Visceral Adiposity and the Prevalence of Hypertension in Japanese Americans

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Background—Visceral adiposity is generally considered to play a key role in the metabolic syndrome, including hypertension. The purpose of this study was to evaluate cross-sectionally whether visceral adiposity is associated with prevalence of hypertension independent of other adipose depots and fasting plasma insulin.

Methods and Results—Study subjects included 563 Japanese Americans with normal or impaired glucose tolerance or diabetes but not taking oral hypoglycemic medication or insulin at entry. Variables included plasma glucose and insulin measured after an overnight fast and during an oral glucose tolerance test, and abdominal, thoracic, and thigh fat areas by CT. Total fat area (TFA) was calculated as the sum of these fat areas. Hypertension was defined as having a systolic blood pressure ≥140 mm Hg, having a diastolic blood pressure ≥90 mm Hg, or taking antihypertensive medications. Intra-abdominal fat area (IAFA) was associated with a higher prevalence of hypertension. Adjusted odds ratio of hypertension by IAFA was 1.68 for a 1-SD increase (95% CI, 1.20 to 2.37) after adjusting for age, sex, fasting plasma insulin, a nonlinear transformation of 2-hour plasma glucose, and TFA. IAFA remained a significant predictor of prevalence of hypertension even after adjustment for total subcutaneous fat area, abdominal subcutaneous fat area, body mass index, or waist circumference, but no measure of regional or total adiposity was associated with the odds of prevalence of hypertension in models that contained IAFA.

Conclusions—Greater visceral adiposity increases the odds of hypertension in Japanese Americans independent of other adipose depots and fasting plasma insulin. (Circulation. 2003;108:1718-1723.)

Key Words: epidemiology ■ hypertension ■ visceral fat ■ obesity ■ risk factors

Metabolic syndrome is the cluster of obesity, insulin resistance, hyperinsulinemia, dyslipidemia, glucose intolerance, and hypertension.1 A central pattern of body fat distribution is now generally considered to play an important role in this syndrome. In particular, visceral adiposity has been reported to play a key role in these diseases compared with other measurements of regional or generalized obesity.2–4 But not known is whether visceral adiposity directly measured by CT increases the odds of hypertension, independent of other adipose depots, and insulin resistance.

Previous research on the association between greater central obesity, measured by the ratio of waist-to-hip circumference, or the ratio of subscapular to triceps skinfold thickness and the risk of hypertension were inconclusive.5–9 In these studies, controlling for other potentially confounding variables such as insulin resistance was not performed, subjects with borderline hypertension were classified as having normotension, or the definition of hypertension was not clear. The contribution of visceral fat was not distinguished from that of subcutaneous abdominal fat. Only limited cross-sectional data are available on relating visceral adiposity directly measured by CT to blood pressure. Two studies have reported a significant or borderline significant association,2,10 but one study has reported a null association.11 Differences in exclusion criteria, age distribution of study subjects, or the method of enrolling subjects may explain the inconclusive associations. In the present study, we therefore examined the relationship between directly measured visceral adiposity and the odds of hypertension independent of other measurements of total and regional adiposity and fasting plasma insulin.

Methods

Study Population
The study population consisted of second- and third-generation (mean age, 49.5±11.8 years) Japanese Americans with normal (NGT) or impaired glucose tolerance (IGT) or diabetes but not taking oral hypoglycemic medications or insulin, who were enrolled in the Japanese-American Community Diabetes Study. Details about selection and recruitment of the sample population have been described previously.12,13
Data Collection
All evaluations were performed at the General Clinical Research Center, University of Washington. The protocol for this research was reviewed by the Human Subjects Review Committee at the University of Washington, and signed, informed consent was obtained from all participants. An average blood pressure was calculated from the second and third of 3 consecutive measurements with a mercury sphygmomanometer read to the nearest 2 mm Hg with the patient in the recumbent position. Systolic blood pressure was determined by the first perception of sound and the diastolic fifth phase blood pressure at Korotkoff sound disappearance. Hypertension was defined as having a systolic blood pressure ≥140 mm Hg, having a diastolic blood pressure ≥90 mm Hg, or taking antihypertensive medications. A 75-g oral glucose tolerance test was used to classify all subjects as having NGT, IGT, or type 2 diabetes based on the American Diabetes Association 1997 criteria.14 Plasma glucose was assayed by an automated glucose oxidase method. Fasting plasma insulin was measured by radioimmunoassay as reported previously.14

Body mass index (BMI) was calculated as the weight in kilograms divided by the height in meters squared. Single CT scans were obtained of the thorax, abdomen, and right thigh to measure fat areas (cm²) as described previously.14 Visceral adiposity was measured as intra-abdominal fat area (IAFA) at the umbilicus level. This measurement has been reported to have a high correlation with directly ascertained total visceral fat volume by CT or magnetic resonance image.16,17 Subcutaneous fat area was also measured by CT scans of the thorax, abdomen, and right thigh. Total fat area (TFA) was calculated as the sum of IAFA and thorax and subcutaneous abdominal fat areas, and twice the right thigh subcutaneous fat area. In research that we have previously conducted, TFA correlates highly with fat mass as measured by densitometry among Japanese Americans (r=0.89 to 0.94; M.J. McNeely, unpublished data, 1999). Total subcutaneous fat area was defined as TFA minus IAFA. Waist circumference was measured at the level of the umbilicus to the nearest tenth centimeter.

Statistical Analysis
Multiple logistic regression analysis was used to estimate the odds ratio (OR) for hypertension presence in relation to an increase of 1 SD in baseline variables. Nonlinear effects of continuous independent variables were evaluated using quadratic and log transformations. The presence of an effect modification was tested by the insertion of first-order interaction terms into appropriate regression models. We calculated the 95% confidence interval for each OR. Probability values are two tailed. Statistical analyses were performed using the SPSS version 10.0 software package (SPSS Inc).

Results
Among the 563 eligible men and women, we confirmed 203 cases of hypertension. In univariate logistic regression analysis, IAFA, abdominal subcutaneous fat area, TFA, BMI, and waist circumference, but not total subcutaneous fat area, were associated with a higher prevalence of hypertension. Age, male sex, fasting plasma insulin, fasting plasma glucose, and 2-hour plasma glucose (2hPG) were also associated with higher prevalence of hypertension (Table 1).

The crude prevalence of hypertension according to tertiles of regional or total adiposity, fasting plasma insulin, and gender is shown in Table 2. All regional fat areas, total adiposity, fasting plasma insulin, and gender were associated with prevalence of hypertension. After subjects were stratified according to tertiles of TFA, subcutaneous abdominal fat area, total subcutaneous fat area, BMI, waist circumference, fasting plasma insulin, or gender, greater visceral adiposity was found to be associated with a higher prevalence of hypertension in all groups (Table 2). On the other hand, after subjects were stratified according to IAFA, no measure of regional or total adiposity other than IAFA, fasting plasma insulin, or gender was found to be associated with prevalence of hypertension (Table 2).

To assess the effect of IAFA on prevalence of hypertension independent of glycemic status, subjects were dichotomized by diabetes status (defined as hyperglycemia [IGT or type 2 diabetes] or NGT) (Table 3). In both categories, greater

**TABLE 1. Characteristics of Study Subjects According to Hypertension Status**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total (n=563)</th>
<th>Normotension (n=360)</th>
<th>Hypertension (n=203)</th>
<th>Crude OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female sex (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting plasma insulin (pmol/L)</td>
<td>83.5±45.9</td>
<td>76.8±37.8</td>
<td>95.4±55.8</td>
<td>1.45 (1.25–1.79)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting plasma glucose (mmol/L)</td>
<td>5.46±1.26</td>
<td>5.26±1.15</td>
<td>5.81±1.36</td>
<td>1.64 (1.32–2.05)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2hPG (mmol/L)</td>
<td>8.23±3.39</td>
<td>7.55±2.97</td>
<td>9.43±3.76</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>2hPG model [loge(2hPG)−0.0588(2hPG)]†</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>2.23 (1.79–2.78)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are mean±SD or %. ORs for continuous variables reflect a 1-SD-magnitude increase. Total subcutaneous fat area and TFA represent sums of adipose tissue areas as determined by multiple CT slices. P value is for univariate logistic regression analyses.

*See model below (†).†2hPG model = loge(2hPG)−β1/β2×(2hPG); β1 and β2 denote coefficients of 2hPG and loge(2hPG), respectively; β1 = −0.283, β2 = 4.815. Loge denotes natural logarithm.
visceral adiposity was associated with a higher prevalence of hypertension (Table 3). Furthermore, after subjects were stratified by IAFA tertile, subjects with IGT or type 2 diabetes had a higher prevalence of hypertension than those with NGT in each IAFA category (Table 3).

In multiple logistic regression analysis, insertion of quadratic or log transformations of all variables except 2hPG into all models of Table 4 did not improve their fit compared with the linear model. Because both the linear and the log transformation of 2hPG were significant in all models at $P<0.05$, both of these variables were retained in all models that included 2hPG. We examined the significance of the first-order interaction terms in all models of Table 4 between IAFA, TFA, subcutaneous abdominal fat area, total subcutaneous fat area, BMI, or waist circumference and the other variables. None of these interactions was significant.

A number of regression models were tested to assess the effects of body fat distribution on prevalence of hypertension (Table 4). After adjusting for age, sex, fasting plasma insulin, 2hPG, log transformation of 2hPG, and TFA, IAFA was associated with prevalence of hypertension (model 1, Table 4). Model 2 of Table 4 was identical to model 1, with the exception that quintiles of 2hPG were used in place of 2hPG and log transformation of 2hPG to make it easily understood.

### TABLE 2. Prevalence of Hypertension According to IAFA, TFA, Abdominal Subcutaneous Fat Area, Total Subcutaneous Fat Area, BMI, Waist Circumference, Fasting Plasma Insulin, and Gender

<table>
<thead>
<tr>
<th>Tertiles of IAFA</th>
<th>Total</th>
<th>Tertile 2</th>
<th>Tertile 3</th>
<th>$P$ for Trend</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>33/187 (17.6)</td>
<td>55/188 (29.3)</td>
<td>115/188 (61.1)</td>
<td>$&lt;0.001$</td>
<td>203/563 (36.1)</td>
</tr>
<tr>
<td>Tertiles of TFA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>21/118 (17.8)</td>
<td>16/57 (28.1)</td>
<td>10/12 (83.3)</td>
<td>$&lt;0.001$</td>
<td>47/187 (25.1)</td>
</tr>
<tr>
<td>Tertile 2</td>
<td>10/53 (18.9)</td>
<td>20/64 (31.3)</td>
<td>40/71 (56.3)</td>
<td>$&lt;0.001$</td>
<td>70/188 (37.2)</td>
</tr>
<tr>
<td>Tertile 3</td>
<td>2/16 (12.5)</td>
<td>19/67 (28.4)</td>
<td>65/105 (61.9)</td>
<td>$&lt;0.001$</td>
<td>86/188 (45.7)</td>
</tr>
<tr>
<td>$P$ for trend</td>
<td>0.750</td>
<td>0.968</td>
<td>0.802</td>
<td>$&lt;0.001$</td>
<td></td>
</tr>
<tr>
<td>Tertiles of abdomen subcutaneous fat area</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>20/107 (18.7)</td>
<td>16/49 (32.7)</td>
<td>19/31 (61.3)</td>
<td>$&lt;0.001$</td>
<td>55/187 (29.4)</td>
</tr>
<tr>
<td>Tertile 2</td>
<td>10/58 (17.2)</td>
<td>17/63 (27.0)</td>
<td>40/67 (59.7)</td>
<td>$&lt;0.001$</td>
<td>67/188 (35.6)</td>
</tr>
<tr>
<td>Tertile 3</td>
<td>3/22 (13.6)</td>
<td>22/76 (28.9)</td>
<td>56/90 (62.2)</td>
<td>$&lt;0.001$</td>
<td>81/188 (43.1)</td>
</tr>
<tr>
<td>$P$ for trend</td>
<td>0.586</td>
<td>0.770</td>
<td>0.822</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>Tertiles of total subcutaneous fat area</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>18/94 (19.1)</td>
<td>17/59 (28.8)</td>
<td>21/34 (61.8)</td>
<td>$&lt;0.001$</td>
<td>56/187 (29.9)</td>
</tr>
<tr>
<td>Tertile 2</td>
<td>10/64 (15.6)</td>
<td>16/54 (29.6)</td>
<td>45/70 (64.3)</td>
<td>$&lt;0.001$</td>
<td>71/188 (37.8)</td>
</tr>
<tr>
<td>Tertile 3</td>
<td>5/29 (17.2)</td>
<td>22/75 (29.3)</td>
<td>49/84 (58.3)</td>
<td>$&lt;0.001$</td>
<td>76/188 (40.4)</td>
</tr>
<tr>
<td>$P$ for trend</td>
<td>0.584</td>
<td>0.755</td>
<td>0.816</td>
<td>0.042</td>
<td></td>
</tr>
<tr>
<td>Tertiles of BMI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>23/122 (18.9)</td>
<td>17/53 (32.1)</td>
<td>9/12 (75.0)</td>
<td>$&lt;0.001$</td>
<td>49/187 (26.2)</td>
</tr>
<tr>
<td>Tertile 2</td>
<td>9/54 (16.7)</td>
<td>24/76 (31.6)</td>
<td>29/58 (50.0)</td>
<td>$&lt;0.001$</td>
<td>62/188 (33.0)</td>
</tr>
<tr>
<td>Tertile 3</td>
<td>1/6 (16.7)</td>
<td>14/59 (23.7)</td>
<td>77/118 (65.3)</td>
<td>$&lt;0.001$</td>
<td>92/188 (48.9)</td>
</tr>
<tr>
<td>$P$ for trend</td>
<td>0.437</td>
<td>0.304</td>
<td>0.281</td>
<td>$&lt;0.001$</td>
<td></td>
</tr>
<tr>
<td>Waist circumference</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>23/136 (16.9)</td>
<td>15/45 (33.3)</td>
<td>2/4 (50.0)</td>
<td>$&lt;0.009$</td>
<td>40/185 (21.6)</td>
</tr>
<tr>
<td>Tertile 2</td>
<td>9/44 (20.5)</td>
<td>22/83 (26.5)</td>
<td>33/62 (53.2)</td>
<td>$&lt;0.001$</td>
<td>64/189 (33.9)</td>
</tr>
<tr>
<td>Tertile 3</td>
<td>1/6 (16.7)</td>
<td>18/59 (30.5)</td>
<td>80/122 (65.6)</td>
<td>$&lt;0.001$</td>
<td>99/187 (52.9)</td>
</tr>
<tr>
<td>$P$ for trend</td>
<td>0.695</td>
<td>0.833</td>
<td>0.095</td>
<td>$&lt;0.001$</td>
<td></td>
</tr>
<tr>
<td>Fasting plasma insulin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>16/93 (17.2)</td>
<td>12/46 (26.1)</td>
<td>19/34 (55.9)</td>
<td>$&lt;0.001$</td>
<td>47/173 (27.2)</td>
</tr>
<tr>
<td>Tertile 2</td>
<td>14/76 (18.4)</td>
<td>24/73 (32.9)</td>
<td>34/57 (59.6)</td>
<td>$&lt;0.001$</td>
<td>72/206 (35.0)</td>
</tr>
<tr>
<td>Tertile 3</td>
<td>3/18 (16.7)</td>
<td>19/69 (27.5)</td>
<td>62/97 (63.9)</td>
<td>$&lt;0.001$</td>
<td>84/184 (45.7)</td>
</tr>
<tr>
<td>$P$ for trend</td>
<td>0.968</td>
<td>0.887</td>
<td>0.389</td>
<td>$&lt;0.001$</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>14/66 (21.2)</td>
<td>26/103 (25.2)</td>
<td>85/137 (62.0)</td>
<td>$&lt;0.001$</td>
<td>125/306 (40.8)</td>
</tr>
<tr>
<td>Female</td>
<td>19/121 (15.7)</td>
<td>29/85 (34.1)</td>
<td>30/51 (58.8)</td>
<td>$&lt;0.001$</td>
<td>78/257 (30.4)</td>
</tr>
<tr>
<td>$P$ for $\chi^2$ test</td>
<td>0.345</td>
<td>0.183</td>
<td>0.687</td>
<td>0.010</td>
<td></td>
</tr>
</tbody>
</table>

Data are n (%). Linear trends were evaluated by using the median value for each category in logistic regression analyses.
Models 3 to 6 of Table 4 were identical to model 2, with the exception that a different adiposity variable was used in place of TFA. Model 7 of Table 4 contained diabetes status in place of quintiles of 2hPG. In all of these models, the association between IAFA and prevalence of hypertension did not change appreciably (models 2 to 7, Table 4). Also, none of the other measures of regional or total adiposity emerged as significantly related to prevalence of hypertension (models 1 to 7, Table 4). Fasting plasma insulin, age, and 2hPG or diabetes status were also associated with a significantly increased prevalence of hypertension in all models of Table 4.

### Discussion

These cross-sectional data demonstrated that greater visceral adiposity was associated with a higher prevalence of hypertension. This finding was independent of other measures of total and regional adiposity, fasting plasma insulin, 2hPG, age, and sex. On the other hand, no other measure of regional or total adiposity was associated with prevalence of hypertension after adjusting for IAFA.

A few epidemiological studies relating CT-measured IAFA to blood pressure were inconclusive.\(^1\) Kanai et al\(^1\) showed that, among severely obese women in Japan, the ratio of the IAFA to subcutaneous fat area measured by CT was related with blood pressure independent of age and BMI. Because this study focused on severely obese women who consulted their clinic for weight reduction, their results may not apply to the general population. Johnson et al\(^1\) showed that, among subjects without diabetes and hypertension, CT-measured IAFA was not correlated with systolic and diastolic blood pressure. This study focused on relatively young men (mean age, 36 years). A 1995 publication, by our group, of cross-sectional data in Japanese Americans demonstrated that, among subjects without type 2 diabetes and not taking antihypertensive medication, the effects of visceral adiposity measured by CT on systolic or diastolic blood pressure were of statistical significance or borderline statis-

### Table 3. Prevalence of Hypertension According to IAFA and Diabetes Status

<table>
<thead>
<tr>
<th>Diabetes Status</th>
<th>NGT</th>
<th>IGT or Type 2 Diabetes</th>
<th>P for</th>
<th>(\chi^2) Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>73/306 (23.9)</td>
<td>130/257 (50.1)</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>19/135 (14.1)</td>
<td>14/52 (26.9)</td>
<td>0.039</td>
<td></td>
</tr>
<tr>
<td>Tertile 2</td>
<td>23/104 (22.1)</td>
<td>32/84 (38.1)</td>
<td>0.017</td>
<td></td>
</tr>
<tr>
<td>Tertile 3</td>
<td>31/67 (46.3)</td>
<td>84/121 (69.4)</td>
<td>0.002</td>
<td></td>
</tr>
</tbody>
</table>

P for trend:
- Tertile 1: <0.001
- Tertile 2: <0.001
- Tertile 3: <0.001

### Table 4. Multivariate Models of Prevalence of Hypertension in Relation to Baseline Values of IAFA, Other Adipose Depots, and Fasting Plasma Insulin

<table>
<thead>
<tr>
<th>Model and Variables in the Model</th>
<th>OR (95% CI)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IAFA</td>
<td>1.68 (1.20–2.37)</td>
<td>0.003</td>
</tr>
<tr>
<td>TFA</td>
<td>0.84 (0.60–1.15)</td>
<td>0.274</td>
</tr>
<tr>
<td>Fasting plasma insulin</td>
<td>1.49 (1.18–1.90)</td>
<td>0.001</td>
</tr>
<tr>
<td>Age</td>
<td>2.25 (1.74–2.92)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female sex</td>
<td>0.84 (0.48–1.46)</td>
<td>0.529</td>
</tr>
<tr>
<td>2hPG model (\log_2(2hPG)–0.0781(2hPG))^*</td>
<td>1.41 (1.10–1.81)</td>
<td>0.007</td>
</tr>
<tr>
<td>Model 2: same variables as in model 1, except quintiles of 2hPG are substituted for 2hPG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IAFA</td>
<td>1.67 (1.19–2.35)</td>
<td>0.003</td>
</tr>
<tr>
<td>Quintiles of 2hPG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quintile 1 (2.66–5.94)</td>
<td>1.00 (reference)</td>
<td></td>
</tr>
<tr>
<td>Quintile 2 (5.95–6.94)</td>
<td>1.31 (0.62–2.77)</td>
<td>0.482</td>
</tr>
<tr>
<td>Quintile 3 (6.95–8.05)</td>
<td>1.72 (0.83–3.57)</td>
<td>0.146</td>
</tr>
<tr>
<td>Quintile 4 (8.06–9.83)</td>
<td>2.21 (1.07–4.58)</td>
<td>0.033</td>
</tr>
<tr>
<td>Quintile 5 (9.84–32.86)</td>
<td>2.21 (1.04–4.72)</td>
<td>0.040</td>
</tr>
<tr>
<td>Model 3: same variables as in model 2, except abdominal subcutaneous fat area is substituted for TFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IAFA</td>
<td>1.53 (1.16–2.04)</td>
<td>0.003</td>
</tr>
<tr>
<td>Abdominal subcutaneous fat area</td>
<td>0.96 (0.74–1.23)</td>
<td>0.738</td>
</tr>
<tr>
<td>Model 4: same variables as in model 2, except total subcutaneous fat area is substituted for TFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IAFA</td>
<td>1.60 (1.20–2.15)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total subcutaneous fat area</td>
<td>0.87 (0.66–1.16)</td>
<td>0.339</td>
</tr>
<tr>
<td>Model 5: same variables as in model 2, except BMI is substituted for TFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IAFA</td>
<td>1.48 (1.09–2.00)</td>
<td>0.012</td>
</tr>
<tr>
<td>BMI</td>
<td>1.04 (0.77–1.39)</td>
<td>0.817</td>
</tr>
<tr>
<td>Model 6: same variables as in model 2, except waist circumference is substituted for TFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IAFA</td>
<td>1.44 (1.05–1.98)</td>
<td>0.024</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>1.07 (0.79–1.45)</td>
<td>0.682</td>
</tr>
<tr>
<td>Model 7: same variables as in model 1, except diabetes status is substituted for 2hPG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IAFA</td>
<td>1.70 (1.21–2.39)</td>
<td>0.001</td>
</tr>
<tr>
<td>Diabetes status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NGT</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>IGT or type 2 diabetes</td>
<td>1.65 (1.09–2.49)</td>
<td>0.018</td>
</tr>
</tbody>
</table>

ORs for continuous variables reflect a 1-SD–magnitude increase.

\(^*\)2hPG model = \(\log_2(2hPG)–\beta_1/\beta_2×(2hPG)\); \(\beta_1\) and \(\beta_2\) denote coefficients of 2hPG and \(\log_2(2hPG)\), respectively; \(\beta_2=–0.220, \beta_2=2.816\). \(\log_2\) denotes natural logarithm.
visceral fat volume. Compared with white subjects, Asians have reported ethnic group differences in the amount of visceral fat, which may be representative of the general population. Some studies have found that this measurement correlates highly with fat mass as measured by hydrodensitometry among Japanese Americans (r = 0.89 to 0.94). Visceral fat volume was also measured as a percentage of total body adipose tissue and as a proportion of total body fat mass. Despite these correlations, the association between fasting plasma insulin and visceral fat volume has been reported to have a lower visceral fat volume. Given differences in visceral fat volume by ethnicity, there may also exist ethnic differences in the association between hypertension and visceral adiposity.

In conclusion, the present study provides evidence that visceral fat is a significant correlate of hypertension among Japanese Americans. This association is independent of fasting plasma insulin, which suggests that the effect of visceral fat on prevalence of hypertension may be mediated by processes not reflected by fasting plasma insulin. The mechanism by which visceral fat is associated with a higher prevalence of hypertension remains to be determined. Further prospective research would help to establish the temporal sequence between visceral fat volume and subsequent risk of hypertension.

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