Gender Difference in Postprandial Lipemia
Importance of Visceral Adipose Tissue Accumulation

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Claude Bouchard, Pascale Maurière, Jean-Pierre Després

Abstract—Insulin resistance, hyperinsulinemia, hypertriglyceridemia, and low HDL-cholesterol concentrations are common features of a plurimetabolic syndrome, which increases the risk of coronary artery disease. Although it has been proposed that the development of atherosclerosis through alterations in plasma lipid levels could be a postprandial phenomenon, most studies on gender differences in plasma lipoprotein-lipid concentrations have reported fasting levels. Therefore, the aim of our study was to examine the response of postprandial triglyceride-rich lipoproteins to a standardized meal in 63 men and 25 women. In addition to the measurement of fasting and postprandial plasma lipid levels, numerous physical and metabolic variables were assessed, including body composition by underwater weighing and body fat distribution by computed tomography. Although no gender difference was noted in total body fat mass, men were characterized by a preferential accumulation of abdominal adipose tissue as revealed by an increased waist circumference and a greater visceral adipose tissue accumulation (50% difference) compared with women (P < 0.001). Men also showed a greater plasma triglyceride response (P < 0.005) as well as increased postprandial insulin and free fatty acid levels compared with women (P < 0.01). Visceral adipose tissue was significantly associated with the postprandial triglyceride response in both genders (men: r = 0.49, P < 0.0001; women: r = 0.43, P < 0.05). Finally, when men and women were matched for visceral adipose tissue accumulation, the gender difference in postprandial plasma triglyceride response was eliminated. Thus results of the present study suggest that the well known gender difference in visceral adipose tissue accumulation is an important contributing factor involved in the exaggerated postprandial triglyceride-rich lipoprotein response noted in men compared with women. (Arterioscler Thromb Vasc Biol. 1999;19:2448-2455.)

Key Words: postprandial lipemia • gender differences • visceral obesity • free fatty acids

Alterations in plasma lipoprotein-lipid concentrations are known to increase the risk of coronary artery disease (CAD) in both men and women.1,2 However, at all ages, the prevalence of CAD in women is lower than in men, and the gender difference in plasma lipoprotein-lipid levels as well as in the prevalence of type II diabetes are believed to be responsible, at least in part, for the higher CAD risk observed in men. Indeed, men are characterized by an overall less favorable plasma lipid profile, which includes high fasting triglyceride (TG) and low HDL-cholesterol concentrations compared with women.3 Men and women also show marked differences in indices of plasma glucose-insulin homeostasis.4,5 Furthermore, an increased visceral adipose tissue (AT) accumulation has been reported in men compared with women and this factor could also contribute to the gender difference in the CAD risk profile.6,7

Although the contribution of altered plasma lipoprotein-lipid levels to the increased risk of CAD is well known, most studies reporting such a relationship have examined fasting concentrations. However, Zilversmit8 has suggested that the development of atherosclerosis could be a postprandial phenomenon, and the renewed interest for postprandial studies has allowed the identification of various physiological conditions that influence postprandial lipoprotein metabolism. It has therefore been reported that age,9,10 diet,11 physical activity,12–14 non–insulin dependent diabetes mellitus15,16 as well as obesity,17 and body fat distribution18–21 all affect dietary fat clearance. In addition, a gender dimorphism has been reported in postprandial lipoprotein-lipid metabolism, as women generally show a lower postprandial triglyceride response to a dietary fat challenge compared with men.10,22 However, little is known about the physiological mechanisms responsible for this gender dimorphism.

Therefore, the aim of the present study was to examine the contribution of the gender difference in visceral AT accumulation to the variation in postprandial lipoprotein-lipid levels. For that purpose, 63 men and 25 women were investigated and their plasma triglyceride-rich protein (TRL) responses...
measured over a period of 8 hours after the ingestion of a standardized meal.

**Methods**

**Subjects**

Sixty-three men (mean age±SD: 45.0±10.0 years) and twenty-five premenopausal women (41.6±10.9 years) were recruited through the media and selected to cover a wide range of body fatness values. All women were tested during the follicular phase (between days 5 and 12) of their menstrual cycle. None of the women were using oral contraceptives. Subjects gave their written consent to participate in the study, which was approved by the Medical Ethics Committee of Laval University. Subjects with diabetes or with coronary heart disease were excluded from the present study. None of the subjects were on medication known to affect insulin action or plasma lipoprotein levels.

**Anthropometric and Body Composition Measurements**

Body weight, height, waist, and hip circumferences were measured following standardized procedures, and the waist-to-hip ratio was calculated. Body density was measured by the hydrostatic weighing technique. The mean of 6 measurements was used in the calculation of percent body fat from body density using the equation of Siri. Fat mass was obtained by multiplying body weight by percent body fat.

**Computed Tomography**

Visceral AT accumulation was assessed by CT, which was performed on a Siemens Somatom DRH scanner using previously described procedures. Briefly, the subjects were examined in the supine position with both arms stretched above the head. The scan was performed at the abdominal level (between L4 and L5 vertebrae) using an abdominal scout radiograph to standardize the position of the scan to the nearest millimeter. The total AT area was calculated by delineating the abdominal scan with a graph pen and then computing the AT surface with attenuation range of 250 to 400. Furthermore, in a pilot study (n = 5) conducted in our laboratory (Bergeron et al, unpublished observations, 1998) in which apo B-48 and apo B-100 concentrations were measured, it was found that although the protein concentration of the fraction designated large-TRL is very low, it is predominantly rich in apo B-48, with a minor contribution of apo B-100 particles. In contrast, the predominant apolipoprotein found in the fraction designated small-TRL was apo B-100 (>95% of total apo B). Finally, the fraction designated medium contained both apo B-48 and apo B-100. HDL particles were isolated from the bottom fraction (d<1.006 g/mL) after precipitation of apo B–containing lipoproteins with heparin and MgCl₂. The triglyceride and cholesterol contents of each fraction, ie, large-, medium-, and small-TRL, as well as HDL, were quantified on the Auto-Analyzer. All lipoprotein isolation procedures were completed within 2 to 3 days of the fat load. Plasma free fatty acid (FFA) levels were also measured at 0, 2, 4, 6, and 8 hours using an enzymatic method. Total apo B concentration was measured in plasma by the rocket immunoelectrophoretic method, as previously described. The lyophilized serum standard for apo B measurement was prepared in our laboratory and calibrated with reference standards obtained from the Centers for Disease Control.

**Glucose and Insulin Concentrations**

Fasting and postprandial plasma glucose concentrations were determined using the glucose oxidase assay (SIGMA). Plasma insulin levels were measured by a commercial double antibody radioimmunoassay (LINCO Research) that shows little cross-reactivity (<0.02%) with proinsulin.

**Statistical Analyses**

Pearson product-moment correlation coefficients were used to quantify associations between variables. Differences between men and women were tested for significance using the Student’s t test. The different areas under the curve of TG, FFA, insulin, and glucose concentrations were determined by the trapezoidal method. Multiple regression analyses were performed to quantify the independent contributions of age, gender, fat mass, abdominal visceral, and subcutaneous AT to the variance of postprandial plasma triglyceride residues. Fasting TG, HDL-cholesterol, insulin, and FFA levels, as well as postprandial insulin and FFA responses, were also included in the statistical model. All analyses were conducted on the SAS statistical package (SAS Institute).

**Results**

Physical characteristics and fasting metabolic profiles of men and women are presented in Table 1. Although both genders had the same amount of total body fat, there were significant differences in body fat distribution. Indeed, men were characterized by an increased abdominal AT accumulation as expressed by higher waist circumference and waist-to-hip ratio values compared with women. Furthermore, men displayed a greater amount of visceral AT than women. In contrast, significantly higher levels of abdominal subcutaneous AT were noted in women compared with men. Gender differences in the fasting metabolic risk profile were also observed as men were characterized by increased plasma cholesterol, triglyceride, and glucose levels, as well as by decreased plasma HDL-cholesterol concentrations compared with women. Men also showed higher fasting plasma insulin levels than women, but this difference did not reach statistical significance.
Figure 1 illustrates plasma TG as well as the triglyceride content of the various TRL subfractions of men and women throughout the entire postprandial period. Men showed significantly higher triglyceride levels at all times compared with women. These higher plasma triglyceride levels noted during the postprandial period resulted in a greater triglyceride response in men compared with women and were also reflected by significantly higher TG levels in all TRL fractions (total, large, medium, small) at all time points. Furthermore, gender differences were also observed in postprandial insulin and FFA concentrations, as men displayed higher postprandial insulin and FFA levels compared with women (Figure 2).

In both genders, increased adiposity was associated with a greater postprandial lipemia, as body fat mass as well as abdominal visceral and subcutaneous AT were positively correlated with the plasma triglyceride response (Figure 3). However, the relationship of visceral AT to plasma triglyceride response did not appear to differ between men and women (Figure 3C). We also found that in men, abdominal visceral AT, but not subcutaneous AT, was positively associated with the postprandial FFA response (Figure 4). However, this association was not observed in women.

As shown in Table 2, some variables of the fasting metabolic profile were associated more closely with the postprandial plasma triglyceride response than adiposity indices. Indeed, increased fasting plasma triglyceride and insulin levels were predictive of a greater triglyceride response in men, whereas women showed a greater response to increased fasting plasma triglyceride levels.

**TABLE 1. Physical Characteristics and Fasting Metabolic Profile of the Subjects**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>63</td>
<td>25</td>
</tr>
<tr>
<td>Age (y)</td>
<td>45.0 ± 10.0</td>
<td>41.6 ± 10.9</td>
</tr>
<tr>
<td>Body-mass index (kg/m²)</td>
<td>29.0 ± 4.1</td>
<td>26.9 ± 6.0</td>
</tr>
<tr>
<td>% Body fat</td>
<td>27.4 ± 6.3</td>
<td>26.4 ± 11.4</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>24.5 ± 8.4</td>
<td>26.8 ± 14.3</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>98.3 ± 10.2</td>
<td>82.6 ± 13.1§</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>0.95 ± 0.06</td>
<td>0.81 ± 0.06§</td>
</tr>
<tr>
<td>Abdominal adipose tissue areas (cm²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visceral</td>
<td>148 ± 63</td>
<td>99 ± 57†</td>
</tr>
<tr>
<td>Subcutaneous</td>
<td>275 ± 108</td>
<td>348 ± 194*</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>5.09 ± 0.80</td>
<td>4.39 ± 0.75‡</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>1.87 ± 0.93</td>
<td>1.24 ± 0.61*</td>
</tr>
<tr>
<td>HDL-cholesterol (mmol/l)</td>
<td>0.91 ± 0.21</td>
<td>1.14 ± 0.28†</td>
</tr>
<tr>
<td>Cholesterol/HDL-cholesterol ratio</td>
<td>5.84 ± 1.55</td>
<td>4.07 ± 1.20§</td>
</tr>
<tr>
<td>Free fatty acids (mmol/l)</td>
<td>0.64 ± 0.24</td>
<td>0.71 ± 0.24</td>
</tr>
<tr>
<td>Apolipoprotein B (g/l)</td>
<td>1.07 ± 0.21</td>
<td>0.90 ± 0.19‡</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>5.09 ± 0.61</td>
<td>4.70 ± 0.45*</td>
</tr>
<tr>
<td>Insulin (pmol/l)</td>
<td>96.2 ± 52.6</td>
<td>78.1 ± 30.8</td>
</tr>
</tbody>
</table>

Significantly different from the men at *P < 0.005; †P < 0.001; ‡P < 0.0005; §P < 0.0001.

Figure 1. Postprandial plasma (A), as well as total- (B), large- (C), medium- (D), and small-TRL (E) triglycerides concentrations in men (M; n = 63; black circles and bars) and women (W; n = 25; white circles and bars). Bars represent the areas under the incremental curves (responses). Values are expressed as means ± SEM. Significantly different from women at *P < 0.05; †P < 0.01; ‡P < 0.005.

Figure 2. Postprandial glucose (A), insulin (B), and FFA (C) concentrations in men (M; n = 63; black circles and bars) and women (W; n = 25; white circles and bars). Bars represent the areas under the incremental curves (responses). Values are expressed as means ± SEM. Significantly different from women at *P < 0.05; †P < 0.01; ‡P < 0.005.
both men and women. We also found that low fasting HDL-cholesterol levels were associated with an increased triglyceride response and that elevated fasting apo B concentrations were correlated with higher postprandial triglyceride levels. However, these latter relationships were only noted in men.

To further examine the importance of visceral AT accumulation to the gender difference in postprandial lipemia, we matched men and women on the basis of visceral AT (Table 3) regardless of total body fat mass and examined their respective postprandial triglyceride responses. Despite having identical levels of visceral AT, women were characterized by increased overall adiposity and abdominal subcutaneous AT accumulation. After the matching procedure, the difference in postprandial plasma triglyceride levels was no longer significant between men and women (Figure 5). We also compared the plasma triglyceride responses of men and women who were matched for total body fat mass and abdominal subcutaneous AT. These comparisons revealed a greater postprandial plasma triglyceride response in men than in women (Figure 5). We also examined the impact of matching subjects for visceral AT on the triglyceride responses in the various TRL subfractions (Figure 6). We found no significant difference in total-, large-, and medium-TRL triglyceride responses between men and women. On the other hand, the matching procedure failed to eliminate the difference in small-TRL levels as women were characterized by a lower triglyceride response in this subfraction compared with men. Matching men and women for visceral AT also affected postprandial plasma glucose and insulin responses as shown both genders were characterized by similar glucose and insulin responses after pairing men and women for visceral AT accumulation (Figure 7). However, women were still characterized by a lower FFA response after the fat load compared with men.

Finally, we conducted multiple regression analyses to quantify the independent contributions of age, gender, and adiposity indices, as well as fasting and postprandial metabolic profile variables to the variance of the postprandial plasma triglyceride response (Table 4). Fasting triglyceride concentration was by far the best predictor of plasma triglyceride response, accounting for more than 61% of its variance (Model 1). However, when fasting TG level was removed from the model (Model 2), fasting apo B level showed the greatest contribution to the plasma triglyceride response (37%). In Model 3, both fasting TG and apo B levels were excluded on purpose from the statistical model. In this restricted model, 27% of the variance of the plasma triglyceride response was attributed to visceral AT accumulation. It seems important to point out that, in all models, postprandial FFA response and fasting insulin concentration were significant predictors of the postprandial plasma triglyceride response. However, gender per se did not contribute to the variation in postprandial triglyceride levels after control for visceral AT accumulation and related fasting metabolic variables (TG, apo B, FFA, and insulin).

**Discussion**

Gender differences in fasting plasma lipoprotein-lipid concentrations have already been reported. In the present study, we also found that men were characterized by increased fasting plasma cholesterol and triglyceride levels as well as by decreased HDL-cholesterol concentrations compared with women. In addition, men also displayed higher fasting plasma glucose and insulin levels than women, although the gender difference in fasting insulinemia did not reach statistical significance. These metabolic alterations are considered as features of the insulin-resistance syndrome. On the other hand, it has been suggested that differences in adiposity, especially in body fat distribution, between men and women may be involved in the gender dimorphism noted

**TABLE 2. Correlations Between Fasting Metabolic Profile Variables and the Postprandial Triglyceride Response in Men and Women**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Men (n=63)</th>
<th>Women (n=25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.20</td>
<td>0.36</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>0.78†</td>
<td>0.74†</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>$-0.43^*$</td>
<td>$-0.10$</td>
</tr>
<tr>
<td>Free fatty acids</td>
<td>$-0.08$</td>
<td>0.02</td>
</tr>
<tr>
<td>Apolipoprotein B</td>
<td>0.58†</td>
<td>0.39</td>
</tr>
<tr>
<td>Fasting insulin</td>
<td>0.43†</td>
<td>0.77†</td>
</tr>
</tbody>
</table>

Significant at *P < 0.005, †P < 0.001.
TABLE 3. Body Fatness and Adipose Tissue Distribution of Men and Women Matched for A) Total Body Fat Mass, B) Abdominal Subcutaneous Adipose Tissue, or C) Visceral Adipose Tissue

<table>
<thead>
<tr>
<th>Variables</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>A) Matched for fat mass</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of subjects</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>24.8±2.3</td>
<td>24.9±2.3</td>
</tr>
<tr>
<td>Abdominal adipose tissue areas (cm²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visceral</td>
<td>152±17</td>
<td>98±12*</td>
</tr>
<tr>
<td>Subcutaneous</td>
<td>282±28</td>
<td>320±29</td>
</tr>
<tr>
<td>B) Matched for abdominal subcutaneous AT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of subjects</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>27.9±8.4</td>
<td>23.4±8.7</td>
</tr>
<tr>
<td>Abdominal adipose tissue areas (cm²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visceral</td>
<td>168±51</td>
<td>94±52†</td>
</tr>
<tr>
<td>Subcutaneous</td>
<td>305±100</td>
<td>304±98</td>
</tr>
<tr>
<td>C) Matched for abdominal visceral AT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of subjects</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>21.1±7.5</td>
<td>31.1±13.4*</td>
</tr>
<tr>
<td>Abdominal adipose tissue areas (cm²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visceral</td>
<td>117±51</td>
<td>117±52</td>
</tr>
<tr>
<td>Subcutaneous</td>
<td>234±103</td>
<td>409±179†</td>
</tr>
</tbody>
</table>

Significantly different from the men at *P<0.01; †P<0.001.

in plasma lipoprotein-lipid levels. Indeed, men are known to present a preferential accumulation of AT in the abdominal visceral depot, whereas women are characterized by a more peripheral AT distribution. In the present study, we found that despite having similar levels of total body fat in kg compared with women, men were characterized by an increased abdominal fat accumulation as indicated by higher waist circumference and visceral AT values.

Significant gender differences were also noted in postprandial lipemia men being characterized by a greater postprandial plasma triglyceride response compared with women. This result is concordant with previous observations that reported higher postprandial triglyceride levels in men than in women. In the present study, gender differences in the postprandial triglyceride response profile were also noted. In men, plasma triglyceride levels peaked later during the postprandial period than in women. Because the increase in triglycerides (from 2 to 4 hours) after meal consumption mainly reflects dietary TG absorption, whereas the return to fasting levels (from 6 to 9 hours postprandially) is presumably a function of TRL clearance, our results of a delayed postprandial TG peak in men suggest an impaired postprandial clearance of TRL compared with women.

The strong association between fasting TG and the postprandial plasma triglyceride response indicates that fasting triglyceridemia is an important correlate of the gender difference in postprandial lipemia. Indeed, on their entry into the circulation, both newly synthesized and endogenous TRL compete for lipoprotein lipase (LPL) to be hydrolyzed. Thus, in men, the increased quantity of TRL before the meal as indicated by their fasting hypertriglyceridemic state may contribute to the delayed clearance of postprandial triglycerides from the plasma. This accumulation of TRL related to the presumed “saturation” of LPL would also postpone the postprandial peak plasma triglyceride concentration. The gender difference in TRL clearance after a meal could also be the result of an increase in the contribution of hepatic TRL to total-TRL at the late stages of the postprandial period. Under insulin-resistant conditions, the antilipolytic effect of insulin on AT is very weak, which would explain the raised FFA levels observed postprandially in subjects with visceral obesity. This increased flux of FFA to the liver would promote the synthesis and secretion of VLDL. This model is supported by results presented in Figure 2. Indeed, we noted that, in men, there was a progressive increase in plasma FFA levels, which resulted in 8-hour plasma FFA concentrations that remained well above fasting values, whereas in women, plasma FFA levels at the end of the postprandial period were near fasting concentrations. Furthermore, our results are supported by a previous study in which men and women had been shown to differ significantly in the postprandial regulation of AT lipolysis. Indeed, men were characterized by an AT nonesterified fatty acid release that was more resistant to the postprandial antilipolytic effect of insulin. On the other hand, the increase in postprandial FFA concentrations may also be resulting from an increased lipolysis of TRL by LPL paralleled by an inadequate esterification of FFA into triglycerides by the AT. However, the present study did not allow us to quantify the contribution of both physiological processes, ie, increased AT or TRL lipolysis, to the increased postprandial FFA response in men. Further studies in this area are clearly warranted.

Nevertheless, our results indicate that visceral AT accumulation plays a major role in the gender difference in postprandial lipemia. Accordingly, we found no difference in postprandial lipemia among men and women matched for visceral AT accumulation. We also have examined the impact of the matching procedure on the different TRL subclasses. We found that there was no difference in total- as well as in large- and medium-TRL triglyceride responses between men and women with similar levels of visceral AT. However, a gender difference remained in the small-TRL triglyceride response as men had higher 2-, 4-, and 6-hour small-TRL triglyceride concentrations in men (M; black circles and bars) and women (W; white circles and bars) matched for body fat mass (20 pairs) (A), as well as abdominal subcutaneous (16 pairs) (B) and visceral AT accumulation (19 pairs) (C). Bars represent the areas under the incremental curves (responses). Values are expressed as means±SEM. Significantly different from women at *P<0.05; †P<0.01; ‡P<0.005.
concentrations compared with women matched for similar visceral AT accumulation. It is suggested that the remaining increased postprandial FFA response noted in men, after the matching procedure, may have contributed to their higher small-TRL triglyceride response compared with women. Particle size has also been suggested to affect the rate of TRL clearance, with large particles being better substrates for lipolysis by LPL than smaller particles. Our finding of a more prolonged accumulation of small-TRL triglycerides but not of large- and medium-TRL in men than women matched for visceral AT is concordant with this observation.

Multiple regression analyses revealed that the fasting triglyceride levels were by far the best predictors of the plasma triglyceride response to the fat load (Model 1). The contribution of apo B–containing lipoproteins to postprandial lipemia was also highlighted as fasting apo B concentration became the strongest predictor of plasma triglyceride response after fasting triglyceridemia was eliminated from the statistical model. Because apo B found in the fasting plasma is secreted through lipoproteins of hepatic origin, our results provide further support to the concept of a hepatic contribution to the delayed clearance of TRL in men. However, further studies are needed to support this notion and measurements of apo B-48 and apo B-100 containing TRL, which are respectively used as markers of TRL of intestinal and hepatic origin, will have to be performed in future studies.

It is known that visceral obesity is associated with metabolic abnormalities such as fasting hypertriglyceridemia, hyperinsulinemia, and increased apo B concentrations as well as lower HDL-cholesterol levels. Recently, we have reported that visceral obese men are characterized by an impaired postprandial TRL clearance compared with obese men with low levels of visceral AT. In the present study, when both fasting TG and apo B concentrations were eliminated on purpose from the multiple regression analyses, the amount of visceral AT was found to be the best predictor of TRL triglyceride response. Furthermore, we also quantified (data not shown) the independent contributions of total body fat mass and AT distribution variables (including subcutaneous abdominal fat measured by CT) to the fasting TG and apo B levels. In both cases, visceral AT accumulation was the best and only significant predictor of these metabolic variables. As women have less visceral AT than men, we tested the hypothesis that their preferential accumulation of subcutaneous AT may have a greater capacity for FFA esterification into adipose cell TG than omental AT. This peculiar metabolic behavior of subcutaneous fat is concordant with the faster clearance of TG-rich lipoproteins after a meal in women who have more subcutaneous AT than men. However, results of the present study support the notion that visceral AT may be more critical to the gender difference in postprandial lipemia than subcutaneous AT. Indeed, when men and women were matched on the basis of visceral AT accumulation, women remained characterized by a substantially greater subcutaneous AT depot compared with men (409 versus 234 cm², respectively). Thus, had subcutaneous AT had a greater impact on postprandial lipemia than visceral AT, a significant difference in postprandial TRL levels between men and women would have been expected even after they were matched for visceral AT accumulation. In the present study, such a difference in TRL response was minimal after men and women were matched for visceral AT accumulation.

Figure 6. Postprandial triglyceride concentrations of total- (A), as well as large- (B), medium- (C), and small-TRL (D) in men (M; n=19; black circles and bars) and women (W; n=19; white circles and bars) matched for visceral AT accumulation. Bars represent the areas under the incremental curves (responses). Values are expressed as means±SEM. Significantly different from women at *P<0.05; †P<0.01.

Figure 7. Postprandial plasma glucose (A), insulin (B), and FFA (C) concentrations in men (M; n=19; black circles and bars) and women (W; n=19; white circles and bars) matched for visceral AT accumulation. Bars represent the areas under the incremental curves (responses). Values are expressed as means±SEM. Significantly different from women at *P<0.05.
matched for visceral AT despite the fact that matched women had more subcutaneous AT than matched men.

Although matching men and women for the level of visceral AT eliminated the gender difference in plasma triglyceride response, there was a tendency for men to display a greater triglyceremic response compared with women. This gender difference was evident for the response of the small-TRL fraction. Other factors have been proposed to explain the lower postprandial lipemia in women than in men. For instance, estrogens have been suggested to have a favorable impact on postprandial triglyceridemia. Variation in LPL activity between men and women may also be implicated in the gender difference in postprandial lipemia. Once again, the greater accumulation of subcutaneous fat in women than in men could play a role in the gender difference noted in the clearance of TRL after a dietary fat challenge.

In summary, results from the present study indicate that there is a gender difference in postprandial lipemia as men show a greater postprandial triglyceridemic response to a meal than women. Although this difference is likely to result from the influence of several factors, our results suggest that the increased visceral AT accumulation in men makes an important contribution to this delayed dietary fat clearance. Concomitant impairment of postprandial FFA metabolism after meal consumption and a reduced ability to store lipids in subcutaneous AT may be responsible, at least in part, for this exaggerated lipemic response observed in men compared with women.

Acknowledgments

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