Waist to Hip Ratio in Middle-Aged Women
Associations With Behavioral and Psychosocial Factors and With Changes in Cardiovascular Risk Factors

Rena R. Wing, Karen A. Matthews, Lewis H. Kuller, Elaine N. Meilahn, and Pam Plantinga

Waist to hip ratio (WHR) was measured in 487 middle-aged women participating in the Healthy Women Study. Upper body fat distribution was found to be associated with numerous behaviors that affect cardiovascular risk, including smoking, low exercise levels, weight gain during adulthood, and higher caloric intake. Moreover, WHR was also associated with higher levels of anger, anxiety, and depression and lower levels of perceived social support. Women with upper body fat obesity had higher systolic blood pressure, total cholesterol, low density lipoprotein cholesterol, triglycerides, and apolipoprotein B and lower levels of high density lipoprotein (HDL) and the HDL subfractions 2 and 3. These associations remained significant after adjusting for body mass index. Among 108 women who had repeat measurements of WHR, changes in WHR over a 3-year period were significantly correlated with changes in activity and with decreases in HDL. Thus, WHR appears to be an integral component of the cardiovascular risk profile. WHR is related to those behaviors and psychosocial attributes that influence cardiovascular risk. (Arteriosclerosis and Thrombosis 1991;11:1250–1257)

Recent studies suggest that the distribution of body fat may be a stronger predictor of coronary heart disease (CHD) than the total amount of body fat. For example, in the Paris Prospective study, men who had an upper body fat distribution were at increased risk for CHD, independent of body mass index (BMI). Similarly, in a prospective study conducted in Sweden, the waist to hip ratio (WHR) was found to be positively associated with the incidence of myocardial infarction, stroke, and death from all causes in both men and women, after adjusting for BMI.

The effect of upper body fat distribution on atherosclerotic disease may be mediated by the effect on cardiovascular risk factors. WHR has been associated with increased blood pressure, increased triglycerides, decreased high density lipoprotein (HDL) cholesterol, and decreased high density lipoprotein (HDL) cholesterol. Body fat distribution has also been related to fasting and stimulated levels of glucose and insulin and to increased rates of diabetes.

To date, there have been few studies that have systematically investigated the behavioral or psychosocial factors associated with WHR. Smoking has been related to increased upper body fat distribution in both men and women, and a recent study showed that men who started to smoke had an increase in WHR. Activity level may also be related to WHR; men and women who reported more intense leisure-time activity were found to have lower WHRs, and exercise training has been shown to decrease WHR. Alcohol intake has also been related to WHR, with both positive and negative associations noted.

We are not aware of any previous studies that have examined the association between psychosocial variables and body fat distribution. Experimental studies with monkeys have shown that stress results in redistribution of fat to the abdominal area. Bjorntorp hypothesized that the same may be true for humans and that those individuals who experience a great deal of stress or who have difficulty coping with stress may experience higher cortisol levels and consequently may deposit more body fat in the abdominal area.

The purpose of the present article is to examine the behavioral and psychosocial correlates of WHR in a population-based sample of middle-aged women who were participating in the Healthy Women Study. In addition, we assessed the relation between WHR and cardiovascular risk factors in these women. Moreover, 108 of these women also had measures of WHR completed 3 years previously. Consequently, we could determine the relation between changes in behavior and changes in WHR and the association...
between changes in WHR and changes in cardiovascular risk factors.

Methods

Subjects

Subjects in this study were participants in the Healthy Women Study, a prospective investigation of changes in biologic and behavioral characteristics during the climacteric. Women were recruited into the study during 1983–1984 from a random sample of licensed drivers in Allegheny County, Pennsylvania. Eligibility criteria included age 42–50 years; menstruating within the past 3 months; no surgical menopause; diastolic blood pressure less than 100 mm Hg; and not taking insulin, thyroid medication, lipid-lowering drugs, estrogens, antihypertensive drugs, or psychotropic drugs.

Eighty-nine percent of women who were contacted agreed to a telephone interview, and 60% of those who were eligible volunteered to participate. A total of 541 women entered the study. Comparisons of participants with those who declined participation have been published previously23; participants were better educated and were employed in higher-paying jobs than either those who were ineligible or those who declined participation.

All women were invited to be restudied in 1987 after an average of 3 years in the study. The 487 women who completed the full reexamination form the sample for the present study. Of these 487 women, 282 were still premenopausal at follow-up and 62 were considered to be naturally postmenopausal, defined as not having menstruated for at least 12 months, not having undergone surgical menopause, and not having received hormone therapy in the past year. The remaining women were in the perimenopausal period (n=92), were using hormones (n=32), or had undergone surgical menopause (n=19).

WHR was added late to the baseline protocol and was measured on the final 120 women. One hundred eight of these women completed the follow-up assessment as well, and they form the subsample for the analyses of changes in WHR over time. The women who had WHR assessed at baseline were older (p<0.0001), had lower HDL cholesterol (p<0.05), and had higher apolipoprotein (apo) A-II (p<0.01) and apo B (p<0.0001) levels than did those for whom WHR was not assessed.

Procedures

Subjects were evaluated in the morning after a 12-hour fast for the following measures.

Lipids and apolipoproteins. Serum cholesterol,26 total HDL cholesterol,26 the subfractions 2 and 3 of HDL (HDL2 and HDL3),26 and triglycerides27 were assessed. Low density lipoprotein (LDL) cholesterol levels were estimated with the Friedewald equation (Friedewald et al28). Apos A-I and B29 were assessed by electroimmunoassay, and apo A-II was assessed by enzyme-linked immunosorbent assay.30

Glucose and insulin. Glucose and insulin were measured in the fasting state and 2 hours after a 75-g oral glucose load. Glucose was analyzed by enzymatic assay (Yellow Springs Glucose Analyzer, Yellow Springs, Ohio), and insulin was analyzed by radioimmunoassay.31

Blood pressure. Blood pressure was measured twice by the random-zero muddler method32 by observers certified according to the Multiple Risk Factor Intervention Trial protocol.33

Height, weight, and body mass index. Height and weight were obtained with a balance beam scale, and BMI, a measure of body fat, was obtained by dividing body weight in kilograms by height in meters squared. Subjects were asked to report what they had weighed at age 20.

Waist to hip ratio. The waist was measured at the smallest circumference and the hips at the largest circumference. A nonstretchable tape measure was used.

Measures of health habits. Cigarette smoking was defined by the total number of cigarettes smoked per day. The Paffenbarger activity questionnaire (Paffenbarger et al24) was used to obtain a measure of kilocalories per week spent in leisure-time activity. A 24-hour recall was used to obtain a measure of calories consumed and percent of calories from fat; the data were analyzed by use of a computerized nutrient data base.35,36

Standardized tests of personality and behavior. Subjects also completed a battery of tests of personality and behavior, including the Framingham Type A, Anger-Discuss, and Tension (Haynes et al37) scales; the Beck Depression Inventory (Beck et al38); and the Spielberger Trait Anger Anxiety Questionnaire (Spielberger et al39).

Results

Table 1 shows the basic characteristics of the sample. WHR averaged 0.77±0.07 (mean±SD), with a range from 0.45 to 0.99, and was significantly correlated with body weight (r=0.35, p<0.001) and BMI (r=0.40, p<0.001). WHR was higher in those with less education, but after adjusting for differences in BMI, there was no effect of education on WHR. WHR was not significantly related to age (r=0.04).

Determinants of Waist to Hip Ratio

WHR differed significantly as a function of menopausal status. Women who had remained premenopausal (n=282) had a WHR of 0.760 compared with a WHR of 0.787 in women who had experienced natural menopause and who were not receiving hormone therapy (n=62) (p<0.01). Thus, postmenopausal women had more upper body fat distribution. The difference remained significant (p<0.05) after adjusting for BMI.

WHR was also affected by smoking, as has been reported previously.15,16 The correlation between
Table 1. Characteristics of Sample (n=487)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waist to hip ratio</td>
<td>0.77</td>
<td>0.07</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.9</td>
<td>5.0</td>
</tr>
<tr>
<td>Weight (lb)</td>
<td>150.3</td>
<td>30.3</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>50.1</td>
<td>1.6</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>108.3</td>
<td>13.1</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>72.0</td>
<td>8.7</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>195.5</td>
<td>32.4</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dl)</td>
<td>58.6</td>
<td>14.0</td>
</tr>
<tr>
<td>HDL₂ (mg/dl)</td>
<td>18.6</td>
<td>9.7</td>
</tr>
<tr>
<td>HDL₃ (mg/dl)</td>
<td>39.8</td>
<td>7.5</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>91.9</td>
<td>55.6</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dl)</td>
<td>118.5</td>
<td>30.8</td>
</tr>
<tr>
<td>Fasting glucose (mg/dl)</td>
<td>88.0</td>
<td>12.1</td>
</tr>
<tr>
<td>2-Hour glucose (mg/dl)</td>
<td>93.4</td>
<td>31.7</td>
</tr>
<tr>
<td>Fasting insulin (µunits/ml)</td>
<td>10.6</td>
<td>6.9</td>
</tr>
<tr>
<td>2-Hour insulin (µunits/ml)</td>
<td>60.7</td>
<td>54.4</td>
</tr>
<tr>
<td>Apo A-I (mg/dl)</td>
<td>148.3</td>
<td>20.8</td>
</tr>
<tr>
<td>Apo A-II (mg/dl)</td>
<td>50.4</td>
<td>11.5</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>102.1</td>
<td>23.4</td>
</tr>
</tbody>
</table>

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL, high density lipoprotein; LDL, low density lipoprotein; apo, apolipoprotein.

number of cigarettes smoked per day and WHR was $r=0.13$, $p<0.01$. The correlation increased to $r=0.14$, $p<0.001$, after adjusting for BMI. Current smokers had an average WHR of 0.780±0.07 versus a WHR of 0.765±0.07 in former smokers and of 0.762±0.07 in those who had never smoked ($p<0.05$). The effect become more significant ($p<0.01$) after adjusting for BMI (Figure 1).

Another correlate of WHR was weight change since age 20 ($r=-0.38$, $p<0.0001$), indicating that women who gained the most weight since age 20 had the highest WHR. The correlation was of borderline significance ($r=-0.07$, $p=0.07$) after adjusting for current BMI.

WHR was also correlated with current caloric intake, $r=0.15$, $p<0.0001$. Caloric intake was unrelated to BMI ($r=0.04$), and hence the correlation between caloric intake and WHR remained significant after adjusting for BMI ($r=0.15$, $p<0.001$). Analysis of variance comparing women in the four quartiles of caloric intake showed that women who consumed more calories had higher WHRs (Figure 1), even after adjusting for BMI ($p<0.01$).

The relation between activity and WHR is also shown in Figure 1. Women were divided into quartiles according to their self-reported activity levels (0–500, 501–999, 1,000–1,999, and ≥2,000 kcal/week). As shown, women who reported the lowest levels of activity had the highest WHR. After adjusting for differences in BMI, the differences between quartiles remained marginally significant ($p=0.08$). The difference between the lowest exercise quartile and the other three quartiles was significant (0.784 vs. 0.766, $p<0.05$, after adjusting for BMI).

Stepwise multiple regression examining the effect of these behavioral attributes on WHR indicated that change in weight since age 20, number of cigarettes smoked per day, and caloric intake all contributed independently to the prediction of WHR. However, together these variables explained only 17% of the variance in WHR.

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Table 2 shows the correlations between WHR and psychosocial variables. As shown in the table, upper body fat distribution was associated with more tension and anxiety, more frequent angry feelings and expression of those feelings, higher levels of depressive symptomatology, and lower levels of perceived social support. Most of these associations remained significant when nonparametric correlations were used, after adjusting for BMI, or when the analyses...
were restricted to premenopausal women only. The psychosocial variables appeared to be more closely associated with WHR than with BMI.

**Waist to Hip Ratio and Coronary Heart Disease Risk Factors**

Pearson correlation coefficients were computed to examine the relation between WHR and CHD risk factors. These analyses were restricted to the women who remained premenopausal at the final clinic visit (n = 282), as menopausal status appears to affect both WHR and CHD risk factors. As shown in Table 3, WHR was positively correlated with blood pressure, triglycerides, cholesterol, apo B, glucose, and insulin (the latter two measures both in the fasting state and 2 hours after the glucose load). WHR was negatively correlated with HDL cholesterol and its subfractions. Partial correlations showed that both WHR and BMI were independently related to systolic blood pressure, total HDL cholesterol and its subfractions, triglycerides, 2-hour glucose, and fasting and 2-hour insulin. Figures 2 and 3 show the effects of both BMI and WHR on cholesterol HDL and insulin levels, after dividing the women into tertiles for each of these measures. It is notable that WHR was independently associated with both total cholesterol and LDL cholesterol, but BMI was not.

**Change in Waist to Hip Ratio From Baseline to Final Follow-up**

One hundred eight women had measures of WHR completed at entry into the study and again at the final followup. For these 108 subjects there was a nonsignificant increase in WHR over the 3 years (0.007±0.065). However, there were significant increases in both the waist circumference (79.1±12.1 to 80.9±12.8 cm, p<0.01) and in the hip circumference (101.0±11.0 to 102.5±12.0 cm, p<0.05) considered separately.

Of these 108 women, 58 had remained premenopausal and 31 had experienced natural menopause. There were nonsignificant changes in WHR over time for both groups, and there was no evidence that menopausal status affected the change.

On average, the women gained 5.1±9.8 lb during the 3-year interval. Change in weight was unrelated to change in WHR (r=-0.03). However, change in weight was significantly related to change in waist and hip circumferences considered separately (r=0.42 and 0.53, respectively; p<0.001 for both).

Table 4 presents the correlation between changes in WHR and changes in CHD risk factors. Pearson correlation coefficients, adjusting for changes in BMI, showed that those women who had the greatest increases in WHR had the greatest decreases in HDL2 cholesterol (r=-0.24, p<0.01) and the greatest increases in triglycerides (r=0.16, p=0.053). The correlation between change in WHR and change in HDL2 cholesterol remained significant when only women who were still premenopausal were considered (r=-0.31, p<0.01, unadjusted; and r=-0.32, p<0.01, adjusted for BMI).

Change in activity level was significantly correlated with change in WHR (r=-0.18, p<0.05). The correlation remained significant after adjusting for change in BMI (r=-0.18, p<0.05). Change in intake was not related to change in WHR. Only five women changed from smoking to nonsmoking status, precluding analysis of the effect of this factor on WHR.
Discussion

This study suggests that WHR, independent of BMI, is related to cardiovascular risk factors. WHR was found to be significantly and independently related to systolic blood pressure, total cholesterol, LDL cholesterol, HDL cholesterol and its subfractions, glucose, insulin, triglycerides, and apo B. Moreover, this study is the first to suggest that changes in WHR, independent of changes in BMI, may be related to changes in HDL₂ cholesterol; however, the change in WHR was not related to change in other cardiovascular risk factors.

Second, this study shows that various behaviors, including smoking, activity, and caloric intake, are related to WHR and that the change in activity is related to a change in the WHR. Upper body fat obesity was also associated with higher tension and anxiety scores and greater reports of anger, lower social support, and more depressive symptomatology.

Previous studies with samples of premenopausal and postmenopausal women have shown that WHR is positively associated with triglycerides, blood pressure, apo B, glucose, and insulin levels. Moreover, these studies have shown a negative association between WHR and HDL cholesterol and the HDL₂ subfraction. Although these correlations are not observed in all studies, the majority of the evidence does seem to suggest that WHR, independent of BMI, affects these CHD risk factors. The present study confirms these findings in one of the largest premenopausal cohorts of women studied to date.

The correlation between WHR and BMI was 0.40, p < 0.001, and was extremely similar to the relation observed by others. Moreover, those who had the greatest weight gain since age 20 had greater upper body fat distribution. Previous studies have shown that abdominal obesity is associated with fat cell hypertrophy, characteristic of those with adult-onset obesity. However, we found no evidence that the weight changes exhibited over the 3 years of the study were related to changes in WHR, despite the fact that the women had gained 5 lb on average and 24 of the 108 women had gained 10 lb or more. In contrast, we found that there were significant increases in both the waist and hip circumferences considered separately and that the changes in these circumference measures were related to weight.
change (usually weight gain). Thus, women during the perimenopausal period appear to be gaining weight but to be depositing it in both their waist and hip areas. Several other studies have examined the effect of weight change on WHR, with conflicting results. Wadden et al, for example, studied 68 female participants in a weight control program after a weight loss of 12.3 kg. These women showed decreases in WHR, from 0.827 to 0.817. Others have found no change in WHR with weight loss.

Results regarding the effect of menopause on WHR were unclear. When the total sample of women who had undergone natural menopause was compared with the sample who had remained premenopausal, significant differences were observed, indicating a higher WHR in postmenopausal women. However, when we used a within-subject analysis and examined the 31 women who were measured initially when premenopausal and then again later when postmenopausal, we found no significant change in WHR over time. The small sample of menopausal women may have obscured an effect of menopause; alternatively, differences between the group of premenopausal and postmenopausal women, other than their menopausal status, may have affected the between-group comparison. Furthermore, length of time since cessation of menses may also affect WHR. Previous data on the effect of menopause on WHR have also been unclear. Haffner et al found a weak relation between menopausal status and WHR, with menopausal status explaining 0.4% of the variance in WHR. Lanska et al found that menopausal status explained 0.07% of the variance.

Recent studies suggest that when female monkeys are exposed to standardized stressors, they respond with hyperinsulinemia, hyperlipidemia, hypertension, and increased abdominal body fat distribution. Likewise, Bjorntorp reported that men and women with abdominal body fat distribution have a number of symptoms suggestive of difficulty coping with stress, including psychosomatic disease. From these data, Bjorntorp hypothesized that increased stress, resulting in increased levels of catecholamines and adrenal cortical hormones, may lead to symptoms of hypercortisolism, including abdominal body fat. We examined this hypothesis indirectly by looking at the relation between WHR and several standard psychosocial questionnaires measuring variables related to stress. In support of the


### References


### Table 4. Correlation Between Change in Waist to Hip Ratio and Change in Coronary Heart Disease Risk Factors

<table>
<thead>
<tr>
<th>CHD risk factor</th>
<th>Correlation with change in WHR</th>
<th>Correlation with change in BMI adjusting for change in BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>0.04</td>
<td>0.05</td>
</tr>
<tr>
<td>Total HDL</td>
<td>-0.10</td>
<td>-0.11</td>
</tr>
<tr>
<td>HDL2</td>
<td>-0.24*</td>
<td>-0.24*</td>
</tr>
<tr>
<td>LDL</td>
<td>0.15</td>
<td>0.15</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>0.15</td>
<td>0.16</td>
</tr>
<tr>
<td>SBP</td>
<td>0.09</td>
<td>-0.08</td>
</tr>
<tr>
<td>DBP</td>
<td>-0.04</td>
<td>-0.02</td>
</tr>
<tr>
<td>Apo A-I</td>
<td>-0.06</td>
<td>-0.06</td>
</tr>
<tr>
<td>Apo A-II</td>
<td>0.09</td>
<td>0.09</td>
</tr>
<tr>
<td>Apo B</td>
<td>-0.02</td>
<td>-0.02</td>
</tr>
<tr>
<td>Fasting insulin</td>
<td>0.06</td>
<td>0.07</td>
</tr>
<tr>
<td>2-Hour insulin</td>
<td>0.04</td>
<td>0.05</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>2-Hour glucose</td>
<td>-0.01</td>
<td>-0.01</td>
</tr>
</tbody>
</table>

CHD, coronary heart disease; WHR, waist to hip ratio; BMI, body mass index; HDL, high density lipoprotein; LDL, low density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; Apo; apolipoprotein.

*p=0.007, tp=0.053.
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