Differential Effects of Body Fatness and Body Fat Distribution on Risk Factors for Cardiovascular Disease in Women

Impact of Weight Loss

Karen E. Dennis, Andrew P. Goldberg

This study examines the role of obesity and body fat distribution (i.e., waist-to-hip ratio [WHR]) on cardiovascular disease (CVD) risk factors in 50 non-diabetic, obese (body mass index [BMI], 31±2 kg/m², mean±SD), 45±10-year-old women. The data obtained at baseline and after weight loss were analyzed after dividing subjects by WHR into upper-body (WHR >0.80) and lower-body (WHR ≤0.80) groups and by median-split BMI into more obese (BMI ≥31) and less obese (BMI <31) groups. At baseline, the upper-body obese women, when compared with lower-body obese women, had higher plasma triglycerides (TGs) (175±85 versus 111±47 mg/dL, respectively; \( P<.001 \)) and lower high-density lipoprotein cholesterol (HDL-C) (44±10 versus 54±11, respectively, \( P<.01 \)) but similar total and low-density lipoprotein cholesterol levels and blood pressure. There were no significant differences in these CVD risk factors at baseline by BMI split. Although weight loss (-9±5 kg) lowered blood pressure and TGs irrespective of WHR or BMI, only upper-body obese women raised HDL-C. Moreover, the magnitude of the changes was greatest in women with an upper-body fat distribution. In women with WHR >0.80, HDL-C increased by 11%, to 49 mg/dL (\( P<.001 \)), and TGs decreased by 24%, to 134 mg/dL (\( P<.001 \)). The increase in HDL-C with weight loss was predicted in a linear model by the initial WHR, whereas the reductions in TGs and blood pressure were predicted by the change in body weight. Thus, an upper-body fat distribution in women worsens the lipid risk factors for CVD posed by obesity, and weight loss is an effective intervention to improve lipid profiles in these women. Although weight loss improved CVD risk factors regardless of BMI or WHR, the magnitude of the increase in plasma HDL-C and decrease in TGs in women with an upper-body fat distribution suggests that weight loss in these women has the greatest potentiality of reducing their risk factors for CVD. (Arterioscler Thromb. 1993;13:1487-1494.)

KEY WORDS • obesity • body fat distribution • weight loss • cardiovascular disease risk factors • women

Early descriptions, which characterized men as having an abdominal body fat distribution and women as having a predominantly gluteofemoral body fat distribution,1-2 confirmed gender differences in the relation of obesity to cardiovascular disease (CVD). Intermediary mechanisms by which hyperlipidemia, hypertension, diabetes, and hyperinsulinemia increase risk for CVD seem linked to an abdominal distribution of body fat rather than obesity.3-5 These investigations underscore the importance of the distribution of body fat as a critical risk factor, irrespective of gender.

In women as well as men an upper-body distribution of fat, indexed as waist-to-hip ratio (WHR), is an independent risk factor for ischemic heart disease, diabetes, stroke, and premature mortality.6-8 Nevertheless, the strong relations between WHR and degree of obesity make it difficult to ascertain whether obesity per se or the distribution of body fat is the major determinant affecting risk factors for CVD. An examination of the effects of the interactions between body fat and WHR on CVD risk factors showed that there were no significant differences in CVD risk factors in lean women with a high or low WHR.9 In contrast, obese women with a high WHR had an increased risk-factor profile for CVD, reflected by elevated blood pressure (BP) and increased plasma lipids, glucose, and insulin levels. Yet a recent study of middle-aged women has revealed that an upper-body fat distribution is associated with significantly higher systolic BP, total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides (TGs) and lower high-density lipoprotein cholesterol (HDL-C), even after the data were statistically adjusted for body mass index (BMI).10 In that study, naturally occurring changes in WHR over a 3-year period were correlated with changes in HDL-C independent of changes in BMI. Since association does not imply causality, we sought to extend the focus and findings of these epidemiological studies by evaluating...
the effects of weight loss on risk factors for CVD in obese women.

Most of the studies that have examined the relation of obesity and body fat distribution to risk factors for CVD were cross-sectional in design. Sampling was done at one point in time, often in subjects who were vastly heterogeneous in degree of obesity, were markedly overweight, and/or had already demonstrated additional pathology such as non-insulin-dependent diabetes mellitus, dyslipidemia, or hypertension. Recent prospective population studies have added to the knowledge gained from years of cross-sectional work on the risk factors for CVD posed by obesity. However, there is a paucity of research that addresses the roles of obesity versus body fat distribution on risk factors for CVD and then determines the effects of weight loss in obese, healthy, adult women. The purpose of this study was to ascertain whether obesity or the regional distribution of fat was a more important determinant of lipoprotein lipid profiles and BP in obese adult women and to examine the effects of weight loss on these risk factors for CVD.

**Methods**

**Subjects**

Obese, healthy women were recruited by media advertisements and referrals from healthcare providers. In accordance with procedures approved by the Institutional Review Board at the medical center study site, the research was explained to subjects before their participation, and informed consent was obtained. Women were accepted for the program if they were 20% to 50% over the midpoint of medium-frame weight for their height based on 1983 Metropolitan Life Insurance tables. Medical history and physical examination with blood chemistry analyses from private physicians were reviewed to exclude subjects with endocrine disorders affecting metabolism (eg, diabetes and thyroid disease) as well as those on medications that might affect weight loss. Subjects were considered to be enrolled in the program (n=113) if they provided baseline data and attended at least four weight-loss sessions. Of the 54 women (48% of enrollees) who completed the program, we report data on the 50 who lost weight. The 9 women who reported smoking cigarettes and the 18 who were postmenopausal were naturally and evenly distributed across BMI and WHR groupings and were too few in number for an independent analysis of the effects of weight loss on their CVD risk factors.

**Measures**

Women were weight stable, had fasted for 14 hours, and emptied their bladders before all measurements. Baseline and posttreatment measures were taken within 3 weeks of the onset and termination of the weight loss program, respectively.

Body weight and height were taken with subjects wearing light indoor street clothing but without shoes. Weight and height measurements were used to calculate the BMI as weight (kg)/height (m)². Waist circumference was measured at the narrowest point superior to the hip, and hip circumference was measured at its greatest gluteal protuberance while women stood erect wearing only underwear. BP was taken with a mercury sphygmomanometer on the left arm after subjects had been sitting quietly for 5 minutes, with disappearance of Korotkoff sounds recorded as the diastolic pressure.

Fasting blood samples were drawn into tubes containing EDTA (1 mg/mL blood), and plasma was separated for analysis of TGs and cholesterol by enzymatic methods using an Abbott ABA Series II bichromatic analyzer. HDL-C was measured in the supernatant after precipitation of apoprotein B–containing lipoproteins with dextran sulfate; LDL-C was calculated as LDL-C=total cholesterol−(TG/5+HDL-C). For the assays in which these samples were analyzed, the coefficient of variation in 63 separate assays of standard pools for plasma TGs was 3.14%; for cholesterol, 2.81%; and for HDL-C, 3.78%.

**Weight-Loss Program**

The 9-month behavioral intervention for weight loss was based on a healthy approach to eating individualized to the women's values and preferences. The weight-reducing diet followed American Heart Association (AHA) Step I guidelines and approximated 1200 calories per day, with 50% to 55% of the calories composed of carbohydrate, 15% to 20% protein, and 30% to 35% fat. Behavior modification followed well-established formats, and the cognitive, emotional, and social dynamics that influence weight management were addressed in weekly, hour-long group discussions. Although the women were encouraged to begin a program of regular exercise (eg, walking) and were generally encouraged to increase their activity (eg, taking the stairs rather than the elevator), there were no formal exercise sessions. Biweekly monitoring revealed that although some women increased the frequency and duration of walking somewhat during the first weeks of the program, their physical activity patterns at the midpoint and end of the program were essentially stable and considered sedentary. None entered a formal exercise program, and by the second month of the program few indicated they had adopted a regular exercise or walking regimen into their lifestyle.

**Statistical Analysis**

Statistical analyses were performed on the total sample and after the women had been divided into groups based on body measurements. For the first group analysis, women were divided by the median pretreatment BMI of 31 kg/m², since no other naturally occurring data pattern pointed to a distinct empirical division; n=26 with BMI less than the median and n=24 with BMI greater than or equal to the median. For the second analysis, women were divided by pretreatment WHR, with a cutoff point of 0.80, based on the bimodal frequency distribution manifested in these data and the results of other investigators; n=18 with WHR ≤0.80 and n=32 with WHR >0.80. Finally, these cutoffs were used simultaneously to divide the women according to pretreatment BMI and WHR into four groups, with sample sizes ranging from 8 to 16 in each group. TGs were log normalized before analysis, and descriptive statistics (frequency, mean, standard deviation, range, and correlations) were calculated on all variables. Partial correlation and multiple regression analyses were conducted to ascertain the independent influences of WHR and BMI on CVD risk factors in the


Results

Subject Characteristics and CVD Risk Factors at Baseline

**Total group.** These 50 middle-aged (45±10 years; range, 22 to 63 years), obese women had a WHR of 0.83±0.06, indicative of an upper-body fat distribution, consistent with an adult female population. Women participating in this study averaged 163±6 cm in height (range, 147 to 178 cm). However, with a mean weight of 81.8±6.8 kg (range, 66.7 to 102.4 kg), they were 36±10% over their ideal body weight and had a mean BMI of 31±2 (range, 26 to 36). There was no relation between BMI and WHR (r=.12, P=NS) in these women.

Although mean lipoprotein lipid values were within normal limits for the total group, plasma TGs ranged from 36 to 344 mg/dL; total cholesterol, from 130 to 278 mg/dL; plasma LDL-C, from 57 to 210 mg/dL; and plasma HDL-C, from 32 to 79 mg/dL. Similarly, mean BP readings were within normal limits (Table 1), although systolic pressures ranged from 100 to 160 mm Hg and diastolic pressures, from 58 to 100 mm Hg; there was one outlier at 176/120 mm Hg.

**Grouped by BMI.** When women were divided into two groups according to a median-split BMI, the more obese women had larger waist and hip girths but a mean WHR identical to their less obese counterparts. There were no significant differences in lipoprotein lipids or blood pressure levels between the two BMI groups.

**Grouped by WHR.** When women were divided into two groups according to regional body fat distribution, the difference in WHR was due to disparity in the girth of the waist, not the hip. The degree of obesity (weight or BMI) and age of the women with an upper-body fat distribution (WHR >0.80) did not differ from those of women with a lower-body fat distribution (WHR ≤0.80). The women with an upper-body fat distribution had significantly higher TG and significantly lower HDL-C levels than women with a lower-body fat distribution. Fourteen (44%) of the 32 women with an upper-body fat distribution had an initial HDL-C of less than 40 mg/dL, which is the 10th percentile of the Lipid Research Clinics’ reference values, whereas there were no women with a lower-body fat distribution with an HDL-C in that range (χ² = 15.44, df 1, P<.0001).

There were no significant differences in total cholesterol, LDL-C, or BP in the women with an upper-body fat distribution compared with a lower-body fat distribution.

**Relations among CVD risk factors.** As expected from the preceding analyses by group, there was a significant inverse relation between HDL-C and WHR (r = −.36, P<.05) in the entire sample that did not change when the effect of BMI was controlled by using partial correlation analysis (r = −.38, P<.05). There was no relation between HDL-C and BMI (r = .11, P = not significant [NS]), nor did controlling for the effects of WHR increase the significance (r = .16, P=NS). The baseline log₁₀ TGs were directly related to the baseline WHR (r = .35, P<.05), and controlling for the effects of BMI did not alter the significance (r = .34, P<.05). There was no relation between TGs and BMI (r = .15, P=NS), and controlling for the effects of WHR did not alter the results (r = .11, P=NS).

Consistent with analyses by BMI and WHR groupings, there was no relation between
TABLE 2. Effects of Weight Loss on CVD Risk Factors

<table>
<thead>
<tr>
<th>CVD Risk Factor</th>
<th>Total Group</th>
<th>≥31</th>
<th>&lt;31</th>
<th>&gt;0.80</th>
<th>≤0.80</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>50</td>
<td>24</td>
<td>26</td>
<td>32</td>
<td>18</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72.7 ±8.6$§</td>
<td>72.7 ±8.6§</td>
<td>69.5 ±8.1*§</td>
<td>72.8 ±9.6§</td>
<td>72.5 ±6.8§</td>
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<tr>
<td>BMI, kg/m²</td>
<td>27 ±3§</td>
<td>27 ±3§</td>
<td>26 ±3§</td>
<td>28 ±4§</td>
<td>27 ±2§</td>
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<tr>
<td>Waist, cm</td>
<td>84.1 ±9.6$§</td>
<td>84.1 ±9.6§</td>
<td>80.8 ±8.0*$§</td>
<td>87.2 ±10.2$§</td>
<td>78.5 ±6.0$t§</td>
</tr>
<tr>
<td>Hip, cm</td>
<td>104.1 ±7.1§</td>
<td>104.1 ±7.1§</td>
<td>102.5 ±7.1§</td>
<td>104.2 ±7.7§</td>
<td>104.1 ±6.0§</td>
</tr>
<tr>
<td>WHR</td>
<td>0.81 ±0.07$§</td>
<td>0.81±0.07</td>
<td>0.79±0.06$§</td>
<td>0.84±0.06$‡</td>
<td>0.78±0.06‡</td>
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<tr>
<td>TG, mg/dL</td>
<td>117 ±54$‡</td>
<td>117 ±54$‡</td>
<td>117 ±62$‡</td>
<td>134 ±55$†</td>
<td>86 ±37$†</td>
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<tr>
<td>TC, mg/dL</td>
<td>193 ±38</td>
<td>193 ±38</td>
<td>184 ±6</td>
<td>199 ±41</td>
<td>193 ±31</td>
</tr>
<tr>
<td>LDL-C, mg/dL</td>
<td>117 ±33</td>
<td>117 ±33</td>
<td>111 ±25</td>
<td>123 ±35</td>
<td>107 ±28</td>
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<tr>
<td>HDL-C, mg/dL</td>
<td>52 ±11$</td>
<td>52 ±11$</td>
<td>50 ±11$</td>
<td>49 ±10$</td>
<td>56 ±10$*</td>
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<tr>
<td>Systolic BP, mm Hg</td>
<td>122 ±15$</td>
<td>122 ±15$</td>
<td>120 ±11$</td>
<td>123 ±15</td>
<td>121 ±16$</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>80 ±10$</td>
<td>80 ±10</td>
<td>78 ±9</td>
<td>81 ±10</td>
<td>78 ±10</td>
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</tbody>
</table>

BMI. Repeated-measures analysis of variance revealed significant differences in these variables between the baseline and post-weight-loss time periods but not between the groups of more versus less obese women.

Grouped by WHR. Regardless of their body fat distribution, the women significantly decreased weight, waist, and hip measurements with weight loss. Differential reductions in waist (−11±7%, P<.001) and hip girths (−8±5%, P<.001) after weight loss for the women with an upper-body fat distribution resulted in a significant decrement in WHR (−4±6%, P<.01), although after weight loss the mean WHR remained >0.80. In contrast, similar decreases in the waist (−10±5%, P<.001) and hip (−7±5%, P<.001) circumference after weight loss in the women with a lower-body fat distribution left the WHR unchanged (−2±7%, P=NS) in this group. Regardless of their initial body fat distribution, repeated-measures analysis of variance revealed that weight loss significantly lowered TGs by 23% in both groups, but did not change total cholesterol or LDL-C in these women. In contrast, weight loss significantly increased HDL-C in the women with an upper-body fat distribution (+11±14%, P<.001) but not in those with lower-body obesity (+5±17%). With weight loss, 11 of the 14 women with an upper-body fat distribution who presented with an HDL-C less than 40 mg/dL, 5±17%, P<.001) had HDL-C levels greater than 40 mg/dL. HDL-C levels rose in 11 of the 18 women with an upper-body fat distribution (4±6 mg/dL, 9±12%, P<.01) and in the 18 women with a lower-body fat distribution (2±9 mg/dL, 5±17%, P=NS). Despite improvements in lipid profiles with weight loss, women with an upper-body fat distribution continued to have significantly higher TG and lower HDL-C levels than women with a lower-body fat distribution. Weight loss significantly decreased systolic and diastolic blood pressure more than 10±5%, P<.001, and diastolic blood pressure more than 10±5%, P<.001, with these two indices of body composition.

Subject Characteristics and CVD Risk Factors After Weight Loss

Total group. As a group, the women lost a significant amount of their initial body weight (−11±7%, P<.001), with marked decreases in girth in both the waist (−11±7%, P<.001) and the hip (−8±5%, P<.001) sites. The change in circumference of the women was directly related to the amount of weight lost (BMI, r=−.69, P<.001) and to the change in WHR (r=−.76, P<.001). Because the reductions in the circumference of the waist were greater than for the hip, there was a statistically significant reduction in WHR (−1±7%, P<.001). Weight loss significantly lowered TGs (−23±16%, P<.001) and raised HDL-C (+10±23%, P<.001), but there were no changes in the total cholesterol (−3±12%, P=NS) or LDL-C (−3±22%, P=NS) levels. There were significant decreases in both systolic (−6±9%, P<.001) and diastolic (−5±10%, P<.001) BP with weight loss (Table 2).

Grouped by BMI. Regardless of their degree of obesity, weight loss significantly decreased both the waist and hip circumferences in the more as well as the less obese groups. For the more obese women, similar decrements in both waist (−2±8%, P<.001) and hip (−8±5%, P<.001) circumferences meant that the WHR did not change (−1±7%, P=NS). In contrast, with weight loss, the less obese women decreased their waist (−12±7%, P<.001) more than their hip (−7±5%, P<.001) girth, which significantly lowered their WHR (−5±5%, P<.001).

Women who were more obese when they began the program remained more obese at the end, with weight and waist circumferences significantly larger than those of the less obese women. However, hip girth was no longer different between the two groups after weight loss. Lipid profiles and BP significantly improved with weight loss when data were analyzed by median-split
Table 3. Effects of Weight Loss for Less Obese Women (BMI <31) by Body Fat Distribution

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Weight, kg</th>
<th>BMI, kg/m²</th>
<th>Waist, cm</th>
<th>Hip, cm</th>
<th>WHR</th>
<th>TG, mg/dL</th>
<th>TC, mg/dL</th>
<th>LDL-C, mg/dL</th>
<th>HDL-C, mg/dL</th>
<th>Systolic BP, mm Hg</th>
<th>Diastolic BP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td>77.3±4.2</td>
<td>29.0±1</td>
<td>96.0±4.6</td>
<td>110.4±4.0</td>
<td>0.87±0.4</td>
<td>184±90</td>
<td>193±27</td>
<td>115±25</td>
<td>184±90</td>
<td>129±13</td>
<td>84±10</td>
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<tr>
<td><strong>After Weight Loss</strong></td>
<td>68.3±8.25</td>
<td>26.3±6</td>
<td>83.7±7.95</td>
<td>101.8±7.66</td>
<td>0.82±0.4</td>
<td>144±62</td>
<td>188±30</td>
<td>114±28</td>
<td>144±62</td>
<td>119±13</td>
<td>78±9</td>
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<td><strong>Baseline</strong> (n=16)</td>
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<td><strong>WHR &gt;0.80</strong></td>
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<tr>
<td><strong>Baseline</strong></td>
<td>78.9±6.8</td>
<td>29.0±1</td>
<td>85.0±4.65</td>
<td>103.6±6.44</td>
<td>0.77±0.02</td>
<td>98±49</td>
<td>186±30</td>
<td>113±23</td>
<td>178±37</td>
<td>126±14</td>
<td>80±5</td>
</tr>
<tr>
<td><strong>After Weight Loss</strong></td>
<td>71.4±7.8†</td>
<td>26.2†</td>
<td>76.3±6.05</td>
<td>103.6±6.44</td>
<td>0.74±0.06†</td>
<td>74±31†</td>
<td>178±37</td>
<td>108±29</td>
<td>178±37</td>
<td>120±10</td>
<td>77±8</td>
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<td><strong>Baseline</strong> (n=10)</td>
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</table>

BMI Indicates body mass index; WHR, waist-to-hip ratio; TG, triglycerides; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; BP, blood pressure. Values are mean±SD.

*P<.01, †P<.001 between WHR groups.

§P<.01, ‡P<.001 change from baseline to after weight loss.

After weight loss, the more and the less obese women with either an upper-body or lower-body fat distribution had significant decreases in weight as well as waist and hip circumferences (Tables 3 and 4). However, after weight loss only the less obese women with an upper-body fat distribution significantly dropped their waist and hip girths to realize a significant decrease in WHR (-6.4%, P<.001; Table 3). Furthermore, only women with an upper-body fat distribution significantly raised their HDL-C, regardless of their BMI categorization as more or less obese (11±14%, P<.01 and 9±15%, P<.01, respectively). In all four groups, TGs dropped by more than 20%, whereas total cholesterol and LDL-C either did not change or decreased insignificantly. There were significant reductions in systolic BP only for women with an upper-body fat distribution, irrespective of their BMI. Nevertheless, in all four categories mean baseline and post-weight-loss BP readings were within normal limits.

Table 4. Effects of Weight Loss for More Obese Women (BMI ≥31) by Body Fat Distribution

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Weight, kg</th>
<th>BMI, kg/m²</th>
<th>Waist, cm</th>
<th>Hip, cm</th>
<th>WHR</th>
<th>TG, mg/dL</th>
<th>TC, mg/dL</th>
<th>LDL-C, mg/dL</th>
<th>HDL-C, mg/dL</th>
<th>Systolic BP, mm Hg</th>
<th>Diastolic BP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td>86.7±6.2</td>
<td>100.1±5.8</td>
<td>115.7±5.5</td>
<td>84±10</td>
<td>0.87±0.04</td>
<td>166±81</td>
<td>209±40</td>
<td>131±36</td>
<td>46±8</td>
<td>133±13</td>
<td>86±8</td>
</tr>
<tr>
<td><strong>After Weight Loss</strong></td>
<td>77.3±8.6§</td>
<td>90.8±11.2§</td>
<td>106.6±7.1§</td>
<td>78±9</td>
<td>0.85±0.08</td>
<td>125±47</td>
<td>210±48</td>
<td>134±42</td>
<td>51±10‡</td>
<td>127±16</td>
<td>84±9</td>
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<tr>
<td><strong>Baseline</strong> (n=16)</td>
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<td><strong>WHR &gt;0.80</strong></td>
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<tr>
<td><strong>Baseline</strong></td>
<td>84.7±4.2</td>
<td>88.9±3.1†</td>
<td>114.9±3.5</td>
<td>104.6±5.9§</td>
<td>0.77±0.02</td>
<td>127±41</td>
<td>206±33</td>
<td>123±24</td>
<td>57±13*</td>
<td>136±18</td>
<td>87±14</td>
</tr>
<tr>
<td><strong>After Weight Loss</strong></td>
<td>74.0±5.4§</td>
<td>81.1±5.1‡</td>
<td>104.6±5.9§</td>
<td>78±0.04*</td>
<td>0.76±0.04*</td>
<td>102±40</td>
<td>189±23</td>
<td>107±28</td>
<td>58±8</td>
<td>122±22</td>
<td>79±12</td>
</tr>
<tr>
<td><strong>Baseline</strong> (n=8)</td>
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</table>

BMI Indicates body mass index; WHR, waist-to-hip ratio; TG, triglycerides; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; BP, blood pressure. Values are mean±SD.

*P<.01, †P<.001 between WHR groups.

‡P<.01, §P<.001 change from baseline to after weight loss.
relation with WHR held after controlling for BMI ($r=.38, P<.05$) but disappeared with BMI after controlling for WHR ($r=.17, P=NS$). Significant bivariate positive correlations between systolic BP and BMI ($r=.29, P<.05$) as well as WHR ($r=.31, P<.05$) disappeared when the effects of BMI ($r=.22, P=NS$) and WHR ($r=.19, P=NS$) were controlled in partial correlation analyses. Diastolic BP correlated with WHR ($r=.30, P<.05$) in bivariate analysis, but this relation disappeared after controlling for BMI.

To examine the role of WHR in predicting risk-factor response to weight reduction, we ran a regression analysis (percent change risk factor = baseline WHR + percent change BMI + percent change WHR) for TGs, HDL-C, and systolic BP, variables that manifested significant relations in bivariate correlation analyses. However, because percent change in WHR was never a significant predictor of percent change in risk factor, we used a more parsimonious model (percent change risk factor = baseline WHR + percent change BMI). For percent change in HDL-C with weight loss, baseline WHR was a significant predictor ($P<.05$), whereas percent change in BMI was not significant. For both TGs and systolic BP, however, the percent change in BMI was a significant predictor ($P<.01$), and the initial WHR was not significant.

**Discussion**

Both obesity and upper-body fat distribution are risk factors for CVD in women. The improvements in lipid profiles and BP with weight loss that occurred in these subjects, regardless of body fat distribution, emphasize the role of obesity and the importance of weight reduction in decreasing risk for CVD in moderately obese women. However, the significantly lower baseline HDL-C and higher TGs found in middle-aged women with an upper-body fat distribution, independent of the degree of obesity, as well as the relation of HDL-C and TG levels to the initial WHR level but not BMI, strengthens the case for an upper-body regional distribution of fat as constituting the greater risk factor for CVD in obese women.

In the obese women in this study, HDL-C and TG levels were related to WHR independent of their degree of obesity. In the women with upper-body obesity, HDL-C and TG levels were lower and higher, respectively, at baseline than in women with a lower-body fat distribution, and these differences persisted after weight loss. When data were analyzed by a median-split BMI, however, there were no differences in lipid profiles or BP between the more and less obese women, and the improvement in these CVD risk factors with weight loss was of a comparably significant magnitude in both groups. The improvement in HDL-C with weight loss in these obese women was predicted by the initial WHR, not the magnitude of weight loss, whereas the reduction in TGs and BP correlated with the magnitude of the weight loss, not the WHR. This suggests that once a woman becomes obese, there seems to be no incremental worsening of these CVD risk factors within the 20% to 50% range of overweight or at a BMI between 26 and 36 kg/m². In contrast, within a more narrow range of WHR (>0.80), considered indicative of an upper-body pattern of fat distribution, there is a significant increase in lipoprotein risk factors for CVD. This is consistent with the heightened risk for mortality from CVD in women with an upper-body fat distribution. There was a significant increase in HDL-C with weight loss in the women with an upper-body distribution of fat but no significant change in the women with a lower-body fat distribution. It is of particular clinical relevance that 44% of the women with an upper-body fat distribution presented with an HDL-C below 40 mg/dL, whereas none of the women with a lower-body fat distribution had values that low. In the women with an upper-body fat distribution and low HDL-C, weight loss had a dramatic effect. The 23% increase in HDL-C raised their mean level above the 10th percentile. In the women with HDL-C above 40 mg/dL, the mean increase in HDL-C after weight loss was less, yet levels increased significantly in the women with an upper-body fat distribution. Irrespective of women’s regional body fat distribution, weight loss significantly lowered TGs and systolic BP from baseline values by a similar percentage. However, the 23% reduction in TGs in women with an upper-body fat distribution meant a larger absolute change in plasma TGs, since the initial TG value was higher in these women. Thus, from a clinical perspective, weight loss in women with an upper-body fat distribution brought their initially lower HDL-C and higher TG levels closer to the normal range, whereas weight loss in women with a lower-body fat distribution simply made already normal values even better. Since the relative change in HDL-C with weight loss was related to the initial WHR, and the magnitude of the reduction in TGs was greater in women with an upper-body fat distribution, even a small weight loss is likely to be beneficial for upper-body obese women, especially those with HDL-C below the 10th percentile of normal. We suspect that had women subjects with abdominal obesity achieved their ideal body weight, plasma HDL-C and TG levels might have normalized.

Most studies that examine obesity and an upper-body regional distribution of fat as risk factors for abnormal lipids, glucose intolerance, and elevated BP are cross-sectional. They focus on relations, not on the effects of body composition on risk factors for CVD in the same individuals before and after weight loss, where subjects are their own controls. Moreover, these studies usually examine relations in individuals diverse in the amount and location of adiposity and rarely account for the potential that there might be differential effects in women with an upper-body versus a lower-body fat distribution. Although the relations derived from studies of this type are convincing, they must be tempered by the fact that these analyses are correlational rather than causal. Thus, the potential benefits of interventions that change WHR or BMI on CVD risk-factor status can only be surmised by extrapolation. Moreover, in many studies the potentially high correlations between WHR and BMI are not considered in the analysis of the data, making it impossible to distinguish the effects of change in body weight from those of WHR.

In this study, the WHR was the same in the more and less obese women, and the subjects’ data were analyzed before and after weight loss: first, according to degree of adiposity; then according to body fat distribution; and finally, according to degree of adiposity within WHR grouping. Therefore, the effects of obesity and the
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Regional distribution of body fat on CVD risk factors were evaluated simultaneously as well as independently from each other. These discrete analyses were supported by regression analyses, the results of which emphasized the importance of upper-body fat distribution, regardless of degree of adiposity, on lipid risk factors for CVD in women who were 20% to 50% over recommended body weight. The results of the weight-loss intervention further supported previous findings of the deleterious effects of upper-body fat distribution on metabolic risk factors for CVD in obese subjects and demonstrated the potential health benefits of weight loss on CVD risk factors in women with abdominal obesity.

In a recent study of men and women before and after weight loss, Wing et al.27 found significant improvements in risk factors for CVD with weight loss. However, there were no significant changes in WHR in the women despite degrees of abdominal obesity and weight loss comparable to those observed in our subjects. Furthermore, for a weight loss of similar magnitude, the men in that study had greater decreases in waist circumferences than the women and smaller reductions in the hips, which resulted in a greater decrease in WHR. This was associated with greater improvements in HDL-C and plasma glucose levels in the men compared with the women. The changes in CVD risk factors were related to the changes in BMI in both men and women, not to changes in WHR. The differences in the results between studies may be due to the healthier status of our study population of obese women; the fact that we measured the minimal waist and not the circumference at the umbilicus; the fact that our sample size was smaller; and our focus on determining relative effects of weight loss on subjects divided by WHR and degree of obesity rather than only by correlational analyses. Reanalysis of the data of Wing et al.27 by WHR and BMI median cutpoints might reveal important differences between baseline and post-weight-loss levels of CVD risk factors in the women with an upper-body distribution of fat.

Weight reduction notwithstanding, alternative explanations for changes in lipid profiles in this study population should be considered. Exercise probably was not an influential factor, since women generally reported retention of their pretreatment, typically sedentary lifestyles. Changes in diet composition, however, probably accounted for some of the changes, since all women were encouraged to follow AHA dietary guidelines while reducing caloric intake. Group discussions emphasized the role of dietary fat and its unhealthy consequences. Although the lack of written documentation of dietary intake was a study limitation (food records were used as teaching tools for the women rather than for data collection), these records indicated that most of the women regularly made food selections that were lower in fat than their pretreatment choices. Therefore, the improvement in lipid profiles may reflect the adoption of AHA recommended food patterns as well as weight loss, since baseline sampling was performed before the implementation of these modifications in dietary composition. However, the results of some studies show that the adoption of AHA dietary guidelines under weight-stable conditions can have effects on lipoprotein lipids that are opposite to our results. Changing the fat and carbohydrate composition of the diet alone has not consistently lowered TGs28 or raised HDL-C.29-31 Moreover, Crouse32 recently suggested that the benefits of a low-fat diet in low-risk women are far from clear, since low-fat–high-carbohydrate diets, including the AHA Step I diet, may have the disadvantage of lowering HDL-C and raising TGs. Significant reduction in body weight in all groups, regardless of degree of adiposity or fat distribution, mediates the likelihood that only the changes in the type of food ingested influenced the physiological outcomes and supports the beneficial effects of weight loss on these lipoprotein lipid risk factors for CVD in obese women with an upper-body pattern of fat distribution. Therefore, weight loss as well as the changes in dietary composition must be considered the primary and synergistic factors that improved the lipid profiles of these obese women.

These results affirm the deleterious effects of an upper-body regional fat distribution in obese women, independent of the degree of obesity, on lipoprotein lipid risk factors for CVD and underscore the effectiveness of weight loss as a potentially effective intervention to ameliorate coexistent metabolic abnormalities in obese women with an upper-body distribution of fat. These findings also suggest that obese women with an upper-body fat distribution should be the primary target for intervention, since they are likely to benefit the most in prevention of CVD. However, the overall health benefits of weight reduction in preventing or mediating the myriad number of health complications posed by obesity, regardless of body fat distribution, cannot be ignored.

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