Original Article

# Associations of postprandial lipemia with trunk/leg fat ratio in young normal weight women independently of fat mass and insulin resistance

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**Background and Objectives:** To determine whether postprandial lipemia is associated with fat distribution even in young, normal weight women independently of fat mass, adipokines, insulin resistance and systemic inflammation. **Methods and Study Design:** Female college students (ages 21-24, n=35) underwent dual-energy X-ray absorptiometry and a standardized breakfast providing 17 g triglycerides (TG). Serum lipids, lipoproteins, apolipoproteins, adipokines and markers of insulin resistance and inflammation were measured in fasting blood samples. **Results:** In crude analyses, postprandial lipemia, as assessed by 0-2 h area under the curve of serum TG (TG-AUC), was positively associated with fasting TG, trunk/leg fat ratio, apolipoprotein B, leptin/adiponectin ratio and log high-sensitivity CRP. Multiple linear regression analysis with these 5 variables as independent variables revealed that fasting TG ($p$<0.001) and trunk/ leg fat ratio ($p$=0.001), were independent positive predictors of TG-AUC ($R^2$=0.923). Women with high compared to low TG-AUC were characterized by higher trunk/leg fat ratio, elevated apolipoprotein B and leptin/adiponectin ratio. **Conclusion:** Trunk/leg fat ratio, a marker of central adiposity, is a significant predictor of postprandial lipemia even in young women who are normal weight and insulin-sensitive, suggesting a modifiable pathway to postprandial hypertriglyceridemia, a cardiometabolic risk factor. These findings should be confirmed in studies employing more participants.

**Key Words: postprandial triglyceridemia, trunk fat, leg fat, trunk/leg fat ratio, insulin resistance**

## INTRODUCTION

High levels of serum triglycerides (TG) play a role in cardiovascular disease.[1] New insights strongly suggest that TG-rich lipoproteins represent causal risk factors for low-grade inflammation, atherosclerotic cardiovascular disease, and all-cause mortality.[2] TG levels are commonly measured in the fasting state; TG levels, however, increase significantly postprandially, and an important role in the pathogenesis of atherosclerosis-related diseases has been postulated for postprandial lipids. Post-alimentary lipemia was first described as an atherogenic phenomenon by Zilversmit.[3] Later, it received more attention after the discovery that TG-rich lipoproteins are atherogenic.[4] Postprandial abnormalities in TG metabolism (increased plasma TG concentrations and prolonged postprandial response after a fatty meal) are a hallmark of patients with established cardiovascular disease.[5] As recently reviewed,[6,7] the postprandial lipemic response is influenced by numerous factors including background dietary pattern and meal composition, lifestyle conditions (smoking, alcohol consumption and physical activity), physiological factors (age, gender and menopause), pathological conditions (obesity, metabolic syndrome and type 2 diabetes) and genetic factors.

It has been reported that obesity in general,[8] abdominal obesity in particular,[9-12] is associated with postprandial lipemia. Many investigators have shown repeatedly that fasting TG is the major determinant of postprandial lipemia.[9,10,13] In the present study, therefore, we tested the hypothesis that the ratio of trunk fat to leg fat, measured using whole-body dual-energy X-ray absorptiometry (DXA), is a predictor of postprandial lipemia independent of fat mass, fasting TG, insulin resistance and inflammation even in young female college students, a population in which confounding factors are so scarce.[14]

## PARTICIPANTS AND METHODS

Female collegiate athletes and untrained female students (n=17 and 18, respectively, aged 21-24 years) were re-

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cruited as volunteers. Athletes and non-athletes were students from the Department of Health and Sports Sciences and the Department of Food Sciences and Nutrition of the Mukogawa Women's University, respectively. Athletes had been training regularly for 2 years or longer prior to the study, 5 h a day, and 6 days a week and participated regularly in competitive events in their respective sport specialties. Non-athletes were not engaged in any regular sport activity. Subjects with clinical diagnosed acute or chronic inflammatory diseases, endocrine, cardiovascular, hepatic, renal diseases, hormonal contraception, unusual dietary habits were excluded. Nobody reported to receive any medications or have regular supplements. The study was approved by the Ethics Committees of the University (No. 07-28) to be in accordance with the Helsinki declaration. All subjects gave written consent after the experimental procedure had been explained.

After 12 h overnight fasting, participants underwent a standardized meal test as described later and measurements of anthropometric indices and body composition in the morning, as previously described.[14] Athletes had regular training on the day before measurements. In fasted blood samples, the following was measured as previously reported;[14] plasma glucose, serum insulin, triglycerides, cholesterol, HDL cholesterol, apolipoprotein A-1 (apoA1), apolipoprotein B-100 (apoB), non-esterified fatty acid (NEFA), liver enzymes, adiponectin, leptin, high- sensitivity C-reactive protein (hsCRP), tumor necrosis factor-α (TNF-α), plasminogen activator inhibitor-1 (PAI-1). Insulin resistance was determined by homeostasis model assessment (HOMA-IR)[15] and leptin/adiponectin ratio.[16] Serum concentrations of hsCRP and TNF-α below the limit of detection were assigned a value of 20 μg/mL and 0.50 pg/ml (the lowest limit of detection), respectively.

Lean tissue mass, fat mass, and bone mineral mass for arms, legs, trunk and the total body were measured using whole-body DXA (Hologic QDR-2000, software version 7.20D, Waltham, MA) as previously reported.[14] Although DXA cannot distinguish between intraabdominal and subcutaneous abdominal fat, research showed strong correlations between trunk fat mass measured with DXA and intraabdominal fat measured with CT.[12]

The standardized test meal was developed by the Japanese Diabetes Society to assess both postprandial hyperglycemia and hyperlipemia. This meal was composed as a breakfast meal (total energy 450 kcal) and provided 33.3% of calories from fat (16.7 g), 51.4% from carbohydrates (57.8 g), and 15.3% from protein (17.2 g). The test meal contained more fat than a typical Japanese breakfast (20-25%) but comparable energy content (median: 423 kcal). Participants were asked to consume the entire meal within 15 min. Venous blood was drawn at baseline (0 min), 30, 60, and 120 min after the start of the meal for the measurement of plasma glucose, serum insulin, TG and NEFA concentrations. The area under the concentration curve (AUC) was calculated with the trapezoidal method. Meal-stimulated insulin secretion (MSIS) was calculated as follows;

MSIS = (insulin at 30 min-fasting insulin)/ (glucose at 30 min-fasting glucose), where insulin in μU/mL and glucose in mg/dL.

Data were presented as mean ± SD unless otherwise stated. Due to deviation from normal distribution, HOMA-IR and inflammatory markers were logarithmically transformed for analysis. Correlation coefficients of TG AUC with cardiometabolic parameters were evaluated by Pearson correlation analysis. Stepwise multiple linear regression analyses were performed to further identify the most significant variables contributing to the variation of TG-AUC. A two-tailed $p<0.05$ was considered statistically significant. All calculations were performed with SPSS system 15.0 (SPSS Inc, Chicago, IL).

## RESULTS

As previously reported,[14,17] participants in total were normal weight, normolipidemic and normoglycemic (Table 1). They were also insulin-sensitive as demonstrated by HOMA-IR of 0.76. Athletes compared to non-athletes had lower percentage fat in any region measured in the present study. Although fat mass in arms and legs did not differ between the 2 groups, trunk fat and trunk/leg fat ratio were tended to be higher in athletes. The 2 groups did not differ in TG at 4 time points during a meal test, TG-AUC, HDL cholesterol and apolipoprotein A1 and B. Athletes compared to non-athletes had lower leptin but comparable adiponectin and hence lower leptin/adiponectin ratio. Although glucose-AUC did not differ between groups, insulin-AUC was lower in athletes, suggesting lower muscle insulin sensitivity in athletes.

When analyzed as a whole or separately (Table 2), TG-AUC showed a positive association with a ratio of trunk fat to leg fat whereas no association was found with BMI, waist circumference, fat mass and percentage fat of any region studied. TG responses were strongly and positively associated with fasting TG and TG at 30, 60 and 120 min in meal tests (data not shown) and apoB whereas there was no association with HDL cholesterol and apoA1. As a whole group, TG-AUC was positively associated with leptin/adiponectin ratio and log hsCRP but not with glucose, insulin, HOMA-IR, serum adiponectin, leptin, TNF-α and PAI-1.

We conducted multiple linear regression analysis for TG AUC as a dependent variable (Table 3, model A). The model included 5 variables as independent variables, which showed significant associations with postprandial lipemia in univariate analysis. Fasting TG emerged as the strongest determinant and explained 88.9% of variations of postprandial lipemia. In addition to fasting TG, trunk/leg fat ratio emerged as an independent determinant of postprandial triglyceridemia and the 2 variables explained 92.3% of variabilities of postprandial lipemia. Including total body fat, leg fat and trunk fat into the model as independent variables did not change the results (data not shown).

In order to confirm the association between trunk/leg fat ratio and TG-AUC, participants were stratified into 2 groups according to the median TG-AUC (133 mg/dL. 2h) (Table 4). Young women with high compared to low TG-AUC had higher trunk fat but comparable leg fat, and consequently, higher trunk/leg fat ratio. They had elevated leptin/adiponectin ratio as the result of elevated leptin and comparable adiponectin. ApoB and TG concentrations at 4 time points were all substantially higher in women with high compared to low TG-AUC while there

**Table 1.** Anthropometric and biochemical characteristics of young women and serum triglyceride responses to standardized meal

| | Total n=35 | Non-athlete n=18 | Athlete n=17 | *p*-value |
|---|---|---|---|---|
| BMI (kg/m$^2$) | 21.8±2.4 | 22.0±2.8 | 21.7±1.9 | 0.704 |
| Waist circumference (cm) | 73.8±6.3 | 73.5±6.9 | 74.1±5.7 | 0.766 |
| Arm fat (kg) | 1.2±0.6 | 1.4±0.7 | 1.1±0.6 | 0.121 |
| Leg fat (kg) | 6.1±2.1 | 6.5±2.5 | 5.6±1.5 | 0.211 |
| Trunk fat (kg) | 7.3±2.7 | 8.2±2.8 | 6.5±2.4 | 0.059 |
| Body fat (kg) | 15.2±5.2 | 16.7±5.7 | 13.6±4.2 | 0.083 |
| Percentage arm fat (%) | 22.5±8.7 | 26.4±8.3 | 18.4±7.3 | 0.005 |
| Percentage leg fat (%) | 29.1±6.7 | 32.5±6.9 | 25.5±4.3 | 0.001 |
| Percentage trunk fat (%) | 27.0±7.4 | 31.0±6.3 | 22.7±6.1 | 0.000 |
| Percentage body fat (%) | 26.3±6.6 | 29.9±6.1 | 22.5±5.0 | 0.000 |
| Trunk/leg fat ratio | 1.22±0.25 | 1.29±0.27 | 1.14±0.22 | 0.074 |
| Cholesterol (mg/dL) | 178±23 | 175±22 | 182±24 | 0.373 |
| Fasting TG (mg/dL) | 55±19 | 54±19 | 55±19 | 0.827 |
| TG 30 min (mg/dL) | 61±23 | 62±25 | 60±21 | 0.874 |
| TG 60 min (mg/dL) | 73±26 | 76±29 | 70±23 | 0.465 |
| TG 120 min (mg/dL) | 79±30 | 78±26 | 81±34 | 0.830 |
| TG-AUC (mg/dL. 2 hr) | 139±47 | 141±49 | 137±46 | 0.805 |
| HDL cholesterol (mg/dL) | 74±11 | 73±14 | 75±8 | 0.511 |
| Apolipoprotein A1 (mg/dL) | 159±14 | 155±16 | 164±12 | 0.080 |
| Apolipoprotein B (mg/dL) | 67±14 | 64±13 | 69±14 | 0.359 |
| PAI-1 (ng/mL) | 29±20 | 33±27 | 26±9 | 0.303 |
| TNF-α (pg/mL) | 0.63±0.20 | 0.65±0.26 | 0.60±0.11 | 0.409 |
| Leptin (ng/mL) | 7.0±4.3 | 9.0±4.7 | 4.9±2.6 | 0.003 |
| Adiponectin (μg/mL) | 11.2±4.5 | 11.3±4.3 | 11.1±4.8 | 0.923 |
| Leptin/adiponectin ratio | 0.72±0.55 | 0.92±0.63 | 0.51±0.36 | 0.025 |
| IL-6 (pg/mL) | 0.7±0.3 | 0.7±0.3 | 0.7±0.3 | 0.869 |
| hsCRP (μg/dL) | 42±91 | 31±50 | 54±121 | 0.471 |
| Fasting glucose (mg/dL) | 84±4 | 82±4 | 85±4 | 0.033 |
| Fasting insulin (μU/mL) | 3.7±2.0 | 4.1±2.0 | 3.3±2.1 | 0.258 |
| HbA1c (%) | 5.2±0.2 | 5.1±0.2 | 5.2±0.2 | 0.202 |
| HOMA-IR | 0.76±0.43 | 0.83±0.41 | 0.69±0.44 | 0.349 |
| Glucose-AUC (mg/dL. 2 hr) | 174±20 | 174±20 | 175±21 | 0.852 |
| Insulin-AUC (μU/mL. 2 hr) | 44±23 | 54±24 | 34±17 | 0.009 |

HOMA: homeostasis model assessment; IR: insulin resistance; AUC: area under the curve; PAI-1; plasminogen activator inhibitor-1; TG: triglycerides; TNF-α; tumor necrosis factor-α; IL-6; interleukin-6; hsCRP; high-sensitivity C-reactive protein.
Mean±SD.

was no difference in HDL cholesterol and apoA1 between the 2 groups. Markers of inflammation did not differ between groups.

We have done another analysis on associations of fat distribution and triglyceridemia (Table 5). In simple correlation analyses, trunk/leg fat ratio showed strong and positive associations with not only fasting TG but also postprandial triglyceridemia and hence TG-AUC. In contrast, there was no association of triglyceridemia with leg fat and associations with trunk fat were weak. However, after mutual adjustment, leg fat was negatively and trunk fat was positively associated with not only fasting TG but also postprandial triglyceridemia and hence TG-AUC. .

Although there was no difference in glucose at baseline and 120 min, insulin concentrations at 4 time points and HOMA-IR, plasma glucose at 30 and 60 min and hence glucose-AUC were lower in women with high compared to low TG-AUC. MSIS was calculated and logarithmically transformed. MSIS was tended to be higher (3.6±4.2 vs 1.3±1.6, $p$=0.052) and log MSIS was significantly higher in women with high compared with low TG-AUC (0.30±0.50 vs -0.04±0.31, $p$=0.026). Association between TG-AUC and log MSIS was significant in the total group (r=0.36, $p$<0.05) and in women with high TG-AUC (r=0.69, $p$<0.01) but not in those with low TG-AUC (r=0.08, NS). When log MSIS was added as an independent variable to model A of multiple linear regression analysis, log MSIS emerged as a significant determinant of TG-AUC (Table 3, model B).

## DISCUSSION

The present study has shown that even in healthy, normal weight women in early adult life postprandial lipemia was associated with fasting TG, trunk/leg fat ratio, apoB, leptin/adiponectin ratio, which would reflect compromised adipose tissue function and provide a useful index of insulin action,[16] and hsCRP, a marker of systemic low-grade inflammation. Among those, fasting TG and trunk/leg fat ratio were independent determinants of postprandial triglyceridemia. Women with high compared to low TG-AUC were characterized by higher trunk/leg fat ratio, elevated apolipoprotein B and leptin/adiponectin ratio. It should be emphasized that this investigation was conducted in 35 young, normal weight normolipidemic women, a population in which confounding factors are so scarce.[14,17]

Positive association between diurnal triglyceridemia and waist circumference, a crude marker of abdominal fat

**Table 2.** Correlation coefficients of the area under the curve of triglycerides (TG-AUC)

| | Whole | Non-athletes | Athletes |
|---|---|---|---|
| BMI (kg/m$^2$) | 0.26 | 0.37 | 0.07 |
| Waist circumference (cm) | 0.12 | 0.20 | 0.01 |
| Arm fat (kg) | 0.18 | 0.18 | 0.17 |
| Leg fat (kg) | 0.05 | 0.17 | -0.20 |
| Trunk fat (kg) | 0.33 | 0.42 | 0.22 |
| Body fat (kg) | 0.22 | 0.30 | 0.08 |
| % arm fat (%) | 0.16 | 0.11 | 0.21 |
| % leg fat (%) | 0.03 | 0.00 | 0.01 |
| % trunk fat (%) | 0.28 | 0.34 | 0.29 |
| % body fat (%) | 0.18 | 0.19 | 0.20 |
| Trunk/leg fat ratio | 0.55$^{**}$ | 0.54$^{*}$ | 0.60$^{*}$ |
| Fasting TG (mg/dL) | 0.92$^{***}$ | 0.97$^{***}$ | 0.88$^{***}$ |
| HDL cholesterol (mg/dL) | -0.20 | -0.28 | -0.06 |
| apolipoprotein A1 (mg/dL) | 0.01 | -0.09 | 0.20 |
| apolipoprotein B (mg/dL) | 0.65$^{***}$ | 0.72$^{**}$ | 0.62$^{*}$ |
| PAI-1 (ng/mL) | 0.11 | 0.09 | 0.18 |
| TNF-α (pg/mL) | 0.30 | 0.47$^{*}$ | -0.11 |
| Leptin (ng/mL) | 0.29 | 0.22 | 0.52$^{*}$ |
| Adiponectin (μg/mL) | -0.31 | -0.41 | -0.22 |
| Leptin/adiponectin ratio | 0.40$^{*}$ | 0.39 | 0.49$^{*}$ |
| IL-6 (pg/mL) | -0.06 | -0.17 | 0.12 |
| Log hsCRP | 0.35$^{*}$ | 0.45 | 0.26 |
| Fasting glucose (mg/dL) | 0.07 | 0.09 | 0.08 |
| Fasting insulin (μU/mL) | 0.28 | 0.30 | 0.25 |
| HbA1c (%) | 0.06 | 0.14 | 0.00 |
| HOMA-IR | 0.28 | 0.29 | 0.25 |
| G-AUC (mg/dL. 2 hr) | -0.31 | -0.22 | -0.40 |
| I-AUC (μU/mL. 2 hr) | 0.13 | -0.10 | 0.47 |

Abbreviations are the same as in Table 1.
$^{*}p<0.05$; $^{**}p<0.01$; $^{***}p<0.001$.

**Table 3.** Multiple linear regression analysis for area under the curve of triglycerides (TG-AUC) during meal tests as a dependent variable

| | Standardized β | *p* values | Cumulative $R^2$ |
|---|---|---|---|
| Model A | | | |
| Fasting triglycerides | 0.808 | 0.000 | 0.889 |
| Trunk/leg fat ratio | 0.175 | 0.001 | 0.923 |
| Model B | | | |
| Fasting triglycerides | 0.808 | 0.000 | 0.889 |
| Trunk/leg fat ratio | 0.175 | 0.001 | 0.923 |
| log MSIS | 0.097 | 0.039 | 0.930 |

Abbreviations are the same as in Table 1.
Model A included variables which showed significant associations with TG-AUC as independent variables: fasting triglycerides, trunk/leg fat ratio/apoB, leptin/adiponectin ratio and log hsCRP. Model B included log meal-stimulated insulin secretion (MSIS) in addition to model A.

accumulation, has been demonstrated in lean and overweight middle-aged adults.[10] Studies by computed tomography have shown positive association between postprandial lipemia and visceral adipose tissue accumulation in nonobese, normal glucose-tolerant middle-aged men,[11] in overweight/obese adults[9] and in men with impaired glucose tolerance.[12] In the present study using DXA, a gold standard measure of body composition, postprandial triglyceridemia was associated not with trunk fat, but with the ratio of trunk to leg fat in crude analyses. These findings have a twofold significance. Even in young female college students who were normal weight and normolipidemic postprandial TG clearance was associated with trunk/leg fat ratio. Independent and opposite associations of trunk and leg fat depots with adipokines, inflammatory markers, and metabolic syndrome including fasting TG have been shown in studies employing DXA including ours.[14,18,19] We confirmed and extended that young women showed the opposing associations between upper-body and lower-body adipose accumulation with not only fasting TG but also postprandial triglyceridemia and hence TG-AUC in the present study. Therefore, it is reasonable to assume that association of postprandial TG clearance with trunk/leg fat ratio in the present study could be combined effects of trunk and leg fat although the ratio of trunk to peripheral or leg fat ratio is a good marker of abdominal or central adiposity.[20]

In the present study, women with higher TG-AUC had elevated apoB, higher trunk/leg fat ratio, a marker of abdominal fat accumulation,[20] and higher leptin/adiponectin ratio, a marker of insulin resistance.[16] These findings may be in line with the observation that abnormalities in

**Table 4.** Anthropometric, biochemical and postprandial variables of young women stratified into 2 groups according to the median area under the curve of triglycerides (TG-AUC)

| | TG-AUC | | *p* values |
|---|---|---|---|
| | Low n=17 | High n=18 | |
| BMI (kg/m$^2$) | 20.9±1.6 | 22.7±2.8 | 0.029 |
| Waist circumference (cm) | 72.1±3.9 | 75.4±7.6 | 0.124 |
| Arm fat (kg) | 1.1±0.5 | 1.4±0.7 | 0.096 |
| Leg fat (kg) | 5.7±1.2 | 6.4±2.6 | 0.327 |
| Trunk fat (kg) | 6.2±1.5 | 8.4±3.2 | 0.017 |
| Total fat (kg) | 13.5±3.0 | 16.8±6.4 | 0.065 |
| Percentage arm fat (%) | 20.5±8.5 | 24.4±8.7 | 0.186 |
| Percentage leg fat (%) | 28.1±5.6 | 30.0±7.6 | 0.421 |
| Percentage trunk fat (%) | 24.5±6.4 | 29.3±7.8 | 0.058 |
| Percentage body fat (%) | 24.6±5.6 | 28.0±7.3 | 0.136 |
| Trunk/leg fat ratio | 1.10±0.23 | 1.33±0.23 | 0.006 |
| Glucose: fasting (mg/dL) | 84±5 | 84±4 | 0.708 |
| 30 min (mg/dL) | 113±12 | 100±14 | 0.007 |
| 60 min (mg/dL) | 91±19 | 78±16 | 0.027 |
| 120 min (mg/dL) | 76±12 | 73±10 | 0.507 |
| Insulin: fasting (μU/mL) | 3.1±1.1 | 4.3±2.5 | 0.078 |
| 30 min (μU/mL) | 29.1±15.7 | 36.8±19.3 | 0.206 |
| 60 min (μU/mL) | 28.1±20.0 | 27.2±13.1 | 0.871 |
| 120 min (μU/mL) | 11.3±7.8 | 13.6±8.2 | 0.399 |
| HbA1c (%) | 4.7±0.2 | 4.8±0.2 | 0.232 |
| HOMA-IR | 0.63±0.21 | 0.89±0.54 | 0.077 |
| log HOMA-IR | -0.22±0.14 | -0.13±0.28 | 0.244 |
| Glucose-AUC (mg/dL. 2 h) | 184±16 | 166±20 | 0.007 |
| Insulin-AUC (μU/mL. 2 h) | 42.0±25.1 | 46.7±22.0 | 0.566 |
| Cholesterol (mg/dL) | 168±18 | 188±22 | 0.006 |
| TG: fasting (mg/dL) | 41±10 | 68±16 | 0.000 |
| 30 min (mg/dL) | 43±11 | 78±17 | 0.000 |
| 60 min (mg/dL) | 51±12 | 94±16 | 0.000 |
| 120 min (mg/dL) | 56±14 | 102±22 | 0.000 |
| TG-AUC (mg/dL. 2 h) | 98±22 | 177±27 | 0.000 |
| HDL cholesterol (mg/dL) | 75±13 | 73±10 | 0.574 |
| Apolipoprotein A1 (mg/dL) | 158±14 | 160±15 | 0.689 |
| Apolipoprotein B (mg/dL) | 59±9 | 73±14 | 0.001 |
| PAI-1 (ng/mL) | 29±27 | 30±11 | 0.984 |
| TNF-α (pg/mL) | 0.6±0.1 | 0.7±0.3 | 0.211 |
| Leptin (ng/mL) | 5.1±2.2 | 8.8±5.1 | 0.010 |
| Adiponectin (μg/mL) | 12.4±4.9 | 10.1±3.8 | 0.123 |
| Leptin/adiponectin ratio | 0.44±0.20 | 0.98±0.64 | 0.003 |
| IL-6 (pg/mL) | 0.7±0.4 | 0.6±0.2 | 0.244 |
| hsCRP (μg/dL) | 28±49 | 55±118 | 0.383 |
| log hsCRP | 1.09±0.49 | 1.33±0.52 | 0.171 |

Abbreviations are the same as in Table 1.
Mean±SD.

**Table 5.** Associations (correlation coefficients) of trunk/leg fat ratio, trunk and leg fat with serum triglyceride levels during meal tests

| | Trunk/leg fat ratio | Trunk fat | | Leg fat | |
|---|---|---|---|---|---|
| | Simple | Simple | Leg fat adjusted | Simple | Trunk fat adjusted |
| TG 0 min | 0.450$^{**}$ | 0.262 | 0.511$^{**}$ | 0.049 | -0.487$^{**}$ |
| TG 30 min | 0.524$^{**}$ | 0.299 | 0.585$^{**}$ | 0.044 | -0.537$^{**}$ |
| TG 60 min | 0.607$^{**}$ | 0.399$^{*}$ | 0.665$^{**}$ | 0.097 | -0.571$^{**}$ |
| TG 120 min | 0.413$^{*}$ | 0.211 | 0.543$^{**}$ | -0.007 | -0.492$^{**}$ |
| TG-AUC | 0.549$^{**}$ | 0.327 | 0.630$^{**}$ | 0.053 | -0.563$^{**}$ |

$^{*}p<0.05$; $^{**}p<0.01$.
Abbreviations are the same as in Table 1.

apolipoprotein B metabolism have been reported to occur in the early phase of insulin resistance with abdominal obesity.[21] Although insulin resistant women with abdominal obesity[21] were normoglycemic and fasting normotriglyceridemic, waist and HOMA-IR averaged 125 cm and 4.1, respectively. In contrast, waist and HOMA-IR averaged 75.4 cm and 0.89, respectively, in our young women with higher high TG-AUC. These findings sug-

gest that modest increase in trunk fat may be associated with decrease in insulin sensitivity and elevated postprandial lipemia and apoB.

Association of post-meal TG with MSIS in our young women may be compatible with a study showing that not only resistance to insulin-mediated glucose disposal (as quantified by determining the steady-state plasma glucose concentration during the insulin suppression test) but the meal-induced insulin response was an independent predictor of degree of postprandial lipemia in healthy, nondiabetic subjects.[22] There are ample data showing that the plasma insulin response is highly correlated with degree of insulin resistance.[23,24]

Long-term endurance training has been shown to result in significant improvements in plasma lipids and lipoprotein profiles, including fasting TG, of sedentary, overweight subjects with mild to moderate dyslipidemia.[25] In the present study, however, long-term endurance training had no effect on triglyceridemia. This may be because of low fasting and postprandial triglyceridemia in a sedentary group, non-athletes: their mean TG increased from 54 at baseline to 78 mg/dL at 120 min after ingestion of test meals.

This study has several strengths, including a homogeneous study population with scarce confounding factors, and accurate and reliable measures of body composition by DXA. The main limitations of our study are small sample size and small amount of TG loaded: standardized meals contained only 17 g of TG. Although this amount when expressed by energy percentage is higher (33%) than usual Japanese breakfast (20-25%), this may be too small to evaluate postprandial TG response and 2 h of follow-up period may be short. However, mean increase in TG from 55 to 79 mg/dL by 24 mg/dL in our young women appeared to be comparable to TG increase by 31 mg/dL over baseline at 2 h after 15-g fat meals in young healthy men.[26] DXA does not allow separate quantification of visceral fat and subcutaneous fat in the trunk. However, the association of trunk fat with HOMA-IR was comparable to that of visceral adipose tissue accumulation by computed tomography.[27] The cross-sectional design of the present study complicates the drawing of causal inferences, and a single measurement of biochemical variables may be susceptible to short-term variation, which would bias the results toward the null. We used several surrogates in the present study, which may be less accurate.

### *Conclusion*

Trunk/leg fat ratio, a marker of central fat accumulation, is a significant predictor of postprandial lipemia even in young, lean and insulin-sensitive women independently of adiposity, insulin resistance, serum adiponectin and inflammation, suggesting a modifiable pathway to postprandial hypertriglyceridemia, a risk factor for coronary heart disease among Japanese men and women whose non-fasting TG averaged 145 and 136 mg/d/L, respectively.[28] Recently, baseline and change in visceral adipose tissue have been reported to be independent predictors for future development of atherogenic dyslipidemia in Japanese Americans.[29]

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**AUTHOR DISCLOSURES**

The authors declare no conflict of interest. No competing financial interests exist.

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