



Physique, subcutaneous fat, adipose tissue distribution, and risk factors in the Québec Family Study

PT Katzmarzyk¹, RM Malina², TMK Song³, C Bouchard^{4*}

¹Department of Kinesiology and Health Science, York University, North York, Ontario, Canada; ²Institute for the Study of Youth Sports, Michigan State University, East Lansing, MI, USA; ³Human Performance Laboratory, Lakehead University, Thunder Bay, Ontario, Canada and ⁴Physical Activity Sciences Laboratory, Laval University, Ste-Foy, Québec, Canada

OBJECTIVE: To investigate the relationships among subcutaneous fatness, subcutaneous adipose tissue (SAT) distribution, somatotype and risk factors for coronary heart disease (CHD).

SUBJECTS: The sample included 1410 (715 male and 695 female) youths and adults from the Québec Family Study. **MEASUREMENTS:** Six skinfolds and the dimensions necessary for the derivation of the Heath-Carter anthropometric somatotype (endomorph, mesomorph, ectomorph) were measured. The six skinfolds were summed to provide an index of subcutaneous adiposity (SUM). In addition, the trunk-to-extremity skinfold ratio, adjusted for SUM using regression procedures (TER), and the first principal component (PC1) of skinfold residuals (also adjusted for SUM) were used to indicate SAT distribution, independent of the overall level of fatness. Risk factors for CHD included systolic and diastolic blood pressures, and fasting glycaemia, triglycerides (TGs), plasma cholesterol, high and low density lipoprotein (HDL-C and LDL-C) cholesterol, and the HDL-C/total cholesterol (CHOL) ratio.

RESULTS: In general, SUM was positively correlated with endomorphy and mesomorphy, and negatively correlated with ectomorphy. On the other hand, SAT distribution was not associated with somatotype, except in females where TER and PC1 were negatively correlated with mesomorphy. Results of forward stepwise regression analyses to predict CHD risk factors, indicated that a significant proportion of the variance in the risk factors could be accounted for by SUM, SAT distribution and somatotype (up to 16%). SUM is the best predictor, entering the regressions first (most important) in six of 15 significant regressions in males and 14 of 16 significant regressions in females. Somatotype components enter as predictors 10 times in males, and six times in females. Similarly, TER and PC1 enter as predictors nine times in males and five times in females.

CONCLUSIONS: Somatotype is related to SUM, while somatotype and SAT distribution are largely independent of one another. Furthermore, SUM, somatotype and SAT distribution are significant predictors of biological risk factors for CHD.

Keywords: risk factors; fat distribution; body composition; physique; coronary heart disease; Québec Family Study

Introduction

The prevention of coronary heart disease (CHD) is a public health priority, as heart disease remains the leading cause of death in the United States.¹ Among the risk factors for CHD and related disorders such as Type 2 diabetes, a truncal, or central distribution of adipose tissue is of concern. Centralized body fat is associated with CHD and mortality,^{2–4} and Type 2 diabetes.^{5,6} Furthermore, central fat distribution is associated with other biological risk factors for CHD and Type 2 diabetes, such as elevated serum triglycerides,^{7,8} low high density lipoprotein (HDL)-cholesterol (HDL-C),⁹ hypertension^{10,11} and indicators of insulin and glucose metabolism.^{12–15}

Physique, or the overall configuration of the body, may also play an important role in health and disease.^{16,17} Historically, physique has been assessed most often using the somatotype, a three component index which includes endomorphy, mesomorphy and ectomorphy.¹⁷ Early methods of somatotyping, such as those of Sheldon and colleagues¹⁸ and Parnell,¹⁹ relied on the analysis of photographs, while the more recent Heath-Carter²⁰ anthropometric method relies completely on anthropometric dimensions. The various types of somatotype (Sheldon, Parnell, and Carter and Heath protocols) have been studied in relation to CHD^{21–28} and risk factors for CHD such as serum lipid levels,^{29–32} blood glucose levels,^{33,34} and blood pressure.^{31,35–37} A physique characterized by high endomorphy and mesomorphy may increase the risk for degenerative diseases such as CHD and Type 2 diabetes.

Given the focus on both subcutaneous adipose tissue (SAT) distribution and physique as risk factors for disease, an issue which remains to be addressed, is the degree to which somatotype and SAT distribution

*Correspondence: Dr Claude Bouchard, Physical Activity Sciences Laboratory, PEPS, Laval University, Ste-Foy, Québec G1K 7P4, Canada.
E-mail: claudio.bouchard@ken.msp.ulaval.ca
Received 20 March 1998; revised 2 November 1998; accepted 2 December 1998

are related, and whether they are independently related to risk factors. Mueller and Joos³⁸ indicated that the Sheldonian photoscopic somatotype was related to the degree of central adiposity in adult males. Sheldon et al³⁹ used photographs of 824 men in the *Atlas of Men* which indicated that android obese men were rated higher on mesomorphy and lower on ectomorphy than gynoid obese men. Among youth, the Heath-Carter²⁰ anthropometric somatotype was related to relative fat patterning, those with a centralized subcutaneous fat distribution were higher in endomorphy and mesomorphy, and lower in ectomorphy.⁴⁰ However, neither study controlled for overall fatness, and further research is thus required to better determine the nature of the relationship in both youth and adults.

The current research focus is largely on fatness and relative SAT distribution and their relationship with disease. However, there is renewed interest in incorporating other measures into risk profiles. Perhaps physique may be an important predictor for disease risk, beyond what is explained by fatness and SAT distribution. The purpose of this study was to (1) determine the relationships between subcutaneous fatness, relative SAT distribution and physique, as assessed by the Heath-Carter²⁰ anthropometric somatotype protocol, and (2) determine the contributions of an estimate of total subcutaneous fat, indicators of SAT distribution and somatotype, to each of the risk factors.

Methods

Sample

The sample included 1410 (715 males and 695 females) participants from Phase I of the Québec Family Study (QFS), 1978–1981.⁴¹ The sample was divided into youth (9–18 y) and adult (30–49 y) gender specific age groups. The sample of youth included siblings; however, the adult sample consisted of unrelated individuals. The subjects were of French Canadian ancestry and were recruited from the greater Québec City area. All procedures were approved by the Medical Ethics Committee at Laval University.

SAT Distribution

A single trained anthropometrist measured skinfolds at the biceps, triceps, medial calf, abdominal, subscapular, and suprailiac sites, with a Harpenden caliper (John Bull, British Indicators Ltd, England) to the nearest 0.5 mm. The skinfold measurements were individually reproducible with intraclass coefficients ≥ 0.94 , whereas the intra-observer technical errors of measurement ranged from 1.0 mm for biceps to 2.1 mm for suprailiac.⁴² The values were summed to provide a single measure of subcutaneous adiposity (SUM).

The ratio of the sum of trunk (subscapular, suprailiac, abdominal) skinfolds to the sum of extremity (triceps, biceps, medial calf) skinfolds (TER) was used as an index of relative SAT distribution. Since ratios of trunk to extremity skinfolds are related to overall fatness,^{43,44} it was regressed on SUM, and the residuals were retained to represent an index of relative fat distribution independent of overall level of subcutaneous fatness.

Principal components analysis was also used to estimate SAT distribution using the procedures described by Deutsch *et al*⁴⁵ and Baumgartner *et al*.⁴⁶ Skinfolds were first transformed to natural logarithms. Each log transformed skinfold was then regressed separately on the log mean skinfold of the individual within gender and age groups. The standardized skinfold residuals were retained for further analysis, to represent the skinfold, adjusted for the effects of mean subcutaneous fatness. Principal components analysis was then performed on the standardized skinfold residuals to evaluate the underlying distribution of SAT. The first principal component score (PC1) was saved and was used as an index of relative SAT distribution, independent of overall level of subcutaneous fatness.

Somatotype

Anthropometric dimensions necessary for the derivation of the Heath-Carter anthropometric somatotype were also taken. Somatotype was estimated²⁰ as follows:

$$A. \text{ Endomorphy} = -0.7182 + 0.1451(X) - 0.00068(X^2) + 0.0000014(X^3)$$

where X is the sum of the triceps, subscapular, and supraspinale skinfolds, adjusted for stature.

$$B. \text{ Mesomorphy} = [0.858 \times \text{biepicondylar breadth (cm)}] + [0.601 \times \text{bicondylar breadth (cm)}] + [0.188 \times \text{CAG}] + [0.161 \times \text{CCG}] - [\text{stature (cm)} \times 0.131] + 4.50$$

where CAG = corrected arm girth = flexed arm circumference (cm) minus triceps skinfold (cm), and CCG = corrected calf girth = maximal calf circumference (cm) – medial calf skinfold (cm)

$$C. \text{ Ectomorphy} = \text{SMR} \times 0.732 - 28.58$$

$$\text{If SMR} < 40.75 \text{ but } > 38.25,$$

$$\text{Ectomorphy} = \text{SMR} \times 0.0463 - 17.63$$

$$\text{If SMR} \leq 38.35,$$

$$\text{Ectomorphy} = 0.1$$

$$\text{where SMR} = \text{stature mass ratio} = \frac{\text{stature (cm)}}{\sqrt[3]{\text{mass (kg)}}}$$

If any somatotype component is zero or negative, a value of 0.1 is assigned, because, by definition, a rating cannot be zero or negative.²⁰ The technical errors of measurement were < 0.3 somatotype units, and intraclass correlations for repeated measurements were ≥ 0.96 .⁴²

CHD risk factors

Blood pressures. Systolic (SBP) and diastolic (DBP) blood pressures were measured in the fasted state with a sphygmomanometer and stethoscope early in the morning, with the subject in a supine position after a 10 min rest period following the recommendations of the American Heart Association.⁴⁷

Blood sample. A venous blood sample was obtained early in the morning, after the blood pressure measurements were taken, following a 12–14 h fast. Blood was collected into vacutainer tubes (Becton Dickinson, Franklin Lakes, NJ, USA) without anticoagulant and was allowed to clot at room temperature for 30 min. The blood was then centrifuged at 1500 g for 20 minutes. Serum was stored at -80°C .

Serum lipids. Total cholesterol (CHOL) and triglycerides (TGs) were determined using the Abbot VP system (Abbott, South Pasadena, CA). CHOL was assayed by an enzymatic method, using the commercial kit CHOD-PAP of Boehringer (Mannheim, Germany). TGs were assayed with the single enzymatic method of Abbott Laboratories using the commercial kit A-GENT (Abbot, South Pasadena, CA). HDL-C was separated from lipoproteins of lower densities by the phosphotungstate $- \text{Mg}^{2+}$ precipitation technique.⁴⁸ The cholesterol content of the supernatant was determined by the same method as CHOL. LDL-C was estimated by using the Friedewald *et al* formula.⁴⁹ Reliability of the serum lipid determinations have been presented elsewhere.⁵⁰

Plasma glucose. Fasting plasma glucose (GLY) was enzymatically measured with the techniques of Richterich and Dauwalder.⁵¹

Statistical analysis

The relationships between indicators of SAT distribution and somatotype components were evaluated using correlation analyses. Since somatotype is a three component index, analyses were performed both with and without adjustment of the other two somatotype components, by regression procedures. Additionally, the sample was divided into those with a predominantly central SAT distribution and those with a more peripheral SAT distribution using the upper and lower tertiles of the TER and PC1. Differences in somatotype between individuals in the upper and lower tertiles of the distribution of TER and PC1 were determined using the protocol of Cressie *et al*.⁵² The first step was to perform an overall MANOVA between the groups. Univariate F-tests were then performed to determine which components were contributing to the significant difference.

To evaluate the relationships among subcutaneous fatness, relative SAT distribution, somatotype and risk factors for CHD, forward stepwise multiple regression analyses were performed with each risk factor as the dependent variable. Given the high degree of concordance between SUM and endomorphy in defining fatness, endomorphy was not used in the prediction equations. Mesomorphy and ectomorphy were retained to represent physique. SUM, TER, PC1, mesomorphy and ectomorphy were first adjusted for age within each age and gender group, by applying the linear regression:

$$Y = \text{age} + \text{age}^2 + \text{age}^3.$$

The residuals of the age-adjustment regressions were retained and used as the independent variables in the prediction of risk factors. All analyses were performed using statistical package for social sciences (SPSS) procedures.⁵³

Results

Descriptive anthropometric characteristics of the subjects are presented in Table 1. In each age group, males are significantly taller and heavier, and have a greater mean TER and lower SUM than females. Male youth are also more mesomorphic and ectomorphic than females, whereas adult males are more

Table 1 Age, body size, somatotype and subcutaneous fatness of youth and adults by gender

	n	Age (y)		Stature (cm)		Mass (kg)		BMI (kg/m ²)		Endomorphy		Mesomorphy		Ectomorphy		SUM (mm)		TER (mm/mm)	
		M	s.d.	M	s.d.	M	s.d.	M	s.d.	M	s.d.	M	s.d.	M	s.d.	M	s.d.	M	s.d.
Youth																			
Males	421	13.7	2.4	157.4	15.5	47.8	14.2	18.8	2.7	2.3	1.2	4.2	0.9	3.5	1.1	46.3	23.4	1.1	0.4
Females	360	13.9	2.6	154.3	11.0*	45.7	11.0*	18.9	2.8	3.3	1.3*	3.4	1.0*	3.3	1.2*	65.4	26.3*	1.0	0.3*
Adults																			
Males	294	42.7	3.9	172.4	6.0	75.7	11.2	25.5	3.3	4.1	1.5	5.5	1.1	1.6	1.0	75.2	29.5	2.3	0.6
Females	335	41.3	4.2	159.5	5.5*	59.2	9.1*	23.3	3.6*	4.9	1.5*	4.2	1.3*	1.8	1.1	100.0	34.8*	1.2	0.4*

M = mean; N = number of subjects; s.d. = standard deviation; BMI = body mass index; SUM = Σ 6 skinfolds; TER = Σ 3 trunk/ Σ 3 Extremity skinfolds.

*Males significantly different than females at $P < 0.05$.

mesomorphic and less ectomorphic than females. The PC1 of the skinfold residuals (adjusted for SUM) accounts for between 30–46% of the variance, while the second component accounts for between 19–25% of the variance (Table 2). PC1 represents a trunk-extremity contrast, whereas the second component varies among age and gender groups, and is difficult to interpret. The direction of the factors for PC1, trunk skinfold residuals positive and extremity skinfold residuals negative, indicate that individuals with high factor scores have a more centralized distribution of SAT, whereas those individuals with lower factor scores have a more peripheral distribution of SAT.

Table 2 Results of principal components analysis of the six skinfolds

Skinfold	Youth		Adults	
	PC1	PC2	PC1	PC2
Males				
Triceps	-0.81	0.19	-0.71	0.25
Biceps	-0.66	0.07	-0.50	0.37
Medial Calf	-0.71	-0.05	-0.69	0.07
Abdominal	0.61	0.60	0.03	-0.95
Subscapular	0.69	0.33	0.09	0.01
Suprailiac	0.52	-0.83	0.71	0.62
Eigenvalue	2.73	1.19	1.75	1.50
Percent Variance	45.5	19.9	29.1	24.9
Females				
Triceps	-0.68	0.08	-0.71	0.17
Biceps	-0.39	0.64	-0.36	0.67
Medial Calf	-0.64	-0.44	-0.75	-0.41
Abdominal	0.70	-0.33	0.61	-0.46
Subscapular	0.26	-0.36	0.50	0.53
Suprailiac	0.53	0.66	0.66	0.11
Eigenvalue	1.85	1.29	2.26	1.15
Percent Variance	30.8	21.4	37.7	19.2

PC1 = first principal component; PC2 = second principal component.

Correlations between subcutaneous fatness (SUM), the two indicators of relative SAT distribution and somatotype components are presented in Table 3. The results are presented both for the raw somatotype component and for each component after adjustment for the other two components by regression procedures. Correlations between SUM and endomorphy (range 0.94–0.98) and mesomorphy (range 0.41–0.53) are positive. On the other hand, correlations between SUM and ectomorphy are consistently negative (-0.64 to -0.71). The correlations are lower when each somatotype component is adjusted for the other components.

Correlations between indicators of central SAT distribution and somatotype are low with few trends. Among females, both TER and PC1 have negative correlations with mesomorphy, indicating that higher trunk fat is associated with lower mesomorphy. The correlations are similar in direction after adjusting for the other somatotype components, but are greater in magnitude. Correlations among TER, PC1 and other components approach zero and generally are not significant.

Results of the MANOVAs for somatotype between individuals in the lower and upper tertiles of the TER and PC1 indicate that males with a relatively greater central distribution of SAT (upper tertile) do not significantly differ in somatotype from males in the lower tertile (Table 4). Among females, however, those in the upper tertile of PC1 and TER have lower mesomorphy than those in the lower tertile. Thus, females with a greater relative proportion of trunk fat appear to be less mesomorphic than those with relatively more extremity fat.

The results of the forward stepwise multiple regression analyses for the prediction of risk factors from

Table 3 Correlations between indicators of subcutaneous fatness, subcutaneous adipose tissue (SAT) distribution and somatotype components

	Unadjusted ^a			Adjusted ^b		
	Endomorphy	Mesomorphy	Ectomorphy	Endomorphy	Mesomorphy	Ectomorphy
Youth						
<i>Males</i>						
SUM	0.98*	0.41*	-0.64*	0.96*	-0.06	-0.09
TER	0.09	0.08	-0.09	0.05	0.02	-0.01
PC1	0.07	0.07	-0.07	0.04	0.03	0.00
<i>Females</i>						
SUM	0.96*	0.44*	-0.70*	0.91*	-0.17*	-0.19*
TER	0.04	-0.13*	0.03	0.03	-0.17*	-0.08
PC1	-0.01	-0.15*	0.05	-0.02	-0.19*	-0.11*
Adults						
<i>Males</i>						
SUM	0.94*	0.53*	-0.66*	0.90*	0.11	-0.04
TER	0.10	0.07	-0.11	0.05	-0.02	-0.06
PC1	0.12*	0.04	-0.05	0.12*	0.01	0.04
<i>Females</i>						
SUM	0.96*	0.57*	-0.71*	0.92*	0.25*	0.13*
TER	0.11*	-0.14*	-0.02	0.13*	-0.25*	-0.11*
PC1	0.10	-0.13*	0.01	0.14*	-0.19*	-0.05

^aAdjusted for age only.

^bAdjusted for age and the other two somatotype components.

*Correlations significant at $P < 0.05$.

SUM = Σ 6 skinfolds; TER = Σ 3 trunk / Σ 3 extremity skinfolds; PC1 = first principal component.

Table 4 Results of MANOVAs of somatotype between upper and lower tertiles of the trunk-to-extremity skinfold ratio and the first principal component (TER and PC1)

	Λ	<i>F</i>	<i>Significance</i>	<i>Component</i>	<i>Lower Tertile</i>		<i>Upper Tertile</i>		<i>F</i>	<i>Significance</i>
					<i>M</i>	<i>s.d.</i>	<i>M</i>	<i>s.d.</i>		
TER										
Youth										
Males	0.99	1.02	0.384	Endomorphy	2.3	1.3	2.5	1.2	3.03	0.083
				Mesomorphy	4.2	0.9	4.3	1.0	0.32	0.571
				Ectomorphy	3.5	1.1	3.4	1.2	1.03	0.312
Females	0.96	3.13	0.026	Endomorphy	3.3	1.4	3.5	1.3	0.06	0.804
				Mesomorphy	3.6	1.0	3.3	0.9	4.31	0.039*
				Ectomorphy	3.2	1.3	3.1	1.2	0.21	0.650
Adults										
Males	0.98	1.28	0.284	Endomorphy	4.1	1.8	4.4	1.4	2.42	0.122
				Mesomorphy	5.5	1.3	5.7	1.0	1.60	0.207
				Ectomorphy	1.6	1.2	1.3	0.9	3.45	0.065
Females	0.90	7.97	< 0.001	Endomorphy	4.9	1.5	5.2	1.5	1.47	0.227
				Mesomorphy	4.4	1.4	3.9	1.0	10.58	0.001*
				Ectomorphy	1.7	1.1	1.8	1.1	0.49	0.485
PC1										
Youth										
Males	0.99	0.74	0.529	Endomorphy	2.2	0.9	2.4	1.1	1.63	0.203
				Mesomorphy	4.1	0.9	4.2	1.0	0.82	0.366
				Ectomorphy	3.6	1.1	3.5	1.2	0.46	0.499
Females	0.96	3.34	0.020	Endomorphy	3.3	1.3	3.4	1.4	0.03	0.866
				Mesomorphy	3.6	1.0	3.3	0.9	4.47	0.035*
				Ectomorphy	3.2	1.3	3.1	1.2	0.28	0.598
Adults										
Males	0.97	1.76	0.156	Endomorphy	3.9	1.7	4.3	1.3	4.58	0.034
				Mesomorphy	5.5	1.2	5.6	1.0	0.70	0.403
				Ectomorphy	1.6	1.2	1.4	0.9	2.78	0.097
Females	0.91	6.92	< 0.001	Endomorphy	4.8	1.5	5.0	1.6	0.63	0.429
				Mesomorphy	4.4	1.3	3.9	1.1	10.85	0.001*
				Ectomorphy	1.7	1.1	1.9	1.1	1.31	0.254

TER = Σ 3 trunk / Σ 3 extremity skinfolds; PC1 = first principal component.

*Correlations significant at $P < 0.05$.

fatness, relative SAT distribution and somatotype are presented in Table 5 and Table 6. Significant equations are obtained for all variables, with the exception of LDL-C in adult males; the variance explained in the significant regressions accounted for up to 16% of the total. Subcutaneous fatness (SUM) is the best predictor of the risk factors, entering the regressions first (most important) in six of 15 significant regressions in males and 14 of 16 significant regressions in females. Somatotype and relative SAT distribution are also important predictors of risk factors. Somatotype components (mesomorphy and ectomorphy) enter as predictors 10 times in 15 significant regressions in males, and six times in 16 significant regressions in females. Similarly, indicators of relative SAT distribution (TER, PC1) enter as predictors nine times in males and five times in females.

Discussion

Somatotype, subcutaneous fatness and SAT distribution

The results for the relationship between subcutaneous fatness (SUM) and somatotype are concordant with other studies. Subcutaneous fatness is positively related to endomorphy and mesomorphy, and negatively related to ectomorphy. Seltzer and Mayer⁵⁴

compared somatotypes of 180 obese adolescent girls with 67 non-obese school girls of the same age. The obese adolescents were more endomorphic and mesomorphic, and less ectomorphic than the school girls. Similar trends have been reported for obese women.⁵⁵

In this sample of youth and adults, there was generally no association between somatotype and SAT distribution. Among males, those in the highest tertile of the TER and PC1 did not differ in somatotype, while females in the upper tertile were less mesomorphic than those in the lower tertile. The results are also consistent with those of the correlational analyses, which demonstrated no association between SAT distribution and somatotype, with the exception of negative correlations between central SAT distribution and mesomorphy in females.

The results are not consistent with those of other studies which have attempted to associate SAT distribution and somatotype. Among Basque youth aged 8–19 y, those with a centripetal fat distribution, were higher in endomorphy and mesomorphy, and lower in ectomorphy than those with a peripheral fat distribution in both sexes.⁴⁰ Alternatively, men who were characterized by an android (central) distribution of body fat, rated higher in mesomorphy, but lower in endomorphy.³⁸ Although the results of these studies suggest an association between relative fat distribution and somatotype, neither controlled for level of overall fatness, as in the present study. Thus, the

Table 5 Results of forward stepwise multiple regression analyses for the prediction of coronary heart disease (CHD) risk factors from subcutaneous fatness, subcutaneous adipose tissue (SAT) distribution and somatotype in males

Risk factor	Youth			Adults		
	R ²	Variable Entered	Coefficient	R ²	Variable Entered	Coefficient
DBP	0.10	Constant	61.91	0.16	Constant	77.65
		TER	16.48		TER	4.48
		PC1	3.11		ECTO	-2.71
		SUM	-0.44		SUM	0.04
		MESO	1.69			
SBP	0.05	Constant	109.72	0.11	Constant	120.25
		TER	9.09		TER	3.30
					ECTO	-4.27
Glycaemia	0.04	Constant	4.81	0.02	SUM	0.06
		SUM	0.004		Constant	5.06
					SUM	0.003
TG	0.03	Constant	0.81	0.14	Constant	1.71
		ECTO	-0.09		TER	0.87
		SUM	-0.02		PC1	0.39
CHOL	0.01	Constant	4.35	0.03	Constant	5.83
		PC1	0.12		ECTO	-0.30
HDL-C	0.03	Constant	1.44	0.06	Constant	1.20
		SUM	-0.002		SUM	-0.003
		ECTO	0.08			
LDL-C	0.02	Constant	2.56	NVE		
HDL-C/CHOL	0.04	PC1	-0.11	0.11	Constant	0.21
		Constant	0.33		SUM	-0.0004
		SUM	-0.0004		ECTO	0.004
		ECTO	0.004		MESO	-0.03

R² coefficient of multiple determination; DBP=diastolic blood pressure; SBP=systolic blood pressure; TG=triglyceride; CHOL=total cholesterol; HDL-C=high density lipoprotein cholesterol; LDL-C=low density lipoprotein cholesterol; NVE: no variables entered; TER=Σ 3 trunk/Σ 3 extremity skinfolds; PC1=first principal component; SUM=Σ 6 skinfolds; MESO=mesomorphy; ECTO=ectomorphy; independent variables included SUM, TER, PC1, MESO and ECTO.

Table 6 Results of forward stepwise multiple regression analyses for the prediction of coronary heart disease (CHD) risk factors from subcutaneous fatness, subcutaneous adipose tissue (SAT) distribution and somatotype in females

Risk Factor	Youth			Adults		
	R ²	Variable Entered	Coefficient	R ²	Variable Entered	Coefficient
DBP	0.07	Constant	63.42	0.12	Constant	73.12
		SUM	0.001		TER	-6.02
		TER	20.13		SUM	0.10
		PC1	3.80		PC1	-4.07
SBP	0.09	Constant	107.72	0.09	Constant	113.76
		SUM	0.11		SUM	0.09
Glycaemia	0.03	Constant	4.69	0.11	Constant	4.77
		SUM	0.002		SUM	0.005
		ECTO	0.09			
TG	0.03	Constant	0.88	0.07	Constant	1.08
		SUM	0.003		SUM	0.004
CHOL	0.01	Constant	4.49	0.04	Constant	5.39
		ECTO	-0.18		SUM	0.005
HDL-C	0.04	Constant	1.42	0.04	Constant	1.56
		SUM	-0.002		SUM	-0.002
		MESO	0.06			
LDL-C	0.03	Constant	2.67	0.06	Constant	3.34
		SUM	0.004		SUM	0.006
		ECTO	-0.16		MESO	0.14
HDL-C/CHOL	0.05	Constant	0.36	0.07	Constant	0.30
		SUM	-0.0004		SUM	-0.0006
		ECTO	-0.02			

R² coefficient of multiple determination; DBP=diastolic blood pressure; SBP=systolic blood pressure; TG=triglyceride; CHOL=total cholesterol; HDL-C=high density lipoprotein cholesterol; LDL-C=low density lipoprotein cholesterol; TER=Σ 3 trunk/Σ 3 extremity skinfolds; PC1=first principal component; SUM=Σ 6 skinfolds; MESO=mesomorphy; ECTO=ectomorphy; independent variables included SUM, TER, PC1, MESO and ECTO.

evidence suggests that somatotype may be related to subcutaneous fatness, and not relative fat distribution *per se*.

Prediction of risk factors

To our knowledge, no single study has attempted to simultaneously quantify the contribution of subcutaneous fatness, SAT distribution and physique, to risk factors. The results suggest that up to 16% of the variance in risk factors for CHD can be explained by subcutaneous fatness, relative SAT distribution and physique. Among males, five of the 15 regressions explained $\geq 10\%$ of the variance, while seven explained $\geq 5\%$. In females, two of the 16 regressions explained $\geq 10\%$ of the variance, while nine explained $\geq 5\%$. Subcutaneous fatness *per se* appears to be the best predictor, especially in females; however, neither SAT distribution nor somatotype, appeared to be a better predictor than the other. Given that subcutaneous and visceral abdominal adiposity are related, some of the associations found between risk factors and subcutaneous adiposity could be explained by the relationship with visceral fatness. Placed within a population health perspective, the ability to explain up to 16% of the variance in risk factors by using anthropometric measures is an important finding.

Blood pressure

SBP and DBP are best predicted by TER in males, suggesting a role for central fat distribution. Similarly, TER and PC1 are important predictors of DBP in females, and of SBP in adult females, in conjunction with SUM. Using the adult sample from the QFS, Després *et al*¹¹ demonstrated significant correlations between subcutaneous fatness (skinfolds), percentage body fat (underwater weighing), the TER, and both systolic and diastolic blood pressures. Furthermore, analysis of variance (2×2 factorial with fixed effects) indicated that percentage body fat had a significant effect on DBP and SBP, whereas fat distribution *per se* only had an effect on DBP in men. On the other hand, Robinson and Bruce³⁵ found that males and females characterized as stocky or lateral in build were at a greater risk for hypertension, based on the ratio of chest circumference to height. Malina *et al*³¹ found low correlations between individual somatotype components and blood pressures; however, in the present study, somatotype was a predictor of both DBP and SBP in males. Thus, the prediction of blood pressure appears to be increased when multiple physique and SAT distribution measures are incorporated into the model.

Lipids

Given the association between blood lipid levels and the risk of cardiovascular disease (CVD), the relationship between physique or SAT distribution and blood

lipids has received considerable attention. In the present study, SUM appears to have an impact on the prediction of HDL-C and HDL-C/CHOL. In both males and females, the regression coefficient for SUM is negative for these variables, which is in the expected direction. In other words, greater subcutaneous fatness is associated with lower levels of HDL-C. The blood concentration of HDL-C was significantly associated with abdominal fat in a sample of men from the QFS.⁹ Although a portion of the association was mediated by serum TG concentrations, there was still a significant association between HDL-C and abdominal fat, independent of obesity and serum TG concentrations. Similarly, among obese premenopausal women, the concentration of plasma TGs increased significantly as the waist-to-hip ratio (WHR) increased.¹³ There is also considerable evidence which suggests that physique is related to blood lipid concentrations. Gertler *et al*³⁰ found that endomorphic men had higher cholesterol and phospholipid concentrations than ectomorphic men, while mesomorphic CHD patients had poorer blood lipid and cholesterol concentrations than less mesomorphic patients. Similarly, Tanner²⁹ demonstrated a correlation of 0.39 between endomorphy and cholesterol concentration in 46 healthy adult men. Among adults of the QFS, correlations between blood lipids and cholesterol were negative with ectomorphy in males, and positive with endomorphy in females.³¹ The evidence thus suggests that a physique characterized by high endomorphy and mesomorphy may increase the risk for CHD by having a poor blood lipid profile.

Blood glucose

The relationship between physique and blood glucose has also been investigated. In the present study, glycaemia is best predicted by SUM in both males and females, suggesting that the absolute amount of subcutaneous fat is important in explaining the variance in glucose metabolism. Among migrant Tamil Indians, diabetic subjects tend to be more endomorphic and mesomorphic than non-diabetics.^{33,34} Similarly, adult males and females from the QFS in the upper quartile for glycaemia were significantly more endomorphic and less ectomorphic than subjects in the lowest quartile, while females in the upper quartile were also more mesomorphic than those in the lowest quartile.³¹ SAT distribution *per se* also appears to be associated with insulin and glucose metabolism. Among healthy, obese women, those in the upper quartile for the WHR, had reduced carbohydrate tolerance and higher plasma insulin concentrations than women in the lowest quartile, independent of age and degree of obesity.¹² Similarly, android Japanese American women demonstrated higher fasting blood glucose and insulin concentrations than gynoid women, as well as a poorer response to a oral glucose tolerance test (OGTT).⁶

Multiple risk factors

Using canonical correlation analysis, skinfolds on the trunk were more related to CHD risk factors (cholesterol, glucose, uric acid, TGs, SBP, DBP) than skinfolds on the extremities, in a sample of men and women aged 18–65 y, suggesting that a central SAT distribution is also a risk factor for CHD.⁷ Using a similar approach in children aged 6–14 y, Sangi *et al*⁵⁶ found that 19–61% of the variance in cholesterol, pulse, SBP, DBP4, DBP5 could be explained by five skinfolds; however, the patterns of the loadings were difficult to interpret. Additionally, Pearson correlations between single anthropometric indices and the risk factors were low, rarely reaching 0.30. Thus, the authors concluded that the association between SAT distribution and risk factors was not as strong in children as it is in adults; however, they acknowledged that different results may have been obtained if they had used a more complete list of risk factors.⁵⁶ Results of the present study indicate significant associations between SAT distribution and risk factors in both youth and adults, once the effects of overall fatness were accounted for.

A few patterns by age and gender can be seen in the regressions. For example, SUM appears to be a very important predictor in females, entering 14 of 16 of the regressions first, whereas it enters first in only six of 15 regressions in males. Thus, total subcutaneous fat is a strong predictor of risk factors in females. The risk factors are generally better predicted in adults than youth. For each risk factor, the variance explained is higher in adults than youth, except for glycaemia and LDL-C in males, and for SBP and HDL-C in females (equal variances).

Conclusions

The rationale for studying the relationship between risk factors and disease is to identify those individuals who are at increased risk for a disease, such that modifiable risk factors may be altered before the disease develops. It is also important, therefore, to determine the relationships between risk factors, so that independence of a given risk factor may be determined. The results of the present cross-sectional study demonstrate that SAT distribution and somatotype may be independent risk factors for disease, as they are not significantly related. Subcutaneous fatness appears to be the best predictor of risk factors, however, both SAT distribution and somatotype (ectomorphy and mesomorphy) are also important predictors. Prospective study designs are required to determine which risk factor (total fatness, somatotype or fat distribution) may be more important in defining future disease risk, and whether incorporating the three factors plus measurements of abdominal visceral fat levels may improve the definition of risk.

Acknowledgements

This research was supported in part by the Medical Research Council of Canada, Fonds de la Recherche en Santé du Québec, Formation de Chercheurs et Aide à la Recherche-Québec, Ontario-Québec Exchange Program and the Institute for the Study of Youth Sports of Michigan State University. Thanks are expressed to colleagues and technicians from the Physical Activity Sciences Laboratory who were involved in the data collection of Phase 1 of the Québec Family Study. Claude Bouchard is funded, in part, by the Donald B. Brown Research Chair on Obesity which is supported by the Medical Research Council of Canada and Hoffman-La Roche Canada.

References

- 1 National Heart, Lung and Blood Institute. *Fact Book Fiscal Year 1997*. US Department of Health and Human Services, National Institutes of Health: Bethesda, 1997.
- 2 Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjöström L. Distribution of adipose tissue and risk of cardiovascular disease and death: A 12 year follow-up of participants in the population study of women in Gothenburg, Sweden. *Br. Med. J.* 1984; **289**: 1261–1263.
- 3 Ducimetiere P, Richard J, Cambien F. The pattern of subcutaneous fat distribution in middle-aged men and the risk of coronary heart disease: The Paris Prospective Study. *Int J Obes* 1986; **10**: 229–240.
- 4 Donahue RP, Abbott RD, Bloom E, Reed DM, Yano K. Central obesity and coronary heart disease in men. *Lancet* 1987; **1**: 822–824.
- 5 Ohlsson LO, Larsson B, Svardsudd K, Welin L, Eriksson H, Wilhelmsen L, Björntorp P, Tibblin G. The influence of body fat distribution on the incidence of diabetes mellitus – 13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes* 1985; **34**: 1055–1058.
- 6 Fujimoto WY, Newell-Morris LL, Shuman WP. Intra-abdominal fat and risk variables for non-insulin dependent diabetes (NIDDM) and coronary heart disease in Japanese American women with android or gynoid fat patterning. In: Oomura O, Tarui S, Inoue S, Shimazu T (eds). *Progress in Obesity Research*. Libbey: London, 1990, pp 317–322.
- 7 Blair SN, Ludwig DA, Goodyear NN. A canonical analysis of central and peripheral subcutaneous fat distribution and coronary heart disease risk factors in men and women aged 18–65 years. *Hum Biol* 1988; **60**: 111–122.
- 8 Bailey S, Campos H, Schosinsky K, Mata L. Relationship of upper body fat distribution to serum glucose and lipids in a Costa Rican population. *Am J Phys Anthropol* 1985; **73**: 111–117.
- 9 Després J-P, Tremblay A, Pérusse L, Leblanc C, Bouchard C. Abdominal adipose tissue and serum HDL-cholesterol: Association independent from obesity and serum triglyceride concentration. *Int J Obes* 1988; **12**: 1–13.
- 10 Blair SN, Habicht JP, Sims EAH, Sylwester D, Abraham S. Evidence for an increased risk for hypertension with centrally located body fat and the effect of race and sex on this risk. *Am J Epidemiol* 1984; **119**: 526–540.
- 11 Després J-P, Tremblay A, Thériault G, Pérusse L, Leblanc C, Bouchard C. Relationships between body fatness, adipose tissue distribution and blood pressure in men and women. *J Clin Epidemiol* 1988; **41**: 889–897.
- 12 Kalkhoff RK, Hartz AH, Rupley D, Kissebah AH, Kelber S. Relationship of body fat distribution to blood pressure, carbohydrate tolerance, and plasma lipids in healthy obese women. *J Lab Clin Med* 1983; **102**: 621–627.

- 13 Evans DJ, Hoffman RG, Kalkhoff RK, Kissebah AH. Relationship of body fat topography to insulin sensitivity and metabolic profiles in premenopausal women. *Metabolism* 1984; **33**: 68–75.
- 14 Stern MP, Haffner SM. Body fat distribution and hyperinsulinemia as risk factors for diabetes and cardiovascular disease. *Arteriosclerosis* 1986; **6**: 123–130.
- 15 Kissebah AH, Peiris AN, Evans DJ. Mechanisms associating body fat distribution to glucose intolerance and diabetes mellitus: Window with a view. *Acta Med Scand* 1988; **S723**: 79–89.
- 16 Damon A. Constitutional medicine. In: von Mering O, Kasdan L (eds). *Anthropology and the Behavioral and Health Sciences*. University of Pittsburgh Press: Pittsburgh, 1970, 179–205.
- 17 Bailey SM. Human physique and susceptibility to noninfectious disease. *Yearbook Phys Anthropol* 1985; **28**: 149–173.
- 18 Sheldon WH, Stevens S, Tucker WB. *The Varieties of Human Physique*. Harper: New York, 1940.
- 19 Parnell RW. *Behavior and Physique: An Introduction to Practical and Applied Somatometry*. Edward Arnold: London, 1958.
- 20 Carter JEL, Heath BH. *Somatotyping: Development and Applications*. Cambridge University Press: New York, 1990.
- 21 Gertler MM, Garn SM, White PD. Young candidates for coronary heart disease. *JAMA* 1951; **147**: 621–625.
- 22 Spain DM, Bradess VA, Huss G. Observations on atherosclerosis of the coronary arteries in males under the age of 46: A necropsy study with special reference to somatotypes. *Ann Intern Med* 1953; **38**: 254–277.
- 23 Spain DM, Bradess VA, Greenblatt IJ. Postmortem studies on coronary atherosclerosis, serum beta lipoproteins and somatotypes. *Am J Med Sci* 1955; **229**: 294–301.
- 24 Spain DM, Nathan DJ, Gellis M. Weight, body type and the prevalence of coronary atherosclerotic heart disease in males. *Am J Med Sci* 1963; **245**: 63–69.
- 25 Gertler MM, White PD. *Coronary Heart Disease in Young Adults: A Multidisciplinary Study*. Harvard University Press: Cambridge, MA, 1954.
- 26 Damon A. Delineation of the body build variables associated with cardiovascular diseases. *Ann NY Acad Sci* 1965; **126**: 711–727.
- 27 Damon A, Damon ST, Harpending HC, Kannel WB. Predicting coronary heart disease from body measurements of Framingham males. *J Chron Dis* 1969; **21**: 781–802.
- 28 Smit PJ, Daehne HO, Hallhuber MJ, Stocksmeier U. Somatotypes of cardiac infarction patients. In: Smit PJ (ed). *Sport and Somatology in Ischaemic Heart Disease*. University of Pretoria: Pretoria, 1979, pp 1–14.
- 29 Tanner JM. The relation between serum cholesterol and physique in healthy young men. *J Physiol* 1951; **115**: 371–390.
- 30 Gertler MM, Garn SM, Sprague HB. Cholesterol, cholesterol esters and phospholipids in health and in coronary heart disease II. Morphology and serum lipids in man. *Circulation* 1950; **2**: 380–391.
- 31 Malina RM, Katzmarzyk PT, Song TMK, Thériault G, Bouchard C. Somatotype and cardiovascular risk factors in healthy adults. *Am J Hum Biol* 1997; **9**: 11–19.
- 32 Katzmarzyk PT, Malina RM, Song TMK, Bouchard C. Somatotype and indicators of metabolic fitness in youth. *Am J Hum Biol* 1988; **10**: 341–350.
- 33 Fredman M. Somatotypes in a group of Tamil diabetics. *S Afr Med J* 1972; **46**: 1836–1837.
- 34 Fredman M. Body constitution and blood glucose and serum insulin levels in a group of Tamil Indians. In: Vague J, Boyer J (eds). *The Regulation of the Adipose Tissue Mass*. American Elsevier: New York, 1974, pp 194–197.
- 35 Robinson SC, Brucer M. Body build and hypertension. *Arch Intern Med* 1940; **66**: 393–417.
- 36 Harlan WR, Osborne RK, Graybiel A. A longitudinal study of blood pressure. *Circulation* 1962; **26**: 530–543.
- 37 Seltzer CC. Some re-evaluations of the build and blood pressure study, 1959 as related to ponderal index, somatotype and mortality. *N Engl J Med* 1966; **274**: 254–259.
- 38 Mueller WH, Joos SK. Android (centralized) obesity and somatotypes in men: Association with mesomorphy. *Ann Hum Biol* 1985; **12**: 377–381.
- 39 Sheldon WH, Dupertuis CW, McDermott E. *Atlas of Men*. Harper Brothers: New York, 1954.
- 40 Rosique J, Rebato E, Arraiz AG, Pacheco JL. Somatotype related to centripetal fat patterning of 8- to 19-year-old Basque boys and girls. *Am J Hum Biol* 1994; **6**: 171–181.
- 41 Bouchard C. Genetic epidemiology, association, and sib-pair linkage: Results from the Québec Family Study. In: Bray GA, Ryan DH (eds). *Molecular and Genetic Aspects of Obesity*. Pennington Center Nutrition Series, Vol 5, 1996. Louisiana State University Press: Baton Rouge, Louisiana, pp 470–481.
- 42 Bouchard C. Reproducibility of body composition and adipose tissue measurements in humans. In: Roche AF (ed). *Body Composition Assessments in Youth and Adults*. Ross Laboratories: Columbus, 1985, pp 9–13.
- 43 Garn SM, Ryan AS, Robson JKR. Fatness-dependence and utility of the subscapular/triceps ratio. *Ecol Food Nutr* 1982; **12**: 173–177.
- 44 Malina RM. Regional body composition: Age, sex and ethnic variation. In: Roche AF, Heymsfield SB, Lohman TG (eds). *Human Body Composition*. Human Kinetics: Champaign, 1996, pp 217–255.
- 45 Deutsch MI, Mueller WH, Malina RM. Androgyny in fat patterning is associated with obesity in adolescents and young adults. *Ann Hum Biol* 1985; **12**: 275–286.
- 46 Baumgartner RN, Roche AF, Guo S, Lohman T, Boileau RA, Slaughter MH. Adipose tissue distribution: The stability of components by sex, ethnicity and maturation stage. *Hum Biol* 1986; **58**: 719–735.
- 47 Kirkendall NM, Burton AC, Epstein FH, Freis ED. Recommendations for human blood pressure determination by sphygmomanometers: Report of a subcommittee of the Post-Graduate Education Committee, American Heart Association. *Circulation* 1967; **36**: 980–988.
- 48 Burstein M, Scholnick HR, Morfin R. Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. *J Lipid Res* 1970; **11**: 583–595.
- 49 Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972; **18**: 499–502.
- 50 Leclerc S, Bouchard C, Talbot J, Gauvin R, Allard C. Association between serum high-density lipoprotein cholesterol and body composition in adult men. *Int J Obes* 1983; **7**: 555–561.
- 51 Richterich R, Dauwalder H. Zur Bestimmung der plasmaglukosekonzentration mit der Hexokinase-Glucose-6-phosphat-Dehydrogenase-Methode. *Schweiz Med Wochensh* 1971; **10**: 615–618.
- 52 Cressie NAC, Withers TT, Craig NP. The statistical analysis of somatotype data. *Yearbook Phys Anthropol* 1986; **29**: 197–208.
- 53 SPSS. *SPSS Reference Guide*. SPSS Inc: Chicago 1990.
- 54 Seltzer CC, Mayer J. Body build and obesity-Who are the obese? *JAMA* 1964; **189**: 677–684.
- 55 Seltzer CC, Mayer J. Body build (somatotype) distinctiveness in obese women. *J Am Diet Assoc* 1969; **55**: 454–458.
- 56 Sangi H, Mueller WH, Harrist RB, Rodriguez B, Grunbaum JG, Labarthe DR. Is body fat distribution associated with cardiovascular risk factors in childhood? *Ann Hum Biol* 1992; **19**: 559–578.