Computed Tomography–Determined Body Composition in Relation to Cardiovascular Risk Factors in Indian and Matched Swedish Males

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Relationships between cardiovascular risk factors, body composition, and tissue distributions were examined in 10 Indian and 10 Swedish males matched by age, height, and weight. The body was divided into 29 compartments by means of a multiscan computed tomography (CT) technique. Fasting glucose, insulin, and triglycerides (TG) were higher in Indians than in Swedes. During the oral glucose tolerance test (OGTT), the glucose area was similar in both groups, whereas the insulin area was 80% larger in Indians. Adipose tissue (AT) and skin volumes were larger and remaining lean tissues were smaller in Indians. Indians had proportionally less muscle and more skeleton in the legs, but no ethnic difference could be demonstrated with respect to AT distribution. The visceral AT to total AT volume ratio was positively related to insulin and TG, and with higher risk factors for Indians at any given ratio. TG and glucose were negatively related to the leg muscle to total muscle volume ratio, and this ratio was smaller in Indians. It is concluded that the metabolic disturbances of Indians are not necessarily dependent on a preponderance of visceral AT, and also that an upper-body muscle distribution—recognized as a new phenotypic companion to the metabolic syndrome—is statistically related to cardiovascular risk factors.

MORTALITY STATISTICS from England and Wales have revealed a 40% excess risk of death from ischemic heart disease among inhabitants originating from the Indian subcontinent.[1] South Asian migrants who have settled in other parts of the world have also been shown to have an increased ischemic heart disease risk relative to the indigenous populations.[2-4] Of the known major cardiovascular risk factors, non–insulin-dependent diabetes mellitus is strikingly and consistently more prevalent among South Asians[5] and South Asian migrants,[6-8] whereas the prevalences of hypertriglyceridemia, low high-density lipoprotein (HDL) cholesterol, and smoking are higher in some but not all examined subgroups from India.[9] Despite similar body mass indices (BMIs), the age-standardized prevalence of impaired glucose tolerance and diabetes mellitus is two to four times higher among Indians living in London than among Europeans.[8]

The cardinal features of the metabolic syndrome—insulin resistance, hyperinsulinemia, impaired glucose tolerance, diabetes, hypertension, hypertriglyceridemia, and/or low HDL cholesterol—are more closely related to the waist to hip ratio than to BMI, both in Indians[7] and Europeans.[7,10] Similarly, cardiovascular morbidity and mortality are more closely related to the waist to hip ratio,[11,12] and, as compared with Europeans, Indians (Sikhs, Punjabi Hindus, Gujarati Hindus, and Muslims) usually have a larger waist to hip ratio.[9]

These findings have caused several groups to suggest an increased intraabdominal adipose tissue (AT) of Indians as a causal factor in the development of diabetes and other characteristics of the metabolic syndrome. However, this is far from clear for several reasons. Despite the fact that the waist to hip ratio is a strong risk predictor,[10-12] it is a poor estimator of visceral AT volume[13] as determined with multiscan computed tomography (CT) techniques.[14-16] These circumstances may indicate that the waist to hip ratio carries risk information that we cannot currently interpret. Furthermore, several neuroendocrine disturbances may cause both peripheral insulin resistance and accumulation of visceral AT.[17] Although visceral AT per se may cause metabolic disturbances,[17] its accumulation may also to a considerable extent be a parallel phenomenon to the development of other disturbances of the metabolic syndrome. Finally, some ethnic groups have a markedly increased incidence of diabetes mellitus despite waist to hip ratios similar to those in whites.[18] Thus, it is not clear if the increased incidences of cardiovascular disease and type II diabetes in Indians are necessarily related to an increased visceral AT volume.

There are circumstances other than the waist to hip ratio that indicate a different body composition in Indians. Thus, as compared with whites, Asians display a lower daily creatinine excretion,[19] different pharmacokinetics,[20] and a lower basal metabolic rate in some,[21,22] but not all[23] studies. However, studies directly comparing body composition in white and Indians are scanty and contradictory (see the Discussion).

Considering these background circumstances, the aim of the current study was to compare body composition and cardiovascular risk factors of Indian males living in Sweden with those of matched Swedish males by using a multicompartment technique based on multiple CT scans.[16] This technique is presently extremely time-consuming (1 to 2 weeks per subject), but permits the determination of a large number of compartments at the tissue and organ level and has an unusually high validity and reproducibility. As compared with Swedes, Indians with similar weight, height, and age had elevated risk factors, less lean tissue, and more AT. However, the fraction of AT located in viscera was not increased. Metabolic disturbances were associated with AT and muscle distribution.

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SUBJECTS AND METHODS

Study Groups

Ten healthy males originating from the Indian subcontinent were available for examination. These subjects had been living in Göteborg for at least 10 years, and some for their whole lives. All were employed and well adjusted to the Swedish society. None were on medication. Swedish males matched to the Indians with respect to age, height, and weight were recruited by advertisement in a local newspaper. From 163 answers, a Swedish male was found for each Indian male who did not deviate more than 3 years in age, 3 cm in height, or 4.4 kg in weight (Table 1). Average age, height, and weight were not significantly different between Indians and Swedes (Table 1). The standard errors of a single determination (SESDs)[24] calculated on the squared differences between Indians and Swedes were 0.6% (height), 2.2% (weight), and 3.8% (age). Both individuals within pair 4 and the Indian male within pair 2 were smokers (Table 1).

Food intake of the subjects was recorded by using a previously validated questionnaire.[24] The estimated energy intake in Indians and Swedes was 2,440 ± 560 and 2,520 ± 620 kcal/d, respectively ($P = .74$). The percentage composition of energy (energy%) from carbohydrate, fat, and protein was 50.7 ± 6.0, 32.1 ± 4.8, and 15.0 ± 1.3 in Indians and 48.1 ± 6.1, 33.8 ± 5.5, and 14.8 ± 2.5 in Swedes, respectively (nonsignificant).

The physical activity level was estimated by a questionnaire ranking activities during work from 0 to 4, and activities during leisure time from 1 to 4. The scores during work were 1.4 ± 0.6 and 1.7 ± 0.7 in Indians and Swedes, respectively ($P = .22$). Corresponding scores during leisure time were 2.0 ± 0.5 and 2.3 ± 0.5, respectively ($P = .24$).

Anthropometric Measurements

Body weight (BW) was determined in underwear to the nearest 0.1 kg after voiding, using the same calibrated scale for all subjects. Body height was measured without shoes to the nearest 0.01 m. Circumferences and sagittal diameter were measured in recumbent subjects on a firm examination table after a normal expiration.

Table 1. Age, Height, and BW in 10 Healthy Males of Indian Origin and in 10 Matched Healthy Swedish Males

Pair No.	Age (yr)		Height (m)		BW (kg)		CT-Estimated BW (kg)	
	I	S	I	S	I	S	I	S
1	38	39	1.76	1.77	75.8	74.5	75.9	74.7
2	58‡	61	1.80	1.79	70.5	70.0	70.5	69.3
3	42	45	1.82	1.81	68.1	69.5	66.7	69.4
4	42‡	39‡	1.76	1.78	58.9	58.8	59.6	58.3
5	21	24	1.81	1.82	69.5	69.1	69.1	70.3
6	40	38	1.82	1.83	82.2	81.0	81.8	80.8
7	38	41	1.78	1.79	78.0	81.0	77.5	81.2
8	52	53	1.68	1.65	78.3	75.0	77.4	76.3
9	48	47	1.66	1.67	64.0	62.0	63.6	61.7
10	36	36	1.81	1.82	74.4	70.0	76.0	71.8
Mean	42	42	1.77	1.77	72.0	71.1	71.8	71.4
SD	10	10	0.06	0.06	7.1	7.2	7.1	7.4
P	.29*		.54*		.22*		.53†	.30†
SESD (%)	3.8*		0.6*		2.2*		0.8†	0.9†

Abbreviations: I, Indians; S, Swedes.

*SESD and *P* calculated on differences between Indians and Swedes.

†SESD and *P* calculated on differences between CT-estimated BW and true BW.

‡Smokers.

Waist girth was measured midway between the caudal part of the thorax and the iliac crest as palpated laterally. Hip girth was measured over the trochanter femoris. The sagittal diameter was measured by means of a carpenter's spirit level and a ruler. The level was placed perpendicular to and over the trunk at the level of the iliac crest. The sagittal diameter was the vertical distance from the horizontal level to the examination table. Sitting height was not measured, but the distance from the lower border of the symphysis to the vertex of the skull was measured on the longitudinal CT scans (see below). The ratio of the vertex-symphysis distance over height was calculated.

Multicompartment Body Composition Examinations by CT

All subjects were examined with a 28-scan CT technique permitting compartmentalization of the body into 12 main compartments, several of which could be further divided by region. The CT technique for multicompartmentation has been described previously.[15,16] In brief, areas of tissues, organs, and gas were measured in 28 cross-sectional scans having defined positions. The area determinations were performed in the following attenuation intervals (in Hounsfield units [HU]): air, gas, and lungs, −1,001 to −191 HU; AT, −190 to −30 HU; all other soft tissues and organs, −29 to +151 HU; and skeleton, 152 to 2,500 HU. Various tissue and organ areas in the −29 to +151-HU interval were obtained by means of cursor circumscriptions, whereas area determinations in other intervals were based on the number of pixels fulfilling given attenuation criteria. Volumes of tissues, organs, and gas were obtained from corresponding areas and the distances between the scans. Precision errors calculated from double determinations ranged from 0.01 to 0.32 L, depending on the tissue under consideration.[16] Lean body volume (LBV) is defined as the non-AT volume.

The CT technique was validated in this study by multiplying each tissue and organ volume with its density as reported previously.[16] The resulting organ weights were summed to obtain individually CT-estimated BWs. These CT-estimated BWs were compared with the actually measured BWs. The SESD was calculated from the differences between CT-estimated BW and true BW.[25] Lean body mass (LBM) determined by CT (LBM_{CT}) was calculated from the components of LBV and their densities. The effective dose equivalent of an examination was 3 to 5 mSv.

Total Body Potassium

Total body potassium (TBK) was calculated from measurements of ^{40}K levels as described previously.[26]

Risk Factors

Systolic and phase 5 diastolic blood pressure were registered in the recumbent position after 45 minutes' rest. Fasting serum cholesterol, triglycerides (TG), insulin and fasting blood glucose were determined with standard techniques at the central laboratory of Sahlgrenska Hospital, Göteborg, Sweden. This laboratory is certified according to Euronom (EN) 45001. A 100-g oral glucose tolerance test (OGTT) was performed in each subject. Glucose and insulin levels were measured at 0, 15, 30, 60, 90, 120, 180, 240, 300, and 360 minutes. The area under the curve (AUC) was calculated with an algorithm based on the area of the trapezium.

Statistical Analyses

All calculations were performed with the Minitab,[27] JMP,[28] or SPSS[29] statistical packages. The means ± SD and confidence intervals have been used for descriptive purposes. Variables were checked for normality with an N-score procedure[27] similar to the

Shapiro-Wilk test and were log-transformed when necessary. The significance of differences between matched subjects have been calculated with Student's paired t test or, when appropriate, with Wilcoxon's nonparametric test for paired comparisons. Pearson's correlation coefficients were calculated, and linear bivariate and multivariate regression analyses were performed. The SESD was calculated according to the method of Dahlberg[25] as

$$\sqrt{\frac{\Sigma d^2}{2n}}$$

where d is the difference between paired observations and n is the number of pairs.

It can be demonstrated that with 10 pairs, a paired comparison is more efficient than a group comparison if the correlation between a given body compartment of Indians and Swedes is higher than .138 (Anders Odén, statistician, personal communication, December 1994). In this study, correlations between body compartments of Indians and Swedes were in the range of .283 to .919. With a general linear model (GLM) adjusting for covariates, the paired comparison is broken up. When using the GLM and adjusting for weight only, compartment differences between Indians and Swedes were much less significant than with the paired t test despite slightly larger differences between most means. This was interpreted as an increased risk for type II errors when using the GLM and one covariate. If compartment means were adjusted for weight, height, and age, then similar significance levels as with the paired t test were obtained, but since three covariates seem inappropriate for 10 pairs of observations, the paired t test was preferred for the main comparisons of this study.

Regional depot volumes of a given tissue (AT, skeletal muscle, or skeleton) were expressed as a percent of the total volume of that tissue for each individual. In the case of skeleton and muscles, four regions were considered (legs, arms, trunk, and neck + head), and in the case of AT, five regions were taken into account (visceral AT and subcutaneous AT of legs, arms, trunk, and neck + head). For a given tissue and for each region, the intrapair difference (Indian minus Swede) was calculated and expressed in percent units. The sum of the four (muscles and skeleton) or five (AT) regional differences was equal to zero for each pair. Thus, in the case of AT, we had a multivariate variable for each pair of subjects with the mean vector ($\mu_1, \mu_2, \ldots \mu_5$). The hypothesis, $\mu_i = 0$, $i = 1, \ldots 4$ (which implies $\mu_5 = 0$) was tested using Hotelling's test applied on the four-dimensional vector of four regions.[29] The outcome of the test was not dependent on which one of five AT regions was excluded. Rejection of the H_0 hypothesis would imply a different AT distribution in Indians as compared with Swedes. Obvious advantages with this method were that multiple testing was avoided and the procedure was applicable on statistically dependent variables. The test variable had an F distribution with *df*s equal to ([number of regions] -1) and ([number of patients] $-$ [4 + 1]). Racial differences in the distribution of muscles and skeleton were analyzed in a similar way.

A simpler but less formal way to judge whether a given tissue was differently distributed in Indians as compared with Swedes was to calculate confidence intervals of the intrapair differences in all examined regions. If at least one of the five (AT) or four (muscle or skeleton) confidence intervals did not include zero, a racial difference in tissue distribution was at hand.

Human Subjects

This study was approved by the Ethics Committee at Göteborg University. Informed consent was obtained from all participants.

RESULTS

Validation of the CT Technique

In Indians, true and CT-estimated BWs were 72.0 ± 7.1 and 71.8 ± 7.1 kg, respectively, and in Swedes the corresponding figures were 71.1 ± 7.2 and 71.4 ± 7.4 kg, respectively (Table 1). The SESD calculated on the difference between CT-estimated and true BW was 0.8% and 0.9% in Indians and Swedes, respectively. When Indians and Swedes were pooled, CT-estimated and true BWs were 71.6 ± 7.1 and 71.5 ± 7.0 kg, respectively. The mean difference was 0.06 ± 0.86 kg, and the SESD was 0.8%.

Physique and TBK

As expected, the matching procedure (Table 1) resulted in similar BMIs in Indians and Swedes both on average (Table 2) and within each pair (Fig 1A). Neither waist, hip, waist to hip ratio, sagittal diameter, trunk + head distance, nor (trunk + head)/height were significantly different between Indian and Swedish males (Table 2). Despite similar age, height, weight (Table 1) and BMI (Table 2), TBK was lower in Indians (3,762 mmol) than in Swedes (3,894 mmol) ($P = .026$; Table 2 and Fig 1B).

Gross Tissue Compartments

The total AT volume was larger in Indians (17.8 L) than in Swedes (14.4 L) ($P = .006$; Table 3). In accordance with the lower TBK in Indians (Table 2), the CT-determined LBV (ie, non-AT) tended to be lower in Indians (51.6 L) than in Swedes (54.0 L) ($P = .057$; Table 3). On average, the volume of skeletal muscles, visceral organs, and skeleton were or tended to be larger in Swedes and skin volume tended to be larger in Indians (Table 3). After exclusion of the skin volume, the remaining LBV was significantly larger in Swedes ($P = .026$; Table 2).

TBK of the pooled study group (N = 20) regressed by LBM_{CT} (kg) and race (coded as Indians = 1, Swedes = 2) resulted in the equation, $TBK = -81 + 71.0 \cdot LBM_{CT} - 46.5 \cdot$ race, where the P values of LBM_{CT} and race were less than .0001 and .51, respectively, and $R^2 \cdot 100$ adjusted for *df* was 87.2%. Similarly, K/LBV was 73.0 ± 3.3 mmol/L in Indians and 72.2 ± 3.4 mmol/L in Swedes ($P = .83$, NS).

Volumes of Visceral Organs and Gas

On average, abdominal and thoracic organs were smaller in Indians than in Swedes, but these differences reached significance only for the retroperitoneal plus intraperitoneal organs (4.0 and 4.9 L, respectively, $P = .006$) and for the heart (0.54 and 0.68 L, respectively, $P = .006$) (Table 4). No ethnic differences were observed with respect to the volumes of brain, contents of the spinal channel, or gastrointestinal gas. The volume of air in the sinuses plus upper airways tended to be ($P = .058$) larger in Swedes. After correction for LBV, heart volumes were 10.5 ± 2.2 mL/L in Indians and 12.7 ± 1.9 mL/L in Swedes ($P = .032$).

Tissue Distributions

AT. The total AT volume (Table 3), as well as the total subcutaneous AT volume (Table 5), were larger in Indians

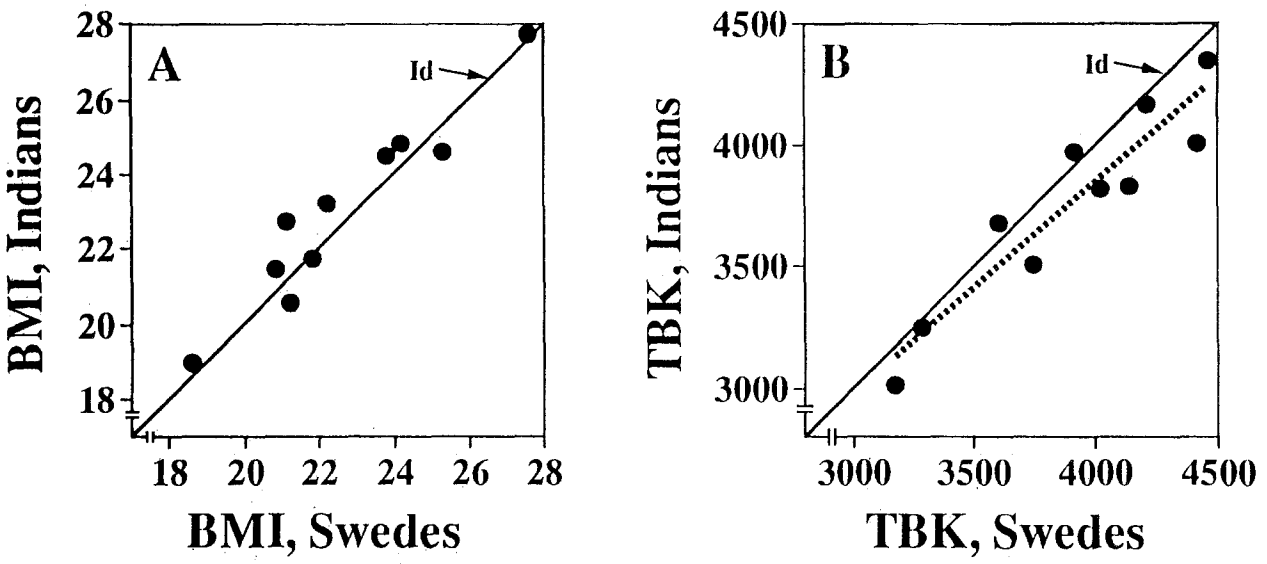

Fig 1. BMI (kg/m²) and TBK (mmol) in Indian (y axis) and Swedish (x axis) males. (—) Line of identity. (A) Regression coefficient and intercept not significantly different from 1 and 0, respectively. SESD = 2.4%; $R^2 \cdot 100$ adjusted for *df* = 91.6%. BMI, 23.0 ± 2.5 (Indians) and 22.7 ± 2.6 (Swedes) kg/m², NS. (B) Equation (-----): y = 401 + 0.86×; $R^2 \pm 100$ adjusted for *df* = 85.9%. TBK, 3,762 ± 410 (Indians) and 3,894 ± 444 (Swedes) mmol (*P* = .026).

than in Swedes. The larger subcutaneous AT in Indians was due to an increase of the true subcutaneous AT (subcutaneous proper AT: Indians, 12.3 L; Swedes, 10.0 L; $P = .003$), whereas the AT volume between muscle bundles (intermuscular AT) was similar in the two races (Table 5). On average, the true and total subcutaneous AT of Indians was larger in the legs, trunk, arms, and head/neck, but full significance against Swedes was achieved only in the trunk and arms (Table 5). A 26% larger intraabdominal and intraperitoneal AT and a 20% larger retroperitoneal AT in Indians did not reach significance, whereas a 16% larger intrathoracic AT volume was significant ($P = .045$; Table 5).

When each AT depot was expressed as a percent of the total AT volume, the depots constituted the following percentages in Indians and Swedes, respectively: legs, 31.4% ± 5.4% and 32.0% ± 4.5%; subcutaneous trunk, 39.0% ± 3.7% and 39.5% ± 4.1%; viscera, 20% ± 5.5% and 19.6% ± 3.9%; arms, 6.6% ± 1.4% and 6.3% ± 1.0%; head and neck, 2.6% ± 2.5% and 2.7% ± 1.4%. AT distribution was not significantly different between Indians and Swedes, neither with Hotelling's test nor with the confidence interval technique.

Table 2. Physique and TBK in 10 Healthy Males of Indian Origin and in 10 Matched Healthy Swedish Males

Variable	I	S	*P*
BMI (kg/m²)	23.0 ± 2.5	22.7 ± 2.6	NS
Waist (cm)	83.9 ± 6.7	82.4 ± 5.3	.30
Hip (cm)	94.5 ± 4.6	95.0 ± 4.9	.62
Waist to hip ratio	0.89 ± 0.04	0.87 ± 0.04	.30
Sagittal trunk diameter (cm)	19.1 ± 1.2	18.5 ± 1.9	.33
Trunk + head (cm)*	86.5 ± 4.3	87.9 ± 2.6	.29
Trunk + head, m/height (m)	0.488 ± 0.02	0.496 ± 0.02	.38
TBK (mmol)	3,762 ± 410	3,894 ± 444	.026

*Distance from lower border of symphysis to vertex of skull.

Skeletal muscles. Although the total skeletal muscle volume was not significantly lower in Indians (30.3 L) than in Swedes (31.1 L; $P = .17$), the muscle volume of legs was lower in Indians ($P = .014$; Table 3). On the other hand, the muscle volume of the arms tended to be larger in Indians (4.1 L) than in Swedes (3.8 L) ($P = .077$; Table 3). When

Table 3. Gross Tissue Compartments of the Body and Some Tissue Subcompartments in 10 Healthy Males of Indian Origin and 10 Matched Swedish Males (mean ± SD)

Variable	Unadjusted Values		*P*
	Indians	Swedes	
Adipose tissue (L)	17.8 ± 5.0	14.7 ± 4.0	.0055
LBV (L)	51.6 ± 5.4	54.0 ± 5.7	.057
Skeletal muscle	30.3 ± 3.4	31.1 ± 4.2	.17
Visceral organs	10.9 ± 1.9	12.2 ± 1.4	.076
Skeleton	8.0 ± 1.0	8.5 ± 0.7	.036
Skin	2.4 ± 0.4	2.2 ± 0.3	.078
LBV minus skin (L)	49.1 ± 5.2	51.8 ± 5.4	.026
Regional subcompartments of muscle			
Legs	12.7 ± 1.3	13.7 ± 1.9	.014
Trunk	12.6 ± 1.6	12.8 ± 1.8	.55
Arms	4.1 ± 0.4	3.8 ± 0.6	.077
Head + neck	0.96 ± 0.16	0.88 ± 0.11	.15
Regional subcompartments of skeleton			
Legs	3.5 ± 0.6	3.5 ± 0.5	.75
Trunk	3.0 ± 0.4	3.3 ± 0.2	.050
Arms	0.73 ± 0.09	0.81 ± 0.10	.13
Head + neck	0.74 ± 0.18	0.94 ± 0.20	.016
Functional subcompartments of skeleton			
Dense skeleton	7.4 ± 1.0	7.9 ± 0.9	.099
Yellow bone marrow	0.09 ± 0.04	0.10 ± 0.05	.67
Red bone marrow	0.43 ± 0.18	0.53 ± 0.28	.34

Table 4. Gas Volumes (L) and Viseral Organ Volumes (L) Except AT in 10 Healthy Males of Indian Origin and 10 Matched Swedish Males (mean ± SD)

Variable	Indians	Swedes	*P*
All visceral organs (except AT and gas)	10.9 ± 1.9	12.2 ± 1.4	.076
Abdomen			
Retroperitoneal plus intraperitoneal organs	4.0 ± 0.7	4.9 ± 0.5	.006
Gastrointestinal gas	0.27 ± 0.11	0.28 ± 0.15	.859
Thorax			
Heart	0.54 ± 0.11	0.68 ± 0.07	.006
Lungs plus air in bronchi and lower trachea	4.3 ± 1.5	4.6 ± 1.3	.64
Other thoracic organs	0.34 ± 0.10	0.37 ± 0.09	.42
Central nervous system			
Brain	1.46 ± 0.15	1.47 ± 0.16	.86
Contents of spinal channel	0.14 ± 0.02	0.15 ± 0.02	.27
Air in sinuses and upper airways	0.19 ± 0.07	0.26 ± 0.07	.058

muscle depot volumes were expressed as a percentage of the total muscle volume, Indians had 2.2% ± 2.1% units less muscles in the legs than Swedes and 0.32%, 0.48%, and 1.40% units more muscles in the neck + head, trunk, and arms, respectively (Fig 2). Muscle distribution was significantly different in Indians as compared with Swedes both according to Hotelling's test ($F = 5.91, P < .05$) and according to the confidence interval procedure ($P < .01$).

Skeleton. The lower total volume of the skeleton in Indians seemed to be explained by the "dense" skeleton rather than the "yellow" or "red" bone marrow as defined by the CT procedure (Table 3).

From a regional point of view, the skeletal volumes of Indians were lower in the trunk ($P = .050$) and neck + head ($P = .016$), but not significantly so in the arms ($P = .13$) or legs ($P = .75$) (Table 3). Hotelling's test did not reveal any difference in the distribution of skeleton between Indians and Swedes ($F = 2.59$, NS), whereas the confidence interval technique indicated ($P < .05$) that Indians had a lower fraction of skeleton in the neck + head and a higher fraction in the legs (Fig 3). Thus, when expressed as a percent of the total skeletal volume, Indians had 2.3% ± 3.9% units more skeleton in the legs and 1.7% ± 2.2% units less skeleton in the neck + head as compared with Swedes.

Risk Factors

Systolic blood pressure was lower in Indian than in Swedish males, but no ethnic differences could be demonstrated with respect to diastolic blood pressure or total cholesterol (Table 6). Fasting TG were 60% higher in Indians than in Swedes (Table 6). Although fasting glucose was higher (Table 6) and the 30-minute glucose value tended to be higher in Indians (Fig 4A), there was no ethnic difference in the glucose AUC during a 100-g OGTT over 6

Table 5. Regional Volume (L) of Adipose Tissue (AT) and Its Subcompartments in 10 Healthy Males of Indian Origin and 10 Matched Swedish Males (mean ± SD)

Variable	Total			Legs			Trunk			Arms			Neck		
	I	S	*P*	I	S	*P*	I	S	*P*	I	S	*P*	I	S	*P*
Subcutaneous AT															
Total	14.1 ± 3.9	11.8 ± 3.1	.007	5.5 ± 1.6	4.6 ± 0.9	.084	6.9 ± 2.0	5.9 ± 2.2	.006	1.2 ± 0.6	0.9 ± 0.2	.041	0.48 ± 0.5	0.38 ± 0.2	.60
Proper	12.3 ± 3.6	10.0 ± 2.8	.003	4.6 ± 1.4	3.7 ± 0.9	.063	6.1 ± 1.9	5.1 ± 2.0	.009	1.17 ± 0.59	0.87 ± 0.24	.032	0.44 ± 0.53	0.35 ± 0.16	.63
Intermuscular	1.75 ± 0.42	1.74 ± 0.52	.97	0.90 ± 0.23	0.93 ± 0.23	.78	0.78 ± 0.22	0.74 ± 0.33	.71	0.04 ± 0.03	0.03 ± 0.02	.90	0.03 ± 0.03	0.03 ± 0.02	.33
Intraabdominal AT															
Total							3.4 ± 1.5	2.7 ± 1.1	.16						
Intraperitoneal							2.4 ± 1.0	1.9 ± 0.9	.18						
Retroperitoneal							1.0 ± 0.52	0.83 ± 0.27	.20						
Intrathoracic AT															
Total							0.29 ± 0.06	0.25 ± 0.06	.045						
Cardiac							0.11 ± 0.03	0.09 ± 0.03	.23						
Other thoracic							0.19 ± 0.05	0.16 ± 0.04	.10						

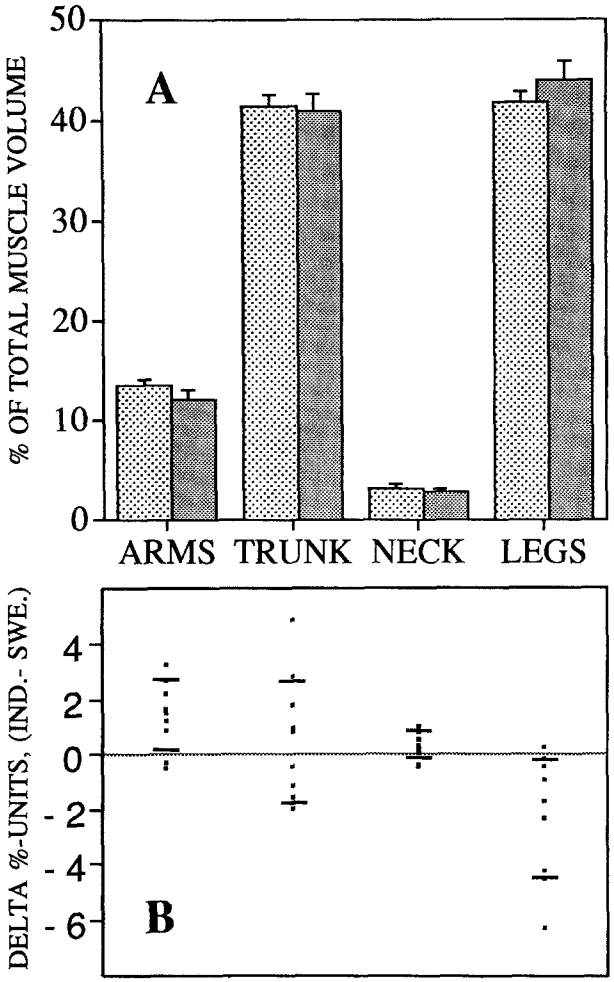

Fig 2. Distribution of skeletal muscles over four regions in Indian and Swedish males. (A) Muscle volume of arms, trunk, neck, and legs expressed as a % of total muscle volume in Indians (▩) and Swedes (▩). (B) Individual intrapair differences from panel A calculated as Indians minus Swedes and expressed in delta % units. Horizontal lines indicate upper and lower 99% confidence limits. The sum of all differences was equal to zero, the undulating line. Muscle distribution was significantly different between Indians and Swedes both according to Hotelling's test ($F = 5.91$, $P < .05$) and with the confidence interval procedure ($P < .01$).

hours (Fig 4A and Table 6). In contrast, insulin AUC was 80% higher in Indians, as was the fasting insulin value (Fig 4B and Table 6).

Relationships Between Body Composition and Risk Factors

To obtain enough subjects for regression analysis, Indians and Swedes were pooled (N = 20). In a preliminary survey, all risk factors were regressed by the absolute amount of muscles in the four regions or the absolute amount of AT in the five regions examined. It then turned out that some risk factors were negatively related to leg muscle volume and positively but not significantly to all other muscle volume. Similarly, some risk factors were positively related to intraabdominal AT, but usually not to other regional AT volumes. Considering the limited number of subjects, the number of independent variables were reduced by creating two ratios describing tissue distribution: leg muscle vol/total muscle vol and intraabdominal AT vol/total AT vol.

Table 7 shows that serum TG were negatively related to a preponderance of leg muscles and positively to a preponderance of visceral AT. Both these relationships were independent of BMI. When ethnicity was also taken into account, muscle distribution lost its explanatory power, but AT distribution did not (Table 7). The different responses to ethnicity were explained by the fact that the visceral AT to total AT ratio covered similar ranges in Indians and Swedes and tended to be positively related to TG in both ethnic groups (Fig 5A), whereas the negative relationship between

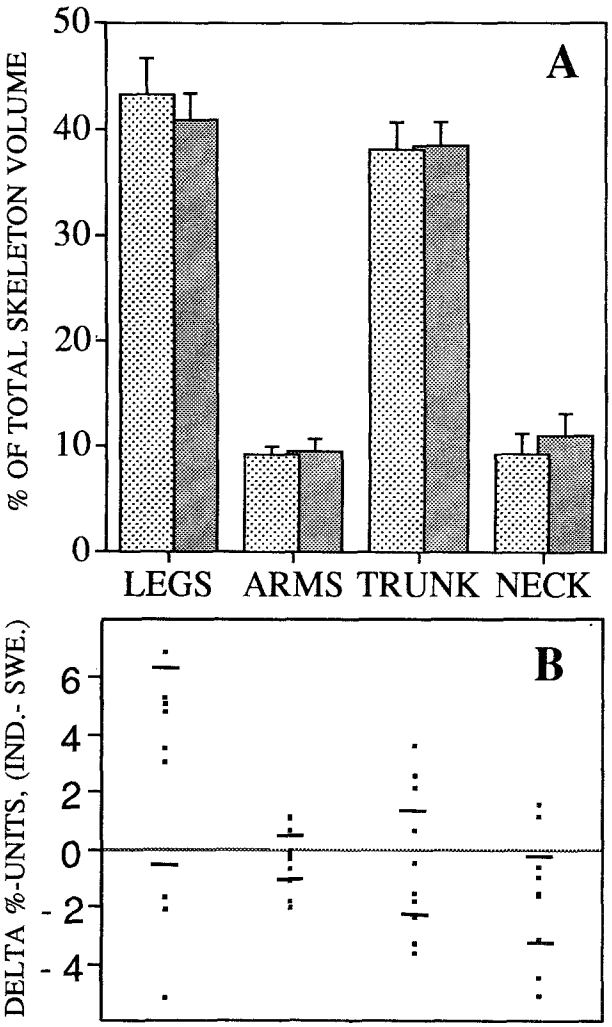

Fig 3. Distribution of skeleton ("dense" skeleton plus "yellow" and "red" bone marrow) over four regions in Indian and Swedish males. (A) Skeletal volume of legs, arms, trunk, and neck expressed as a % of total skeletal volume in Indians (▩) and Swedes (▩). (B) Individual intrapair differences from panel A calculated as Indians minus Swedes and expressed as delta % units. 95% confidence intervals are indicated. Skeleton distribution was not significantly different between Indians and Swedes according to Hotelling's test ($F = 2.59$, NS), but the confidence interval procedure indicated a difference ($P < .05$).

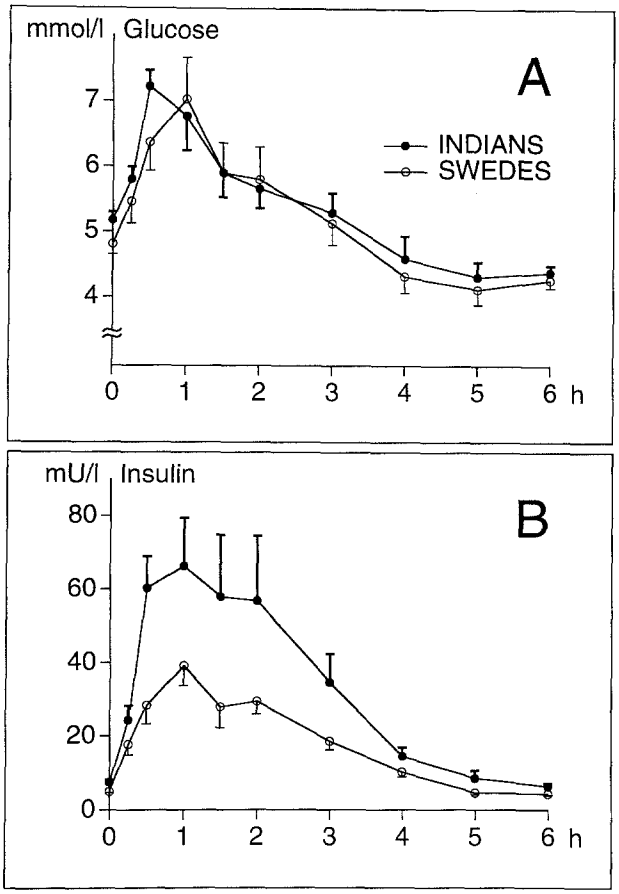

Fig 4. 100-g OGTT in Indian and Swedish males (mean ± SEM). (A) Blood glucose concentration over 6 hours in Indians and Swedes. At 0 and 30 minutes, *P* values for ethnic differences were .04 and .06, respectively. At all other points in time, *P* > .1. The AUC was 31.8 ± .1 (Indians) and 30.9 ± 1.5 mmol ± h/L (Swedes), *P* = .63. (B) Serum insulin concentration over 6 hours. AUC of insulin, 195 ± 34 (Indians) and 107 ± 11 mU ± h/L (Swedes), *P* = .03.

TG and the leg muscle to total muscle ratio was dependent on a number of Indians with low ratios and high TG (not shown). Muscle plus AT distribution explained 45% of the TG variance. The explained variance increased to 70% with BMI and ethnicity also added to the equation.

Table 6. Cardiovascular Risk Factors in 10 Healthy Males of Indian Origin and 10 Matched Swedish Males (mean ± SD)

Risk Factor	Indians	Swedes	*P*
Systolic blood pressure (mmHg)	112 ± 8	125 ± 17	.02
Diastolic blood pressure (mmHg)	75 ± 9	80 ± 7	.19
Cholesterol (mmol/L)	5.7 ± 1.3	5.6 ± 1.3	.99
TG (mmol/L)	1.6 ± 0.6	1.0 ± 0.3	.03
Glucose (mmol/L)	4.9 ± 0.4	4.5 ± 0.3	.01
Insulin (mU/L)	11.6 ± 8.5	6.2 ± 1.4	.046*
100-g OGTT during 6 hours			
Glucose AUC (mmol · h/L)	31.8 ± 3.5	30.9 ± 4.8	.63
Insulin AUC (mU · h/L)	195 ± 107	107 ± 35	.034
Insulin area to glucose area ratio (mU/mmol)	6.23 ± 3.93	3.59 ± 1.42	.051
60-minute insulin (mU/L)	66.3 ± 41.5	39.2 ± 17.0	.082

*Statistical evaluation on 10 log-values.

Whereas the visceral AT to total AT ratio only tended to be positively related to fasting insulin (Table 7), the ratio was positively related to AUC insulin (not shown) and particularly to the 60-minute insulin value during the OGTT (Table 7). The latter relationship was independent of BMI and ethnicity. Figure 5B illustrates the positive relationship between 60-minute insulin values and the visceral AT to total AT ratio in the pooled group and within Indians. The corresponding slope was positive but nonsignificant and much less steep within Swedes. Figure 5A and B indicates that TG and insulin were higher in Indians at all visceral AT fractions except the lowest.

Astonishingly, fasting blood glucose was unrelated to AT distribution but negatively related to the leg muscle to total muscle volume ratio (Table 7). The latter relationship was independent of BMI and ethnicity. Figure 5C shows that the negative relationship between fasting glucose and the leg muscle to total muscle ratio existed in the combined group and within Indians. Again, the slope was less steep and nonsignificant within Swedes. Whereas muscle distribution, AT distribution, BMI, and ethnicity explained 47% of the fasting blood glucose variance, the same independent variables explained only 25% of the AUC glucose variance (not shown).

Blood pressure and cholesterol were not related to body composition.

Neither waist circumference, waist to hip ratio, nor sagittal diameter were related to any of the cardiovascular risk factors of Table 6 in our small study group.

DISCUSSION

This study compared body composition and risk factors in 10 Indian males who were well adapted to the Swedish society with the same variables in 10 Swedish males who were carefully matched to the Indians with respect to age, height, and weight. No differences in energy intake, macronutrient distribution, or physical activity during work or leisure time could be demonstrated between the groups. All 20 males were healthy and without any form of medication. It might be important to stress that Swedes were matched to Indians with respect to weight *and* height rather than BMI, since weight and height are most likely inherited via different genes.[11]

In contrast to large British studies,[9,30] the waist to hip ratio was not significantly increased in our small group of Indian males. This may be a chance phenomenon or related to the fact that our subjects were leaner (average BMI, 22.8 kg/m^2) than in the British studies (25.9 kg/m^2). Nevertheless, our Indian males had higher fasting glucose, insulin, and TG values than the Swedish males and almost twice as large an AUC with respect to insulin values during an OGTT. Several metabolic variables were related to muscle and AT distribution.

Total AT was increased by 21% while lean tissues and TBK were correspondingly decreased in Indians. In a multivariate regression of all subjects (N = 20), TBK was closely related to LBM_{CT} but not to ethnicity, in agreement with similar TBK/LBV ratios in the two groups. As compared with Swedes, Indians had or tended to have a larger

Table 7. Multivariate Relationships Between Cardiovascular Risk Factors, Tissue Distributions, BMI, and Ethnicity in Indian and Swedish Males (N = 20)

Equation No.	Dependent Variable	Independent Variables (*t*)				$R^2 \cdot 100$	*P*
		Skeletal Muscle (leg/total)	AT (visceral/total)	BMI	Ethnicity		
1	TG	−2.42	2.01			44.6	.007
2	TG	−2.51	2.36	2.86		63.3	.001
3	TG	−1.00	2.37	3.13	−1.86	70.2	.001
4	Fasting insulin	−0.77	1.89		−2.14	50.8	.009
5	60-minute insulin	−0.54	3.23			39.4	.014
6	60-minute insulin	−0.45	2.93	0.62		40.8	.035
7	60-minute insulin	0.87	3.04	0.74	−2.07	54.0	.015
8	Fasting glucose	−3.22	−0.40			37.9	.017
9	Fasting glucose	−3.12	−0.75	1.48		45.3	.019
10	Fasting glucose	−2.12	−0.44		−0.65	39.5	.041
11	Fasting glucose	−2.01	−0.79	1.47	−0.76	47.1	.038

NOTE. *t* ratios, explained variance, and *P* values of equations shown; *t* ratios > 2.10 are significant at $P < .05$. Indians coded as 1, Swedes as 2.

fraction of skeleton and a smaller fraction of muscle in the legs, but no ethnic differences could be demonstrated with respect to AT distribution. Since the ethnic difference in tissue distribution went in opposite directions for skeleton and muscle, no simple relation with potential differences in body proportions could explain the findings. As a matter of fact, the ratio of CT-measured trunk + neck + head length over stature was not significantly different between the groups.

We are reporting on the volume of eight visceral organ compartments, 12 AT compartments, four muscle and four skeleton compartments, and one skin compartment, or a total of 29 compartments. Therefore, this is currently the most detailed body composition report on living humans. Unfortunately, the CT technique is extremely time-consuming and expensive, and therefore, it was not feasible to examine more than 20 subjects in this study.

The possibility that the observed racial differences could be related to methodologic problems rather than to genetic dissimilarities must be considered. Theoretically, more unhealthy food choices and a lower physical activity in the Indians could explain some of the observed differences. According to a food questionnaire validated against 24-hour energy expenditure,[24] there was no statistical difference in food intake between Indians and Swedes. If anything, energy intake and fat intake were lower in Indians. Furthermore, there were no differences between the groups with respect to physical activity scores during work or leisure time. Finally, it seems unlikely that any biochemical or body composition technique could have been biased by ethnicity.

The CT technique used in this study has precision errors between 0.01 and 0.3 L depending on the tissue under consideration, and also a high validity (error, 0.8%).[16] The high validity of the CT technique was reconfirmed in this study by comparing CT-estimated BW with true BW in Indians (SESD, 0.8%) and Swedes (SESD, 0.9%).

For the reasons discussed, it seems unlikely that phenotypic dissimilarities between Indians and Swedes could be explained by differences in food habits, physical activity, or methods biased by ethnicity. Instead, differences between the two groups are most likely genetically determined. Our Indians were not a homogenous group coming only from one part of the Indian subcontinent. However, Asians in general are distinctly different from Europeans in several genetic respects.[31] Since the variances with respect to several phenotypic characteristics were small enough to permit detection of differences between the two groups, a certain degree of genetic homogeneity seems plausible both within the Indians and within the Swedes in our study.

Differences observed between studies of indigenous populations in different parts of the world may reflect genetic differences, but are also dependent on major differences in environment. Studies of this type may be of great socioeconomic, political, and nutritional importance. Our study is an example of ethnic comparisons in the same environment after long-term adaptation. Such studies may better reflect genetic differences between groups, since much of the environment is adjusted for. However, the representativeness of migrants can always be questioned, even in large studies. Sometimes the strongest or most intelligent and sometimes the most persecuted have migrated. Small studies such as ours cannot claim representativeness. Nevertheless, it seems plausible that genetic differences between Indians and Swedes are better reflected by examinations of well-adapted subjects carefully matched by sex, age, weight, and height than by studies investigating risk factors and body composition in randomly selected groups of indigenous populations. Whereas we found that Indians were fatter than whites, Berry and Deshmuhk[32] found the opposite when somatotyping 877 male college students in Nagpur in Central India according to the method of Sheldon and comparing them with 4,000 American college students originally studied by Sheldon.[33] It seems likely that the former study[32] reflects nutritional status more than ours, whereas our results uncover genetic predispositions by adjusting for environment, weight, and height.

By using underwater weighing, DeBoer et al[34] compared seven European males with eight males from the Indian subcontinent who had lived in Holland for at least 3 to 6 months. Body fat constituted 21.6% of BW in Europeans and 17.3% in Indians. However, the subjects were not matched and, on average, BMI was 23.3 in Europeans and 20.1 kg/m^2 in Indians. Corresponding BWs were 78.4 and

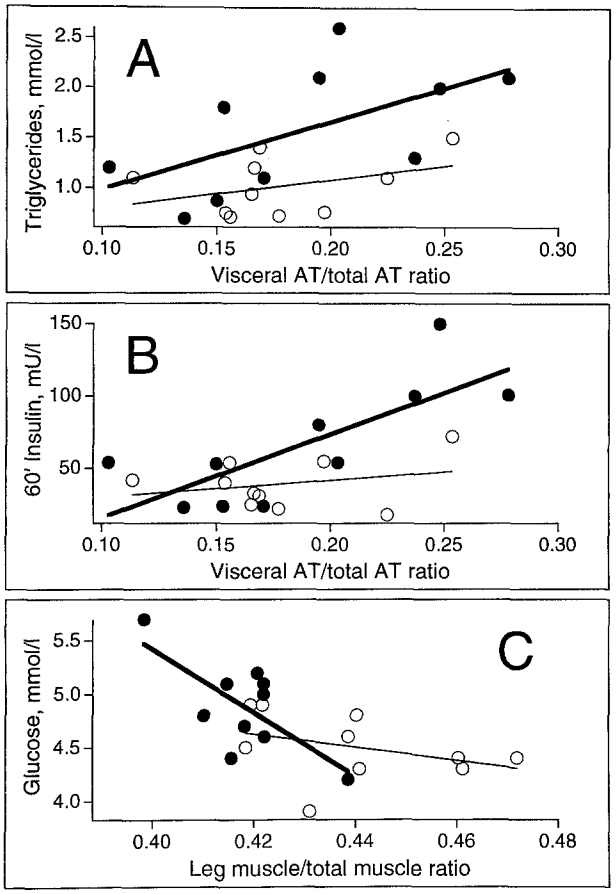

Fig 5. Relationships between cardiovascular risk factors and tissue distributions. (A) Fasting serum TG regressed by the visceral AT to total AT volume ratio in Indians (●) and Swedes (○). Equation and Spearman rank correlation (r_s) of pooled group (N = 20, not drawn): y = 0.202 + 6.00x, P = .023, r_s = .51, P = .021. Equation of Indians (N = 10, thick line): y = 0.314 + 6.73x, P = .073, r_s = .70, P = .024. Equation of Swedes (N = 10, thin line): y = 0.521 + 2.80x, P = .28, r_s = .38, P = .28. (B) Sixty-minute insulin values during OGTT regressed by visceral AT to total AT volume ratio in Indians and Swedes. Equation of pooled group (N = 20, not drawn): y = −29.0 + 448x, P = .004. r_s = .46, P = .042. Equation of Indians (N = 10, thick line): y = −41.7 + 576x, P = .01. r_s = .78, P = .008. Equation of Swedes (N = 10, thin line): y = 18.7 + 115x, P = .46. r_s = cells −.042, P = .90. (C) Fasting blood glucose regressed by the leg muscle to total skeletal muscle volume ratio in Indians and Swedes. Equation of pooled group (N = 20, not drawn): y = 10.5 − 13.6x, P = .004. r_s = −.59, P = .006. Equation of Indians (n = 10, thick line): y = 17.3 − 29.7x, P = .005. r_s = .474, P = .16. Equation of Swedes (N = 10, thin line): y = 7.24 − 6.22x, P = .29 (if the Swede with the lowest blood glucose [3.9 mmol/L, −2 SD in relation to regression line] was excluded, significance was achieved: P = .045). r_s = .50, n = 10, P = .14.

58.9 kg. Therefore, this study does not permit the conclusion that Indians are leaner than Europeans. Several studies have used the four-skinfold technique of Durnin and Womersley[35] to estimate percent body fat in Indians and Europeans. Using this technique, Geissler and Aldouri[36] found 17.7% fat in Asian males (n = 7) and 18.5% fat in European males (n = 8) who had similar averages with respect to age, height, and weight. In contrast, Henry et al[37] found, on average, a higher sum of four skinfolds in 11 Asian men (n = 4) and women (n = 7) than in 11 European men (n = 4) and women (n = 7) (47.9 and 45.7 mm, respectively).[37] Ulijaszek and Strickland[38] examined 17 British soldiers and 17 Gurkha soldiers living in England. On average, all four skinfolds were higher in Gurkhas, and values were significantly higher for the subscapular skinfold (8.9 and 11.2 mm, respectively). The two groups had similar BW (66.8 and 67.1 kg, respectively). However, BMI was slightly but significantly higher in the Gurkhas (English, 22.3; Gurkhas, 24.1 kg/m^2), which makes it difficult to draw firm conclusions.

Finally, McKeigue et al[30] reported on age-adjusted anthropometric means in 1,360 Asian males and 1,506 European males living in London. The age-adjusted weight was lower in Asians (73.8 and 78.7 kg, respectively), but they nevertheless had significantly higher waist circumferences (92.6 and 91.1 cm, respectively), as well as sagittal diameters (21.9 and 21.3 cm, respectively), indicating a larger abdominal fatness in the Asian males.

In summary, some studies have found an increased relative AT in Indians,[7,37,38] but others have not.[34,36] Lack of adequate body composition techniques[7,36-38] or poor matching[34] make it difficult to evaluate these studies. With respect to volumes or masses of other tissues and organs, no previous studies are available for comparison.

Much investigation remains until ethnic differences in body composition are explained in terms of molecular biology. In recent studies, gene polymorphisms with respect to apolipoprotein B[39] and factor VII coagulant activity[40] have been suggested to be related not only to cardiovascular risk factors but also to degree of obesity in men of South Asian descent.

The cluster of metabolic disturbances found in our Indian males is compatible with the metabolic syndrome. This syndrome is caused by disturbances in several neuroendocrine axes, leading to peripheral insulin resistance and visceral fat accumulation.[17] The visceral AT per se may worsen the metabolic situation by causing reduced insulin clearance via increased free fatty acid exposure of the liver.[17] Although no preponderance of visceral AT was found in our Indians, insulin and TG seemed to increase with an increasing visceral AT to total AT volume ratio both in Indians and in Swedes. At any given ratio except the lowest, Indians seemed to have higher risk factors and most given risk factor levels seemed to be reached at a lower visceral AT in Indians, not only in relative but also in absolute terms (compare slopes of Fig 5A and B and total amount of AT in Table 3). This finding is compatible with an increased metabolic sensitivity to visceral fat accumulation in Indians, but our data do not permit definite conclusions on this point, since visceral fat accumulation is to some extent just a phenotypic companion to metabolic disturbances caused by neuroendocrine perturbations.[17] However, our data do show that an increased visceral AT is not an absolute prerequisite for elevated risk factors in Indians. Our findings are also in line with a worsened metabolic situation in other groups of Indians[9,30] with a preponderance of visceral AT.

As stated in the introduction, the waist to hip ratio is an excellent risk indicator[11,12] but a comparatively poor estima-

tor of the visceral AT volume.[13] This indicates that the ratio is carrying information that has not been possible to interpret simply as being due to differences in visceral AT. Since skeletal muscle is by far the largest tissue in non-obese subjects (Table 3), its distribution may have profound influences on the waist to hip ratio. As compared with Swedes, Indians have a smaller fraction of muscle in the lower part of the body, which may contribute to an increased waist to hip ratio in Indians, although not significantly so in our study. This is of particular interest, since a low fraction of muscle in the legs was associated with increased glucose and TG values. Therefore, we hypothesize that the muscle distribution may contribute to the power of the waist to hip ratio as a risk index. In larger studies, the waist to hip ratio has been associated with most traditional cardiovascular risk factors.[11,17] Table 7 suggests that some of these associations may be dependent on AT distribution, whereas others may be related to muscle distribution. Certainly, the suggested associations require confirmation in larger, comparably detailed body composition studies. In this connection, we would like to stress that determination of tissue areas in a restricted number of scans will be of limited value since tissue areas cannot be expressed as fractions of total tissue volumes.

It may well be that in addition to abdominal obesity, an upper-body muscle distribution is also a phenotypic companion to the metabolic syndrome. Although the hormonal regulation of AT distribution is known in large detail,[17] nothing seems to be known about hormonal factors determining muscle distribution. Physical activity was similar in our Swedish and Indian males. Although visceral fat accumulation per se may cause additional metabolic disturbances,[17] it remains to be examined if an upper-body muscle distribution per se is causally related to metabolic or microcirculatory disturbances. Since no data on muscle distribution and metabolic risk factors are available in the literature, our investigation needs to be confirmed.

In conclusion, we have found increased AT and decreased lean tissue in Indian males as compared with Swedish males matched by age, height, and BW. Neither these differences nor differences in tissue distributions were detectable with anthropometric techniques. Insulin, glucose, and TG were higher in Indians than in Swedes. These differences occurred at similar visceral AT to total AT ratios but at a lower leg muscle to total muscle ratio in Indians. We hypothesize that in addition to abdominal obesity, an upper-body muscle distribution is also a phenotypic companion to the metabolic syndrome. Future phenotypic characterizations with the CT technique used in this study will hopefully facilitate gene polymorphism studies with respect to body composition.

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