Insulin Resistance and Hypertension
The Insulin Resistance Atherosclerosis Study

Mohammed F. Saad, Marian Rewers, Joseph Selby, George Howard, Sujata Jinagouda, Salwa Fahmi, Dan Zaccaro, Richard N. Bergman, Peter J. Savage, Steven M. Haffner

Abstract—The association between insulin resistance and insulinemia and hypertension is controversial. We examined the relation between insulin resistance and hypertension in 564 non-Hispanic whites (NHW), 505 Hispanics (H), and 413 African Americans (AA) who participated in the Insulin Resistance Atherosclerosis Study (IRAS). Insulin sensitivity was measured with a frequently sampled intravenous glucose tolerance test with minimal model analysis. The prevalence of hypertension was 32.5%, 49.4%, and 32.3% in NHW, AA, and H, respectively (P<0.001). When subjects without diabetes in all ethnic groups were combined, age, male sex, race (AA), body mass index (BMI), and insulin resistance, but not fasting insulin, were significantly associated with hypertension. When each ethnic group was analyzed separately, insulin resistance was significantly associated with hypertension in NHW and H, but not AA. After excluding subjects taking antihypertensive medications, male sex, BMI, fasting glucose, and insulin resistance, but not fasting insulin, were significant determinants of blood pressure. When the 3 ethnic groups were analyzed separately, insulin resistance was significantly associated with blood pressure in H, but not NHW, or AA. Neither insulin resistance nor fasting insulin was significantly associated with hypertension or blood pressure in subjects with diabetes of the 3 ethnic groups after adjusting for age, sex, BMI, and waist. In conclusion, insulin resistance, but not insulinemia, was related to hypertension and blood pressure in subjects without diabetes, but ethnic differences in these relations appear to exist. Neither insulin resistance nor insulinemia was related to hypertension or blood pressure in patients with type 2 diabetes in the 3 ethnic groups. (Hypertension. 2004;43:1324-1331.)

Key Words: insulin resistance ■ insulin ■ hypertension ■ blood pressure ■ diabetes

In 1966, Welborn and colleagues1 described hyperinsulinemia in normoglycemic patients with hypertension and suggested that these subjects could be insulin resistant. Two decades later, Ferrannini et al2 confirmed this observation by showing that lean white hypertensive patients had lower insulin-mediated glucose disposal than normal controls. It has been hypothesized, therefore, that insulin and/or insulin resistance could contribute to the pathogenesis of hypertension.3 Insulin was shown to stimulate the sympathetic nervous system, increase renal sodium retention, modulate cation transport, and induce vascular smooth muscle hypertrophy (reviewed in Reference 4). It is plausible that insulin resistance through the concomitant compensatory hyperinsulinemia could contribute to the pathogenesis of hypertension by one or more of these mechanisms. Nonetheless, acute insulin infusion was found to have a vasodilator hypertensive rather than a hypertensive effect.5,6 To reconcile these discrepant observations, it was proposed that insulin resistance might lead to hypertension because of diminished insulin-induced vasodilation and an imbalance between its pressor and depressor effects.7

Although a large number of studies examined the relation between insulinemia and hypertension and/or blood pressure (BP), the results have been inconsistent with some reporting strong and others weak, or no association.8–19 A smaller number of studies measured insulin resistance directly, mostly in small groups of subjects with still conflicting results.20–27 Herein, we describe the relation between insulin resistance and hypertension in a large triethnic population that participated in the Insulin Resistance Atherosclerosis Study (IRAS). Insulin sensitivity was measured directly with the insulin-modified frequently sampled intravenous glucose tolerance test (FSIGT) with minimal model analysis (MINMOD) in 1482 subjects.

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Subjects and Methods

Subjects
Details of subject recruitment and study protocol were described.28 In brief, the IRAS included 1625 subjects who were studied at 4 clinical centers at Oakland and Los Angeles, Calif; San Luis, Co; and San Antonio, Tex. Clinical centers in California enrolled non-Hispanic whites (NHW) and African-Americans (AA) from Kaiser Permanente, a nonprofit health maintenance organization. Those in San Antonio, Tex, and San Luis Valley, Co, studied NHW and Hispanics (H) recruited from two ongoing population-based studies (San Antonio Heart Study and the San Luis Valley Diabetes Study), respectively.

The study aimed to include equal numbers of subjects in different categories of glucose tolerance (normal glucose tolerance [NGT], impaired glucose tolerance [IGT], and type 2 diabetes) according to the 1985 World Health Organization criteria. Therefore, subjects with IGT and type 2 diabetes were oversampled. Insulin-treated patients with diabetes and those with a fasting glucose >16.7 mmol/L were excluded. The final IRAS cohort included 721 men and 904 women aged 40 to 69 years; 614 NHW, 548 H, and 464 AA. There were 720 subjects (44%) with NGT, 369 (23%) with IGT, and 537 (33%) with type 2 diabetes. The current analysis includes 1482 subjects in whom insulin sensitivity was measured.

The IRAS examination required 2 visits, ~1 week apart. Participants were asked to fast for 12 hours before each visit and to abstain from heavy exercise and alcohol for 24 hours and from smoking the morning of each visit. An oral glucose tolerance test (OGTT) was performed with a 75-g glucose load in the first visit. An insulin-modified FSIGT was done in the second visit for the determination of the insulin sensitivity index (SI) with the computer program MINMOD.79 An injection of insulin was used to ensure adequate plasma insulin levels for the accurate computation of insulin resistance across a broad range of glucose tolerance. 30–32

BP was measured during each visit with a mercury sphygmomanometer before the OGTT and the FSIGT. A standardized protocol in which the appropriate cuff size was used depending on the arm circumference was followed. Three measurements were taken 5 minutes apart in the right arm in the sitting position after 5 minutes of rest. The average of the second and third measurements was taken as the participant’s BP during each visit. The average BP of the two visits is used in this analysis. Mean BP (MBP) was calculated as 0.33 × systolic BP (SBP) + 0.66 × diastolic BP (DBP). Hypertension was defined as SBP ≥140 mm Hg or DBP ≥90 mm Hg or as taking antihypertensive medications. Body mass index (weight [kg]/height [m]²) is used as a measure of overall adiposity and minimum waist circumference as an index of body fat distribution.

Statistical Analysis
Data are expressed as means ± SE or as means with 95% confidence interval (CI). Insulin levels and SI values were log-transformed to normalize the distribution. The log of SI was used in the analysis because of the existence of zero SI values. Statistical analyses were performed with programs of SPSS, Inc. One-way ANOVA and χ² tests were used to compare continuous and categorical variables, respectively, among the 3 ethnic groups. Two-way ANOVA was used to evaluate the effects of ethnicity and hypertension on different variables. The sample size provided more than 90% to detect differences in fasting and 2-hour insulin concentrations and SI in the subjects without diabetes by race and hypertension status at a significance level of 0.05. For the subjects with diabetes, the sample size provided <60% power to detect significant differences in these variables. The Tukey method was used for multiple comparisons.

Linear regression analysis was used to evaluate the relation between different variables and hypertension. Linear regression and/or Pearson product moment correlations were used to evaluate the relation between BP and different variables. The sample size of subjects without diabetes provided 80% power to detect a correlation coefficient of 0.11 in the 3 ethnic groups combined (0.16 in whites, 0.22 in AA, and 0.18 in H) at a significance level of 0.05. The sample size of subjects with diabetes provided 80% power to detect a correlation coefficient of 0.18 in the 3 ethnic groups combined (0.29 in whites, 0.37 in AA, and 0.28 in H) at a significance level of 0.05.

Results

Glucose Intolerance, Obesity, and Hypertension
The prevalence of hypertension was 32.5% in NHW, 49.4% in AA, and 32.3% in H (P < 0.001). Hypertension was more common in subjects with type 2 diabetes, followed by IGT, and then NGT in each ethnic group (Figure 1). Controlling for age and sex, both overall and central adiposity were significantly associated with hypertension in individuals without diabetes in the 3 ethnic groups. In subjects with diabetes, hypertension was significantly associated with BMI in NHW (P = 0.01) and H (P = 0.04) but not in AA, after adjusting for age and sex. The prevalence of hypertension tended to increase with the waist circumference in subjects with diabetes controlling for age and sex, but the association was statistically significant only in H (P = 0.01). Logistic regression analysis of subjects with and without diabetes combined showed that race, age, BMI (or waist circumference), and glycemia were significantly associated with hypertension.

Adjusting for age, sex, BMI, and glycemia, the prevalence of hypertension was higher in AA than in NHW (odds ratio: 2.35; 95% CI: 1.67 to 3.30) and H (odds ratio 2.96; CI: 1.82 to 4.81), but there was no difference between H and NHW. An increase in 2-hour plasma glucose of 1 mmol/L was associated with a 5% increase in the likelihood of having hypertension (CI 2.7 to 7.1%), controlling for the other variables.

Insulinemia, Insulin Resistance, and Hypertension in Subjects Without Diabetes
Hypertensive subjects without diabetes were older and had significantly higher BMI, waist circumference, and fasting and 2-hour post-load plasma glucose and insulin concentrations and lower SI than their normotensive counterparts (Table 1). After adjusting for age, sex, BMI, and waist, differences in fasting insulinemia between hypertensive and normotensive subjects remained significant only in H and those in SI in NHW and H. The prevalence of hypertension increased with fasting insulinemia. This association was significant in NHW and Hispanics (P < 0.01 for linear trend), but not in AA (P = 0.16). After adjusting for age, sex, BMI, and waist, insulinemia was significantly associated with hypertension only in H (P = 0.001). Similarly, the prevalence
of hypertension was higher at lower $S_I$ values in all ethnic groups ($P<0.05$ in each), but this association remained significant only in H ($P=0.02$) after controlling for age, sex, BMI, and waist (Figure 2).

When subjects without diabetes of all ethnic groups were combined, logistic regression analysis showed that age, race (AA), BMI (or waist), and $S_I$ were significantly associated with hypertension (Table 2). Fasting insulin was significantly associated with hypertension only when $S_I$ was not included in the logistic regression models. The interaction term between $S_I$ and ethnicity was not statistically significant ($P=0.94$), but when each ethnic group was analyzed separately, insulin resistance was significantly associated with hypertension in NHW and H, but not AA (Figure 3).

![Figure 2](image1.png)

**Figure 2.** The prevalence of hypertension by quartiles of fasting insulin (upper panels) and $S_I$ (lower panels) before (left) and after (right) adjusting for age, sex, BMI, and minimum waist in subjects without diabetes in the 3 ethnic groups. The symbols correspond to the median value in each quartile.
TABLE 2. Logistic Regression Analysis Showing Variables Associated with Hypertension in Subjects Without Diabetes (n=1003)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (increase of 10 y)</td>
<td>1.98 (1.64–2.38)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Sex (M=1, F=2)</td>
<td>1.10 (0.81–1.49)</td>
<td>0.534</td>
</tr>
<tr>
<td>Race</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>AA vs NHW</td>
<td>2.00 (1.28–3.13)</td>
<td>0.002</td>
</tr>
<tr>
<td>AA vs H</td>
<td>2.49 (1.35–4.06)</td>
<td>0.004</td>
</tr>
<tr>
<td>BMI, increase of 1 kg/m²*</td>
<td>1.06 (1.03–1.09)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2-h glucose (increase of 1 mmol)</td>
<td>1.08 (0.99–1.18)</td>
<td>0.094</td>
</tr>
<tr>
<td>S₁ (2-fold decrease)†</td>
<td>1.64 (1.28–2.10)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Abbreviations are defined in the text.

†Could be replaced with minimum waist without a change in the strength of the model; a 1-cm increase in minimum waist was associated with an odds ratio of 1.03 (CI:1.01–1.09; P<0.001).

Discussion

The association between insulin resistance and hypertension is controversial. Whereas some studies reported that insulin resistance and/or hyperinsulinemia were strongly related to hypertension, others show only a weak or no association. Our data add some insights and show that insulin resistance is at most a modest determinant of hypertension and BP. This effect was not mediated by insulinemia, which was significantly related to hypertension only when S₁ was not included in the regression models. In addition, neither insulin resistance nor insulinemia was significantly related to hypertension or BP in patients with diabetes after controlling for age, sex, and obesity. Our data show that an increase in insulin resistance from the lowest to the highest quartile of the distribution in NHW and H was associated with a 70% higher likelihood of having hypertension, an effect that was similar to a 10-year increase in age. It is to be noted, however, that several antihypertensive medications could worsen insulin resistance and that we could not estimate the effect of drugs because of the cross-sectional nature of the study. Furthermore, the number of hypertensive subjects (≈7%) not receiving medications was too small to allow analyzing their data separately. Nonetheless, S₁ was weakly associated with BP in Hispanic and NHW subjects without diabetes not receiving antihypertensive medications, explaining 2% to 4% of its variance. Figure 5 shows that a decrease in S₁ of 2 U (equivalent to change from 20 to 80 percentile of the population) was associated with a 1.2-mm Hg increase in MBP in NHW and H.

The relation between insulinemia and/or insulin resistance and BP appears to vary among ethnic groups. Nearly all studies showing a strong association between insulin and BP were conducted among NHW. Studies in African Americans, Hispanics, Nauruans, Pima Indians, and Asian Indians showed a weak or no association. Even among NHW, the association between insulinemia and BP was inconsistent with some studies reporting a weak or insignificant relationship. Similarly, when insulin resistance was directly measured, the findings were discrepant. Pooled European data showed a weak but significant association between insulin resistance and BP. Saad et al found insulin resistance and BP to be related in NHW, but not in AA or Pima Indians. Falkner et al described insulin resistance in young hypertensive AA men. Mattiasson et al showed that insulin resistance and BP were related only in postmeno-
pausal NHW women with IGT, but not in those with NGT. Our data unfortunately add to the confusion. We found S_I to be related to hypertension and BP in H and NHW without diabetes, but not in AA.

The mechanism through which insulin resistance is associated with hypertension and BP is not known. It is thought that insulin resistance could cause hypertension through compensatory hyperinsulinemia. Insulin has been shown to

### Table 3. Correlation Between Blood Pressure and Insulinemia and Insulin Resistance in Subjects Without Diabetes Not Receiving Antihypertensive Medications

| Population | Fasting Insulin | | | | | S_I | | | | |
|------------|----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|            | SBP            | DBP             | MBP             | SBP             | DBP             | MBP             | SBP             | DBP             | MBP             |
| Simple Correlation | | | | | | | | | | |
| All        | 0.18 (<0.001)  | 0.19 (<0.001)  | 0.21 (<0.001)  | -0.21 (<0.001)  | -0.17 (<0.001)  | -0.21 (<0.001)  | | | |
| NHW        | 0.19 (0.001)   | 0.23 (<0.001)  | 0.24 (<0.001)  | -0.19 (0.001)   | -0.17 (0.003)   | -0.20 (<0.001)  | | | |
| AA         | -0.02 (0.771)  | -0.01 (0.944)  | -0.02 (0.848)  | -0.08 (0.298)   | -0.04 (0.633)   | -0.07 (0.412)   | | | |
| H          | 0.26 (<0.001)  | 0.23 (<0.001)  | 0.27 (<0.001)  | -0.29 (<0.001)  | -0.21 (0.001)   | -0.28 (<0.001)  | | | |
| Partial Correlation (controlling for age, sex, BMI, and minimum waist) | | | | | | | | | |
| All        | 0.10 (0.007)   | 0.12 (0.002)   | 0.12 (0.001)   | -0.10 (0.009)   | -0.10 (0.007)   | -0.11 (0.003)   | | | |
| NHW        | 0.13 (0.027)   | 0.15 (0.012)   | 0.15 (0.009)   | -0.10 (0.093)   | -0.09 (0.125)   | -0.10 (0.077)   | | | |
| AA         | -0.10 (0.235)  | -0.05 (0.312)  | -0.08 (0.324)  | 0.01 (0.895)    | -0.01 (0.940)   | 0.001 (0.983)   | | | |
| H          | 0.16 (0.010)   | 0.14 (0.022)   | 0.16 (0.008)   | -0.15 (0.018)   | -0.13 (0.035)   | -0.15 (0.015)   | | | |

The values of the correlation coefficient (r) are given with P values in parenthesis. Abbreviations are defined in the text.

### Figure 4

The relation between each of fasting insulinemia (upper panels) and S_I (lower panels) and MBP in the 3 ethnic groups after adjusting for age, sex, BMI, and minimum waist. The regression lines between fasting insulin and MBP in the 3 ethnic groups were not different in the slope (P=0.0792) or the intercept (P=0.642). Likewise, the regression lines between S_I and MBP in the 3 ethnic groups were not different in the slope (P=0.435) or the intercept (P=0.675).
stimulate the sympathetic nervous system, increase renal sodium retention, modulate cation transport, and induce hypertrophy of vascular smooth muscle (reviewed in 4). Our data do not support this notion, however, because insulin was not independently related to hypertension or BP. In addition, acute insulin infusion was found to have a vasodilator hypotensive and not a hypertensive effect.5,6 It has been known for years that insulin could lower BP substantially in the absence of any other treatment.6 It was later shown that acute insulin infusion was found to have a vasodilator effect of insulin that could lead to a rise in BP. In addition, insulin resistance has been associated with impaired endothelium-dependent vasodilatation,37 which could contribute to increased BP.

Alternatively, the association between insulin resistance and hypertension may not be causal. Instead, they may be linked indirectly through mechanisms of an inherited or acquired nature. A possible link is through the sympathetic nervous system. Enhanced adrenergic tone may lead to increased insulin resistance on the one hand and a rise in BP on the other. Ethnic or racial differences in sympathetic nervous system activity might explain the differences in the relation of insulin resistance to BP. Increased levels of inflammatory markers such as tumor necrosis factor-α could contribute to both insulin resistance and endothelial dysfunction37 and underlie the link between insulin resistance and hypertension. A further possibility is that a cellular or structural defect, genetic or acquired, may constitute the link between insulin resistance and BP. Reduced activity of sodium-potassium ATPase, decreased intracellular magnesium, and increased sodium-lithium countertransport have been proposed as possible links. Racial differences in ion regulation have been described and could account for the observed variation in the relation of insulin resistance to BP.38

Table 5. Characteristics of Hypertensive and Nonhypertensive Subjects With Diabetes

| Variable         | NHW | AA | H | P
<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=88</td>
<td>n=52</td>
<td>n=101</td>
<td>n=96</td>
</tr>
<tr>
<td>Age, y*</td>
<td>56±0.9</td>
<td>58±0.8</td>
<td>58±1.2</td>
<td>58±0.8</td>
</tr>
<tr>
<td>Sex, % F</td>
<td>48.9</td>
<td>50.0</td>
<td>58.1</td>
<td>58.4</td>
</tr>
<tr>
<td>BMI, kg/m²*</td>
<td>30.10±0.62</td>
<td>32.15±0.73</td>
<td>31.99±0.82</td>
<td>32.46±0.57</td>
</tr>
<tr>
<td>Minimum waist, cm*</td>
<td>99.0±1.5</td>
<td>101.1±1.6</td>
<td>98.6±1.5</td>
<td>99.0±1.2</td>
</tr>
<tr>
<td>SBP, mm Hg*</td>
<td>118±1.1</td>
<td>134±1.7</td>
<td>122±1.3</td>
<td>137±1.4</td>
</tr>
<tr>
<td>DBP, mm Hg*</td>
<td>74±0.7</td>
<td>81±1.0</td>
<td>76±0.9</td>
<td>83±1.0</td>
</tr>
<tr>
<td>Fasting glucose, mmol/L*</td>
<td>9.72±0.34</td>
<td>9.20±0.34</td>
<td>10.28±0.52</td>
<td>9.15±0.29</td>
</tr>
<tr>
<td>2-hour glucose, mmol/L*</td>
<td>16.95±0.44</td>
<td>17.21±0.53</td>
<td>17.38±0.71</td>
<td>16.75±0.44</td>
</tr>
<tr>
<td>Fasting insulin, pmol/L†</td>
<td>106 (92–122)</td>
<td>117 (98–140)</td>
<td>107 (91–126)</td>
<td>116 (102–131)</td>
</tr>
<tr>
<td>2-hour insulin, pmol/L†</td>
<td>361 (309–423)</td>
<td>403 (333–488)</td>
<td>375 (302–467)</td>
<td>444 (374–528)</td>
</tr>
<tr>
<td>S₁, 10⁻⁶ (min⁻¹ · µu⁻¹ · mL⁻¹)</td>
<td>0.56 (0.41–0.72)</td>
<td>0.38 (0.27–0.50)</td>
<td>0.49 (0.37–0.61)</td>
<td>0.38 (0.28–0.48)</td>
</tr>
</tbody>
</table>

Abbreviations are defined in the text.
*Mean±SE is shown.
†Mean and 95% CI are shown.
TABLE 6. Logistic Regression Analysis Showing Variables Associated With Hypertension in Subjects With Diabetes (n=479)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (increase of 10 y)</td>
<td>1.68</td>
<td>(1.32–2.16)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Sex (M=1, F=2)</td>
<td>0.82</td>
<td>(0.65–1.22)</td>
<td>0.33</td>
</tr>
<tr>
<td>Race</td>
<td>0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA vs NHW</td>
<td>2.37</td>
<td>(1.39–4.04)</td>
<td>0.001</td>
</tr>
<tr>
<td>AA vs H</td>
<td>3.25</td>
<td>(1.42–7.46)</td>
<td>0.005</td>
</tr>
<tr>
<td>BMI (increase of 1 kg/m²)*</td>
<td>1.05</td>
<td>(1.02–1.09)</td>
<td>0.096</td>
</tr>
<tr>
<td>Fasting glucose, mmol/L</td>
<td>0.95</td>
<td>(0.90–1.01)</td>
<td>0.131</td>
</tr>
<tr>
<td>S, (2-fold decrease)</td>
<td>1.38</td>
<td>(0.48–1.94)</td>
<td>0.275</td>
</tr>
</tbody>
</table>

Abbreviations are defined in the text.

*Minimum waist was not a significant predictor of hypertension in subjects with diabetes.

Our data show that neither insulin resistance nor insulinemia was related to hypertension in patients with diabetes of any ethnic group. This is in agreement with those of Bonora et al. who found no difference in insulin sensitivity between normotensive and hypertensive patients with diabetes. Laakso et al. reported similar data in obese subjects with diabetes, but found lean hypertensive patients with diabetes more insulin resistant than normotensive counterparts. This latter study included only, however, a small number of lean subjects with diabetes (11 hypertensive and 6 normotensive). Thus, insulin resistance does not appear to be a feature of hypertension in subjects with type 2 diabetes.

Perspectives

The current study shows that insulin resistance, but not insulinemia, was weakly but significantly related to hypertension and BP in subjects without diabetes. This weak association argues against a major role for insulin resistance in the regulation of blood pressure or pathogenesis of hypertension. This is especially true in individuals with type 2 diabetes in whom neither insulin resistance nor insulinemia were related to hypertension or BP.

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References


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