	4602
BSA (m^2)	1.40	0.31	2305	1.36	0.27	2286	1.38	0.29	4591
FFM (kg)	36.50	13.02	2261	32.94	9.35	2223	34.73	11.48	4484
BMI (kg/m^2)	19.66	4.18	2305	19.82	4.43	2286	19.74	4.31	4591
PBF (%)	20.81	8.26	2261	25.06	7.06	2223	22.92	7.97	4484
Fat mass (kg)	9.95	6.40	2261	11.72	6.32	2223	10.83	6.43	4484
SSF (mm)	70.71	37.45	2254	82.37	34.01	2205	76.47	36.26	4459

BSA, body surface area; FFM, fat-free mass; LVM, left ventricular mass; PBF, percent body fat; SSF, sum of skinfold thicknesses

Table 2

Estimated model for left ventricular mass on gender and age, Project HeartBeat!, 1991–1995

Parameter	Estimate	SE	*p*-value
Fixed			
Constant	103.0	1.367	0.000
Gender (0, male; 1, female)	−11.26	1.95	0.000
Age (years)	6.706	0.3347	0.000
Age^2	0.0784	0.07887	0.320
Gender*age	−2.426	0.474	0.000
Gender*age^2	−0.630	0.1119	0.000
Between-subjects variance/covariance			
Constant	495.8	31.41	0.000
Age/constant	53.38	5.614	0.000
Age	6.001	1.955	0.002
Within-subjects variance			
Error	292.8	6.977	0.000

Note: Continuous predictive variables were centered before modeling.

Table 3

Significance of each body-size indicator alone and with each of four BFIs

BSI	*p*-value for BSI alone	*p*-value for added BFI Fat mass	PBF	SSF	BMI
Non–fat free BSI					
Weight	<0.0001	<0.0001	0.0002	0.0001	<0.0001
BSA	<0.0001	0.0047	0.0213	0.0578	0.0614
$BSA^{1.5}$	<0.0001	0.0069	0.1573	0.2207	0.1069
Fat-free BSI					
FFM	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
$Height^{3}$	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
$Height^{2.7}$	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
$Height^{2.3}$	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
$Height^{2}$	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Height	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

BSA, body surface area; BFI, body-fatness indicator; BSI, body-size indicator; FFM, fat-free mass; PBF, percent body fat; SSF, sum of skinfold thicknesses

Table 4

Estimated model for left ventricular mass, Project HeartBeat!, 1991–1995

Parameter	Estimate	SE	*p*-value
Fixed parameters			
Constant	100.5	0.8272	0.000
Gender (0, male; 1, female)	−8.959	1.02	0.000
Age (years)	−2.104	0.3265	0.000
Age^2 (years)	−0.113	0.05217	0.030
FFM (kg)	2.425	0.08499	0.000
PBF (%)	0.6759	0.05495	0.000
Between-subjects variance/covariance			
Constant	160.0	13.27	0.000
FFM/constant	7.168	0.7567	0.000
FFM	0.4694	0.08655	0.000
Within-subjects variance			
Error	278.4	6.510	0.000
−2 log (likelihood)	38,998		

Note: Continuous predictive variables were centered before modeling.

Table 5

Estimated models for left ventricular mass on gender, age, body-size, and fat mass, Project HeartBeat!, 1991–1995

	Model A BSI=FFM (kg)		Model B BSI=Weight (kg)		Model C BSI=BSA (m^2)		Model D BSI=Height^3 (m^3)	
Parameter	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**
Fixed parameters								
Constant	100.1	0.8283	101.3	0.9225	99.41	0.9009	100.8	0.9849
Gender (0, male; 1, female)	−8.61	1.006	−10.58	1.254	−9.914	1.195	−12.32	1.328
Age (years)	−2.132	0.3276	−1.508	0.427	−3.167	0.4373	−3.03	0.5037
Age^2 (years)	−0.1023	0.05258	−0.104	0.05262	0.122	0.0509	0.1343	0.0534
Gender*age			−1.039	0.4046	−0.8949	0.3875	−0.9519	0.4056
BSI	2.09	0.09018	2.022	0.08745	110.0	4.184	24.51	1.199
Fat mass (kg)	1.121	0.09	−0.8069	0.1455	−0.3153	0.1252	1.295	0.0904
Between-subjects variance/covariance								
Constant	165.1	13.63	176.9	15.69	190.2	13.76	216.7	17.26
Age/constant			17.05	3.83			8.941^{ns}	5.994
Age			1.157^{ns}	2.816			9.79^{ns}	5.209
BSI/constant	8.091	0.8174	3.26	0.7678	301.7	28.68	72.03	16.45
BSI/age			-0.1046^{ns}	0.4548			−30.65	13.61
BSI	0.5598	0.09268	0.2262	0.09014	410.7	94.46	123.9	38.72
Within-subjects variance								
Error	277.1	6.481	280.7	6.619	276.4	6.347	274.4	6.511
−2 log (likelihood)	39,000		39,017		38,932		39,023	

BSI, body-size indicator

^{ns}not significant at the level of 0.05