Regional Fat Deposition in the Legs Is Useful as a Presumptive Marker of Antiatherogenesity in Japanese (44474)

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Abstract. **To examine the pathological role of regional fat deposition in development of metabolic and cardiovascular disorders, regional fat distribution was evaluated using metabolites and hormones as measures of obesity-related disorders. The subjects enrolled were 100 sex-matched inpatients, who were admitted, regardless of their body mass index values, for further examination of unusual results from periodic medical screening tests, and for examination of obesity-induced complications and treatment of obesity. Body fat distribution was analyzed using dual energy X-ray absorptiometry (DEXA). Analysis of parameters regarding fat distribution showed that gender was one of the determinants affecting correlation between fat distribution and metabolites of fasting plasma glucose (FPG), hemoglobin A_{1c} (HbA_{1c}), total cholesterol (TC), or triglyceride (TG). However, regardless of gender, both leg trunk fat (L/Tr) and arm trunk fat (A/Tr) ratios negatively correlated with a total body fat (% total fat) ratio, whereas the intercept value of female regression line in L/Tr was greater than that in males, but not in A/Tr. Percentage total fat, L/Tr, and A/Tr in males correlated significantly with FPG, TC, TG, low-density lipoprotein (LDL), very low density lipoprotein (VLDL), atherogenic index (A.I.), and apoB/A1 only low density lipoprotein (LDL) was significantly correlated solely to L/Tr and A/Tr. These results indicate that regional fat distribution in males may not be a major determinant for development of metabolic disorders in obese patients. Unlike male regional fat distribution, female L/Tr correlated significantly not only with TC, TG, and LDL, but also with FPG and HbA_{1c}, although both of the latter 2 glucose-related parameters in males showed no correlation with any parameters of fat deposition. The remaining female parameters of fasting plasma insulin, VLDL, A.I., and ApoB/A1 correlated with each of the three parameters of fat deposition, as similarly shown in males. The powerful and negative correlation was thus evident, particularly in females, between leg fat deposition and parameters of glucose and lipid metabolites. The resulting information provides a novel insight that regional fat deposition at the legs is useful as a marker for metabolic and cardiovascular disorders associated with obesity.** [P.S.E.B.M. 2000, Vol 223]

Recent studies have shown that certain types of fat deposition, abdominal fat in particular, are highly correlated with metabolic complications and with abnormalities of humoral factors such as glucose, insulin, and lipids (1, 2). Android obesity shown in men storing their excess fat in the abdominal depots was found to be more frequently associated with diabetes mellitus, atherosclerosis, and gout than gynoid obesity manifested in women storing their excess fat in the subcutaneous gluteal and femoral regions (3). The waist to hip girth ratio is another parameter to evaluate regional fat distribution indirectly, although it is used frequently in Asian countries. The ratio of visceral to subcutaneous fat area (V/S ratio) measured by x-ray computed tomography (CT) at the navel level has been demonstrated to be a more useful marker of metabolic complications including hyperinsulinemia, impaired glucose tolerance, dyslipidemia, hypertension, and ischemic heart disease (4).

Since leptin, a protein product of the recently cloned *ob*

Supported partly by Grant-in-Aid 07457225 from Japanese Ministry of Education, Science and Culture, and by Research Grants for Intractable Diseases from the Japanese Ministry of Health and Welfare, 1991-1992.

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Received December 29, 1998. [P.S.E.B.M. 2000, Vol 223]
Accepted August 13, 1999.

0037-9727/00/2232-0156$14.00/0

(obese) gene (5), is secreted exclusively by adipocytes, adipose tissues are not simply passive storehouses of excess body fat, but are actively involved in weight regulation. There is a growing body of evidence that fat cells comprise a kind of endocrine organ. Indeed, not only leptin but various other kinds of bioactive substances have been reported to be released systemically from the adipose tissues (6). However, most of these bioactive substances including leptin are known to be secreted from whole adipose tissues, and not exclusively from visceral adipocytes except for a few substances (7). These recent findings suggest that visceral fat may not be the sole regional fat deposit related to metabolic complications in obese patients.

Dual energy x-ray absorptiometry (DEXA) was developed originally to examine body mineral composition, particularly bone mineral (8, 9). This device now opens a new field in which not only total body fat deposition but also regional fat distribution can be quantitated accurately, because DEXA enables one to differentiate bone and fat deposits clearly from other tissues and organs (10). Taken as a whole, evidence led us to investigate the normal and pathological topography of fat distribution to reveal possible correlation with metabolic disorders. The topographical analysis of fat distribution was carried out by DEXA by dividing the body into six subdivisions in Japanese obese patients.

Materials and Methods

Subjects. One hundred subjects (50 males and 50 females) were selected among the inpatients who i) were admitted for further examination of obesity-induced complications and therapeutic treatment of obesity because their body mass index (BMI) values were 26 kg/m^{-2} and/or visceral obesity was highly suspected by screening of abdominal echogram, and ii) were selected by their home doctors and company physicians for further precise examination regardless of their BMI values because their periodic medical check showed unusual findings according to tests of urine and peripheral blood, chest x-ray examination, electrocardiography (ECG), and abdominal echography. They were admitted in the Department of Internal Medicine I, Oita Medical University Hospital from April 1992 through March 1993.

The enrolled inpatients fulfilled the age limitation of 35–70 years old, and had neither history of major diseases nor medication known to influence the parameters used in the current study. They were further examined to define the metabolic complications of and therapeutic approaches for simple and secondary obesity, dyslipidemia, impaired glucose tolerance (IGT), type 2 diabetes mellitus, and/or ischemic heart disease. The subjects with ischemic heart disease were excluded from the present study because a recent cardiac event held a possibility of affecting the current parameters. For the same reason, the inpatients in the Intensive Care Unit were discarded from the study. Cardiac events were further examined by ambulatory ECG monitoring, echocardiography, and echocardiography with exercise load. The subjects who had any complaints of cardiac events and/or were not defined normally by these tests were certified by coronary angiography.

The subjects with abnormal serum concentrations of thyroid hormones, ACTH, cortisol, androgen, progesterone, and estradiol were further examined by the corresponding circadian variations and/or the appropriate hormone challenge tests. Throughout those testing procedures, the subjects with abnormal levels of those hormones were discarded from entry because the abnormalities caused by endocrine diseases or stress situations upon admission in the hospital affect lipid metabolism and adipose mobilization. Assessed by laboratory tests of urine and peripheral blood together with abdominal echography, the subjects with liver and kidney diseases were not enrolled. The subjects treated with insulin, sulfonylurea, α-glucosidase inhibitor, antidyslipidemic drugs, pharmacological steroids, and other drugs affecting serum glucose, lipids, or their metabolisms were not assigned entry, either, according to the criteria mentioned above.

Oral Glucose Tolerance Test and Blood Sampling Procedure. A 75-g oral glucose tolerance test (OGTT) was carried out in the morning after an overnight fast. Blood samples were collected at 0, 30, 60, and 120 min after glucose loading through a catheter implanted into the antecubital vein for determination of plasma glucose concentration, hemoglobin A_{1c} (HbA_{1c}), insulin concentration, and insulinogenic index (I.I.). The plasma samples were stocked at −20°C until assay. Plasma glucose was assayed enzymatically (11). HbA_{1c} was measured by ion-exchange chromatography with an upper normal limit of 6.3%. Plasma insulin was measured by a radioimmunoassay method with polyethylene glycol separation (12). The I.I. was calculated from [insulin at 30 min − insulin at 0 min]/[glucose at 30 min − glucose at 0 min].

The blood-sampling procedure for hormone assay was the same as that for glucose measurement, as applicable. Blood samples for circadian variation of hormone were taken at 06:00, 10:00, 14:00, 18:00, 22:00, and 02:00 hr.

Measurement of Plasma Lipids, Lipoproteins, and Apolipoproteins. The high-density lipoprotein–cholesterol (HDL-C) fraction was separated from chylomicrons (13), low-density lipoprotein (LDL), and very low density lipoprotein (VLDL) by an improved phosphotungstate-$MgCl_2$ precipitation technique (14). Total plasma cholesterol (TC) and plasma triglyceride (TG) concentrations were determined enzymatically using kits (Determiner TC555 and Determiner TG555; Kyowa Medex, Tokyo, Japan). Plasma apo A-I and B were determined by turbidimetric immunoassay (TIA) using the reagent kits (i.e., Apo Auto 'Daiichi' and according to the manufacturer's instructions with TBA-30R, Toshiba, Tokyo, Japan) (15). According to these parameters, the atherogenic index [(TC − HDL-C)/HDL-C] (16) and apo B/AI (17) were calculated to address atherogenesity. The blood-sampling procedure for

lipid measurements was the same as that for glucose measurement, as applicable.

Measurement of Regional Fat Distribution. Regional body composition was analyzed by QDR-1000/W x-ray bone densitometer (Hologic Co. Ltd., Bedford, MA). Body composition was analyzed by measures of bone mineral, fat tissue mass, and lean tissue mass (18). The fat tissue mass was measured as a sum of the fatty elements among the whole soft tissues. Similarly, the lean body mass was measured as a sum of the whole fat-free soft tissue element (18).

A scanned image of the whole body was divided into six subdivisions: head, trunk, left and right limbs. The dividing borders between those subregions were differentiated by a line underneath the chin, a line between the humerus head and the glenoid fossa, and a line at the femoral neck. To express regional fat deposition, the following parameters were introduced: i) total body fat ratio (% total fat) expressed as 100 × (total fat tissue weight/body weight); ii) leg fat ratio (L/Tr) expressed as 100 × (leg fat weight/trunk fat weight); and iii) arm fat ratio (A/Tr) expressed as 100 × (arm fat weight/trunk fat weight). Immediately after admission, DEXA and the 75-g oral glucose tolerance test were carried out according to measures of body height (m) and body weight (kg).

Statistical Analysis. All the data were expressed as mean ± SEM. Statistical evaluation of the parameters was carried out using Student's *t* test. Statistical analysis of the correlation between fat distribution and bodily or humoral parameters was based on the simple linear regression analysis method.

The present study was authorized by the ethics committee of Oita Medical University. All patients gave written informed consent to participate in the study according to Helsinki Declaration II.

Results

Protocol of Population. Table IA shows age and body characteristics of 50 female and 50 male subjects. Male subjects were 10 years older on average than the females ($P < 0.01$). However, percentage total fat in the females was larger than that in the males ($P < 0.01$). The other parameters of body weight, BMI, L/Tr, and A/Tr did not differ between genders. Table IB shows profiles of humoral factors in male and female subjects. Reflecting the greater percentage of total fat, both the fasting plasma glucose ($P < 0.05$) and insulin concentration ($P < 0.01$) in the female inpatients were higher than those in the males.

Figure 1 shows the relationship between percentage total fat and BMI in male and female subjects. A positive correlation between the parameters was found in both male ($r = 0.685$, $P < 0.00001$) and female subjects ($r = 0.914$, $P < 0.00001$). Female subjects showed a tight correlation ($P < 0.01$) and steeper inclination of the regression line than male subjects ($P < 0.01$).

Fat Topography and Metabolites. To determine whether L/Tr or A/Tr may have any implications as a parameter of physical fat constitution, a relationship between L/Tr or A/Tr and percentage total fat was investigated. As shown in Figure 2, both L/Tr and A/Tr correlated with percentage total fat in both genders (L/Tr: $y = 1.15 - 0.02x$, $r = -0.596$ in males; $y = 1.46 - 0.02x$, $r = -0.464$ in females; A/Tr: $y = 0.50 - 0.01x$, $r = -0.565$ in males; $y = 0.56 - 0.01x$, $r = -0.718$ in females; $P < 0.01$ for each). The intercept value of the L/Tr regression line in females was greater than that in males ($P < 0.01$), but there was no significant difference between the genders in the case of A/Tr (data for A/Tr not shown in Fig. 2).

Table II shows the correlation coefficients between physical or fat distribution parameters and glucose- or insulin-related indices in both genders. In male subjects, the percentage total fat correlated positively ($P < 0.01$), and both L/Tr and A/Tr negatively with fasting insulin only ($P < 0.05$), but not significantly with the remaining parameters of fasting glucose, HbA_{1c} and I.I. Female percentage total fat correlated positively, but L/Tr and A/Tr negatively with fasting insulin only ($P < 0.01$ for each), similarly to those in the males. Both female body weight ($P < 0.01$) and BMI (P

Table IA. Characteristics of Physical and Fat Distribution Parameters in Male and Female Subjects

	n	Age	BW	BMI	%total fat	L/Tr	A/Tr
Male	50	57.9 ± 1.7	66.6 ± 2.1	24.2 ± 0.6	20.4 ± 0.7	0.75 ± 0.03	0.34 ± 0.01
Female	50	47.4 ± 2.4*	63.5 ± 2.5	26.5 ± 1.1	30.2 ± 1.2*	0.83 ± 0.04	0.35 ± 0.01

Note. Values, mean ± SEM. *n*, number of subjects examined. Age, (years old). In this and succeeding tables, BW: body weight (kg); BMI: body mass index (kg/m^2); %total fat: a total body fat ratio (%); L/Tr: leg trunk fat ratio; A/Tr: arm trunk fat ratio. Male and Female, male and female patients. * = $p < 0.01$ vs the corresponding gender.

Table IB. Characteristics of Humoral Factors in Male and Female Subjects

	n	FSG	IRI-0	TC	TG	HDL-C
Male	50	97 ± 3	5.2 ± 0.6	184 ± 5	139 ± 10	40 ± 2
Female	50	117 ± 7*	8.5 ± 0.8**	189 ± 5	118 ± 7	42 ± 2

Note. Values, mean ± SEM. *n*, number of subjects examined. In this and succeeding tables, FSG: fasting serum glucose (mg/dl); IRI-0: fasting serum insulin (μ U/ml); TC: serum total cholesterol (mg/dl); TG: serum triglyceride (mg/dl); HDL-C: high density lipoprotein cholesterol (mg/dl); Male and Female, male and female subjects. * $p < 0.05$, ** $p < 0.01$ vs the corresponding gender.

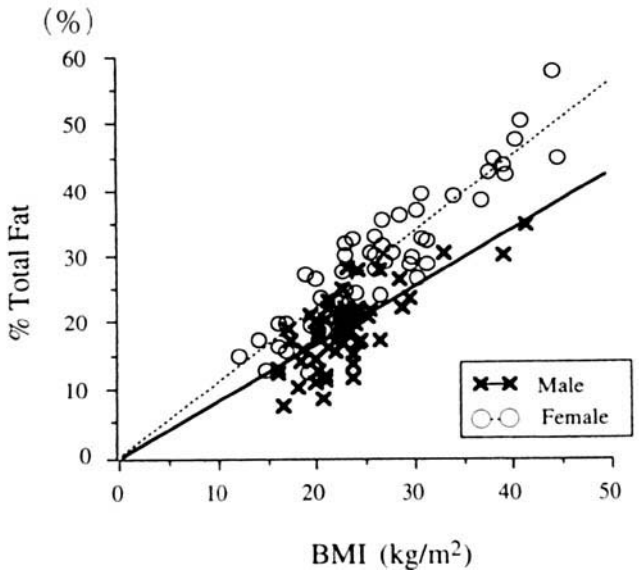

Figure 1. A correlation between total fat ratio (% Total Fat) and body mass index (BMI) in the male and female subjects. A regression line in male subjects: $y = -0.59 + 0.86x$, $r = 0.715$, $P < 0.01$. A regression line in females: $y = -0.23 + 1.13x$, $r = 0.917$, $P < 0.01$. Comparison between these two lines revealed that females showed a tight correlation ($P < 0.01$) and steeper inclination of the regression line than males ($P < 0.01$).

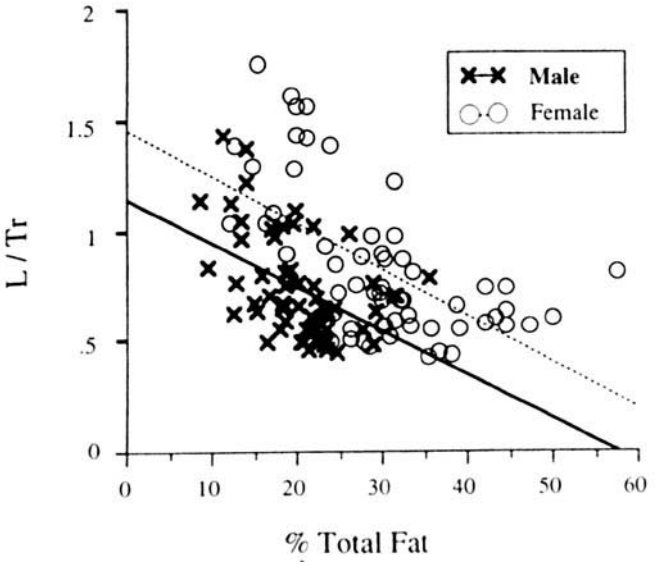

Figure 2. A relation between total fat ratio (% total fat) and a leg fat ratio (L/Tr) in male and female inpatients. The L/Tr ratio correlated negatively with the percentage total fat in both male ($y = 1.15–0.02x$, $r = -0.596$, $P < 0.01$) and female subjects ($y = 1.46–0.02x$, $r = -0.464$, $P < 0.01$). The intercept value of the regression line was greater in females than in males ($P < 0.01$).

< 0.05) correlated positively with HbA_{1c} as well, although percentage total fat showed no significant correlation. In contrast to the males, female L/Tr correlated negatively with fasting glucose ($P < 0.05$), HbA_{1c}, and fasting insulin ($P < 0.01$ for each), whereas female A/Tr did not correlate negatively with fasting glucose. Neither physical nor fat distribution parameter correlated with I.I-calculated during 75 g OGTT.

Table II. Correlation Coefficient in Male and Female Subjects Between Physical or Fat Distribution Parameters and Serum Humoral Factors

Male	FSG	IRI-0	HbA_{1c}	I.I.
BW	0.033	0.434**	0.072	0.067
BMI	0.083	0.409**	0.166	0.092
%total fat	-0.071	0.426**	0.141	0.161
L/Tr	-0.234	-0.310*	-0.015	0.160
A/Tr	-0.043	-0.316*	0.060	0.090
Female				
BW	0.152	0.549**	0.375**	-0.115
BMI	0.195	0.604**	0.359*	-0.089
%total fat	0.052	0.461**	0.260	-0.095
L/Tr	-0.313*	-0.483**	-0.420**	0.270
A/Tr	-0.157	-0.453**	-0.321*	0.169

Note. Values, correlation coefficients. I.I., insulinogenic index; Male and Female, male and female subjects; FSG, fasting serum glucose (mg/dl); IRI = 0, fasting serum insulin (μU/ml); BW, body weight (kg); BMI, body mass index (kg/m²); %total fat, a total body fat ratio (%); L/Tr, leg trunk fat ratio; A/Tr, arm trunk fat ratio. *$p < 0.05$, **$p < 0.01$.

As shown in Table III, a total fat ratio in male subjects showed positive correlation with almost all the lipids and the related parameters [i.e., TC ($P < 0.05$), TG, VLDL, A.I., and apo B/AI ($P < 0.01$ for each) except HDL-C and LDL]. Both male L/Tr and A/Tr correlated negatively with almost all those parameters (L/Tr: $P < 0.05$ for LDL, $P < 0.01$ for the remainder; A/Tr: $P < 0.05$ for LDL; and A.I.: $P < 0.01$ for the remainder) except HDL-C. In contrast to these male parameters, female percentage total fat correlated positively and A/Tr negatively only with VLDL, A.I., and Apo B/AI, respectively (% total fat: $P < 0.01$ for A.I., $P < 0.05$ for the others; A/Tr: $P < 0.05$ for VLDL, $P < 0.01$ for the others). However, L/Tr in the female subjects correlated negatively with all the parameters ($P < 0.05$ for TG and LDL, $P < 0.01$ for the remainder) except HDL-C.

To evaluate clinical implications of these fat distribution parameters, correlation was examined in both genders between fat distribution parameters and indices implicating atherogenecity as shown in Figure 3. The correlation of percentage total fat with the atherogenic indices of A.I. and Apo B/A1 was found to be positive in both genders [male: $y = 1.69 + 0.11x$, $r = 0.413$ in A.I., $y = 0.46 + 0.02x$, $r = 0.447$ in Apo B/AI ($P < 0.01$ for each); female: $y = 2.49 + 0.05x$, $r = 0.408$, ($P < 0.01$ in A.I.); $y = 0.58 + 0.01x$, $r = 0.343$, ($P < 0.05$ in Apo B/AI)]. Contrary to those correlations, L/Tr consistently correlated negatively with both the atherogenic indices in both genders [male: $y = 6.15 - 3.05x$, $r = -0.449$ in A.I.; $y = 1.15 - 0.40x$, $r = -0.511$ in Apo B/AI; ($P < 0.01$ for each); female: $y = 6.41 - 2.90x$, $r = -0.563$ in A.I; $y = 1.26 - 0.54x$, $r = -0.456$ in Apo B/AI; ($P < 0.01$ for each)]. Similar to those L/Tr correlations, A/Tr in both genders correlated negatively with the atherogenic indices [male: $y = 6.57 - 7.07x$, $r = -0.381$, ($P < 0.05$ in A.I.); $y = 1.24 - 1.07x$, $r = -0.435$, ($P < 0.01$ in Apo B/AI); female: $y = 4.24 - 1.38x$, $r = -0.490$, in

Table III. Correlation Coefficients in Male and Female Subjects Between Physical or Fat Distribution Parameters and Serum Lipids

Male	TC	TG	HDL-C	VLDL	LDL	A.I.	Apo B/A1
BW	0.314*	0.551**	−0.263	0.482**	0.033	0.452**	0.530**
BMI	0.420**	0.527**	−0.162	0.453**	0.035	0.400**	0.505**
%total fat	0.299*	0.606**	−0.215	0.375**	−0.011	0.413**	0.447**
L/Tr	−0.437**	−0.570**	0.225	−0.510**	−0.281*	−0.449**	−0.511**
A/Tr	−0.404**	−0.598**	0.159	−0.475**	−0.307*	−0.381*	−0.435**
Female							
BW	0.319*	0.221**	−0.233	0.458**	−0.044	0.426**	0.445**
BMI	0.241	0.137	−0.244	0.372*	−0.034	0.412**	0.390**
%total fat	0.128	0.088	−0.279	0.342*	−0.100	0.408**	0.343*
L/Tr	−0.401**	−0.358*	0.263	−0.613**	−0.369*	−0.563**	−0.456**
A/Tr	−0.123	−0.193	0.302	−0.355*	−0.191	−0.490**	−0.386**

Note. Values, correlation coefficients. VLDL, very low density lipoprotein (mg/dl). LDL, low density lipoprotein (mg/dl). A.I., atherogenic index. Apo B/A_1, apoprotein B/apoprotein A_1. Male and Female, male and female subjects. $^*p < 0.05$, $^{**}p < 0.01$ vs the corresponding gender.

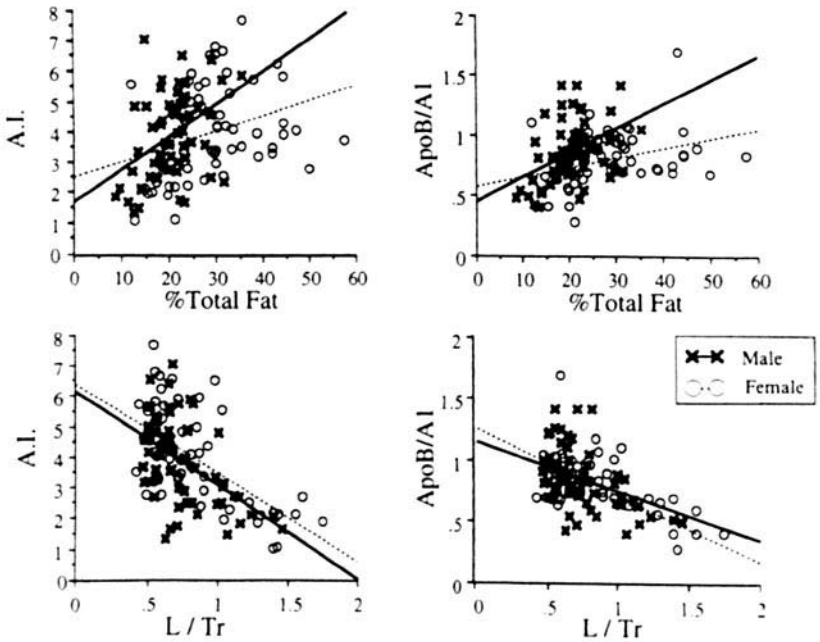

Figure 3. Correlation in male and female subjects between total fat ratio (% total fat) or leg fat ratio (L/Tr) and atherogenic index (A.I.) or apolipoprotein B/apolipoprotein A1 (Apo B/A1). Percentage total fat correlated positively with A.I. (male: $y = 1.69 + 0.11x$, $r = 0.413$; female: $y = 2.49 + 0.05x$, $r = 0.408$; $P < 0.01$ for each) and with Apo B/A1 in both genders (male: $y = 0.46 + 0.02x$, $r = 0.447$, $P < 0.01$; female: $y = 0.58 + 0.01x$, $r = 0.343$; $P < 0.05$). In contrast, the L/Tr correlated negatively with A.I. (male: $y = 6.15 - 3.05x$, $r = -0.449$; female: $y = 6.41 - 2.90x$, $r = -0.563$; $P < 0.01$ for each) and with Apo B/A1 in both genders (male: $y = 1.15 - 0.40x$, $r = -0.511$, female: $y = 1.26 - 0.54x$, $r = -0.456$; $P < 0.01$ for each).

A.I.; $y = 0.89 - 0.31x$, $r = -0.386$ in Apo B/AI; ($P < 0.01$ for each)] as shown in Table III. Indeed, a correlation between the percentage total fat and the L/Tr was shown to be negative in both genders as shown in Figure 2.

Discussion

Obesity plays a key role in the pathogenesis of several metabolic and cardiovascular diseases. Recent years have seen an explosive increase in our understanding of the metabolic impact of differences in fat distribution between visceral and subcutaneous areas. Specifically, increased abdominal adiposity is accepted as a major risk factor for diabetes mellitus, dyslipidemia, hypertension, arteriosclerotic vascular diseases, and mortality (2). The mechanisms whereby obesity causes such metabolic defects remain incompletely understood.

Until the early 1990s, the terms and measures representing fat distribution such as android and gynoid obesity (3), a waist to hip girth ratio (19), and skin fold thickness were frequently used because those indicators were easy and inexpensive ways to estimate obesity-linked impairment of metabolism. The main reasons why clinical use of such measures has been replaced by detection of visceral fat accumulation is that there is a clearer and more accurate correlation of visceral adiposity with metabolic and cardiovascular diseases (4). In the present study, we used DEXA, rather than CT, to investigate fat distribution because this method enabled us to measure total body fat and its regional distribution simultaneously and accurately by differentiating fat mass from fat-free mass (9, 10).

The total fat ratio in the present study correlated positively with BMI in both genders. In particular, the female subjects showed a higher correlation coefficient and steeper

inclination value of the regression line than the males. These results indicated that female body fat accumulation is greater than male deposition even when BMIs are elevated to the same extent in both genders. In other words, it is likely that a given increase in BMI reflects more severely fat deposition in females than in males. These explanations may answer a query as to why female subjects showed greater total body fat than male subjects in the present study, regardless of differences in BMI between genders, and in spite of the females' younger age. The L/Tr and A/Tr in both genders correlated negatively with the percentage total fat, whereas the intercept value of female L/Tr regression line was larger than that in the males, but not in A/Tr, indicating greater fat deposition in the lower segment of the female body when excess fat mass is deposited at the same extent in males and females. In addition, this negative correlation between regional fat and the percentage total fat implies as well that the increase in percentage total fat induces predominant fat deposition in the trunk body segment regardless of gender.

Fasting plasma insulin concentration correlated significantly with all the parameters representing fat deposition and body weight regardless of gender although the correlation of L/Tr with insulin was more potent in female subjects than in males. According to the results, it is highly probable that increasing total body mass may contribute to the development of hyperinsulinemia. From the viewpoint that BMI was found to correlate positively and tightly with the percentage total fat in the present study, and in addition, that fat-free mass such as muscle and bone is not a main component of weight gain, it seems reasonable to say that the increase in total body mass depends mainly on fat mass, but not on fat-free mass.

One of the notable findings in the present study is that the difference in the correlation between fat topographical and metabolic or atherogenic parameters depended on gender. In the males, all the fat topographical parameters (i.e., % total fat positively, but L/Tr and A/Tr negatively) correlated in a similar fashion with fasting insulin and most of the lipid and its related parameters. However, in the females it was solely L/Tr that correlated negatively with TC, TG, and LDL, in contrast to correlation of percentage total fat or A/Tr with the corresponding parameters. Additionally, female L/Tr correlated broadly with fasting plasma levels of glucose, HbA_{1c}, and insulin although either remaining fat topographical parameter did not correlate with the former two glucose parameters. Together with these findings, male trunk fat deposition appears to play an essential role in development of hyperinsulinemia together with lipid metabolic disorders. In females, however, regional fat deposition particularly at the legs rather than trunk fat deposition seemed to be a major determinant for disorders related to lipid and glucose metabolism.

The present study demonstrated that the degree of total fat deposition and its regional distribution to the lower segment of the body were greater in females than in males. As noted previously the negative correlation between the percentage total fat and the L/Tr indicates that progression of obesity accelerates fat deposition in the upper body segment. It has been demonstrated that administration of testosterone reduces visceral fat deposition but not subcutaneous fat deposition, consequently changing the ratio of visceral and subcutaneous adipose tissues (20). Abdominal fat that contributes mainly to fat deposition at the upper body segment possesses high activities of both lipogenesis and lipolysis (21). These characteristics of lipid metabolism in visceral and subcutaneous adipose tissues may cause differences in the development of several metabolic disorders including dyslipidemia and hyperinsulinemia. Precisely how L/Tr contributes to developmental steps in obesity-related diseases is as yet unknown, but speculation has centered on the following possibility. The reversed relationship of L/Tr with LDL suggests clinically that regional fat deposition in the legs may play an important role in protection from metabolic and cardiovascular diseases. Indeed, L/Tr correlated negatively as well with A.I. and Apo B/A1. Taken together, it is reasonable to conclude that along with the development of obesity, an increase in trunk fat deposition in females induces a re-distribution from gluteal-leg (gynoid) to abdominal (android) obesity, whereas the additional trunk fat deposition in males simply reinforces fat deposition in the abdomen (android) without changes in the distribution.

We thank Dr. D. S. Knight, Department of Cellular Biology of Anatomy, Louisiana State University for help in preparation of the manuscript.

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