Left Ventricular Concentric Remodeling Rather Than Left Ventricular Hypertrophy Is Related to the Insulin Resistance Syndrome in Elderly Men

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Background—Associations between left ventricular (LV) geometry and the insulin resistance syndrome have been found, mostly in small studies of middle-aged hypertensives. The purpose of this study was to elucidate these associations through the use of a large sample of elderly men.

Methods and Results—We investigated 475 men (157 hypertensives) 71 years of age who were attending a population-based health survey in Uppsala County with echocardiography, oral glucose tolerance test (OGTT), hyperinsulinemic euglycemic clamp, and lipid and 24-hour ambulatory blood pressure monitoring. LV relative wall thickness was significantly related to clamp insulin sensitivity index ($r = 0.14$), fasting insulin, 32-33 split proinsulin, triglycerides, nonesterified fatty acids, OGTT glucose and insulin levels, waist-to-hip ratio, body mass index, 24-hour blood pressure, and heart rate ($r = 0.10$ to $0.22$). Only 24-hour systolic pressure ($r = 0.15$), OGTT 2-hour insulin ($r = -0.10$), and heart rate ($r = -0.14$) were significantly related to LV mass index. Comparing subjects with various LV geometry (normal, concentric remodeling and concentric and eccentric hypertrophy) showed that 24-hour heart rate, OGTT glucose and insulin levels, waist-to-hip ratio, and body mass index were significantly higher ($P < 0.001$ to $0.05$) and clamp insulin sensitivity index was significantly lower ($P < 0.01$) in the concentric remodeling geometry group than in the normal LV geometry group. The 24-hour blood pressure was significantly higher in the concentric hypertrophy group than in the normal LV geometry group ($P < 0.001$).

Conclusions—Several components of the insulin resistance syndrome were related to thick LV walls and concentric remodeling but less to LV hypertrophy in this population-based sample of elderly men. (Circulation. 2000;101:2595-2600.)

Key Words: hypertrophy • insulin • glucose • risk factors

Echoangiographically determined left ventricular hypertrophy (LVH) is a condition associated with a marked increase in cardiovascular morbidity and mortality. LVH has also been proved to be an independent risk factor for cerebrovascular stroke and death due to any cause. Thick left ventricular walls seem particularly unfavorable, because concentric LVH, in which relative wall thickness (RWT) is increased, has been associated with a poorer prognosis than eccentric LVH, in which RWT is normal. In patients without LVH, an increased left ventricular RWT (concentric remodeling) has been related to increased cardiovascular morbidity and mortality.

Hypertension is traditionally regarded as the most important factor in the development of LVH, but the variation in 24-hour blood pressure explains only 25% to 30% of the variation in left ventricular mass. Thus, other factors must be of importance in the development of LVH. It is well known that important cardiovascular risk factors, such as hypertension, glucose intolerance, hyperinsulinemia, dyslipidemia, and obesity, often cluster in the same individuals. Therefore, the existence of a syndrome involving these disorders has been proposed in which insulin resistance has been suggested to be of particular importance. An association between this insulin resistance syndrome and LVH has recently been found. In 2 of these studies, insulin resistance determined by hyperinsulinemic euglycemic clamp was more closely related to thick left ventricular walls than to LVH. In addition, impaired glucose tolerance at an oral glucose tolerance test (OGTT) has been related to increased left ventricular RWT.

Because most of the above-mentioned studies have comprised a limited number of middle-aged, predominantly hypertensive subjects and have shown somewhat inconsistent results, the purpose of this study was to further investigate the...
relationships between left ventricular geometry and the insulin resistance syndrome through the use of a large sample of elderly men attending a population-based health survey.

Methods

Materials

In 1970–1973, all men born in 1920–1924 and residing in Uppsala County were invited to a health survey aimed at identifying risk factors for cardiovascular disease. Of the invited subjects, 2322 (82%) participated. The cohort was reinvested 20 years later with echocardiographic and Doppler examinations, ambulatory blood pressure monitoring, hyperinsulinemic euglycemic clamp, OGTT, and lipid determinations, in addition to the previous study protocol. All investigations in the same subject were performed within 1 month. The population of the present study consisted of 475 of the first 583 consecutive men (of 1221) in the latter investigation who had technically satisfactory echocardiographic examinations. The men included in the study did not differ significantly from the excluded men in any of the investigated variables. Fifty-four subjects had been hospitalized as a result of ischemic heart disease (ICD-9 codes 410 to 414) before the investigation. One hundred fifty-seven subjects were hypertensive, defined as an office diastolic blood pressure (DBP) \( \geq 95 \) mm Hg and/or treatment for hypertension. One hundred sixty-seven subjects were regularly using antihypertensive medication; 6 were using \( \beta \)-receptor blockers; 60, calcium antagonists; 24, ACE inhibitors; 57, diuretics; and 88, \( \beta \)-receptor blockers, as monotherapy or in combination. The estimated duration of antihypertensive treatment in the hypertensive subjects was \( 8.5 \pm 7.8 \) years. Only 17 subjects had significant echocardiographic valvular disease (aortic or mitral stenosis or regurgitation grade 3 or 4). All analyses were made on a subset (\( n = 458 \)) without valvular disease, controlling for ischemic heart disease (ICD-9 codes 410 to 414). A reproducibility study was made of all investigations in 22 subjects \( \sim 1 \) month after the original investigations. The intradividual coefficients of variation (CVs) presented are from this reproducibility study. All subjects gave written informed consent, and the study was approved by the Ethics Committee of Uppsala University. All procedures were in accordance with department guidelines.

Echocardiography

A comprehensive 2-dimensional and Doppler echocardiography was performed as described previously.\(^6\) Left ventricular mass was determined from M-mode measurements by use of the cube formula according to the recommendations of the American Society of Echocardiography (ASE). This formula can easily be transformed to reflect anatomic measurements: left ventricular mass = \( 0.80 \times \text{ASE mass} + 0.6 \).\(^7\) Left ventricular mass was divided with body surface area to obtain left ventricular index (LVMI). LVH was defined as an LVMI \( > 50 \) g/m\(^2\), according to data from the Framingham Heart Study.\(^8\) A partition value of 0.44 was used for RWT = (IVS + PW)/LVEDD, where IVS is interventricular septum, PW is posterior wall, and LVEDD is left ventricular end-diastolic diameter.\(^7\) Thus, left ventricular geometry was considered normal if RWT was \( < 0.44 \) and LVMI was \( < 150 \text{g/m}^2 \). A normal LVMI with increased RWT was denoted concentric remodeling,\(^3\) and a hypertrophic left ventricle was denoted eccentric if the RWT was normal and concentric if the RWT was increased. Stroke volume was calculated from Doppler measurements of left ventricular outflow tract diameter (LVOT) and the flow velocity integral (FVI) as \( \pi \times \text{LVOT}^2/4 \times \text{FVI} \) and was divided by body surface area to obtain stroke index.\(^8\) Examinations and readings of the images were done by an experienced physician (Dr André) who was unaware of other data of the subjects. The CVs were as follows: for IVS, 8.8%; PW, 6.7%; LVEDD, 3.5%; RWT, 6.9%; and LVMI, 12.5%.

Ambulatory Blood Pressure Monitoring

The ambulatory blood pressure measuring device Accutracker II (Suntech Medical Instruments) was attached to the subject’s non-dominant arm. Systolic blood pressure (SBP), DBP, and heart rate were measured over 24 hours, every 30 minutes during daytime (6 AM to 11 PM) and every hour during nighttime. Data were edited to a limited extent, omitting all readings of 0, all heart rate readings <30 bpm, DBP readings >170 mm Hg, SBP readings >270 and <80 mm Hg, and all readings for which the difference between SBP and DBP was <10 mm Hg. The CV for 24-hour mean arterial blood pressure \( \text{DBP} + (\text{SBP} - \text{DBP})/3 \) was 5.5%.

Oral Glucose Tolerance Test

Blood samples for determining fasting concentrations were drawn in the morning after an overnight fast. An OGTT was performed by measuring the concentrations of plasma glucose and “immunoreactive insulin” immediately before and 30, 60, 90, and 120 minutes after 75 g anhydrous dextrose was ingested. In the present study, fasting, 2-hour levels and the incremental areas under the curves (AUC) of glucose and immunoreactive insulin were analyzed. Glucose was measured by the glucose dehydrogenase method (Gluc-DH, Merck), and immunoreactive insulin was analyzed by use of an enzymatic-immunological assay (Enzymun, Boehringer Mannheim) performed in an ES300 automatic analyzer (Boehringer Mannheim). Fasting specific insulin and 32-33 split and intact proinsulin concentrations were measured with a specific 2-site immunoradiometric assay technique\(^19\) in Cambridge, UK, on a Nuclear Enterprises 1600 gamma counter of \( 1^{2} \). The CV for fasting plasma glucose was 5.8%, and for immunoreactive insulin, it was 15.4%.

Hyperinsulinemic Euglycemic Clamp

Insulin sensitivity was determined with the hyperinsulinemic euglycemic clamp, performed according to the method of DeFronzo et al\(^20\) with a slight modification: insulin was infused at a constant rate of 56 instead of 40 mU/(min \( \times \) m\(^2\)). Insulin sensitivity index was calculated by dividing glucose disposal (milligrams of glucose infused divided by minutes times kilograms of body weight) by the mean plasma insulin concentration times 100 (mU/L) during the last 60 minutes of the 2-hour clamp. The CV for insulin sensitivity index was 13.9%.

Lipid and Lipoprotein Measurements

HDL was separated by precipitation with magnesium chloride/phosphotungstate. Cholesterol and triglyceride concentrations in serum and HDL were assayed by enzymatic techniques (Instrumentation Laboratories) in a Monarch 2000 centrifugal analyzer. LDL cholesterol was calculated with Friedewald’s formula: LDL = serum cholesterol − HDL − (0.45 \times serum triglycerides). Serum nonesterified fatty acids (NEFAs) were measured by an enzymatic colorimetric method (Wako Chemical GmbH) applied for use in the Monarch 2000. CVs were as follows: for serum total cholesterol, 5.7%; HDL cholesterol, 11.1%; LDL cholesterol, 6.6%; serum triglycerides, 14.8%; and NEFA, 24.2%.

Statistical Analyses

Variables with a skewed distribution (fasting plasma glucose, immunoreactive insulin, specific insulin, proinsulin, 32-33 split proinsulin, serum triglycerides and NEFA, 2-hour glucose and immunoreactive insulin levels, and the AUC of immunoreactive insulin at the OGTT) were logarithmically transformed to achieve normal distribution, and these transformed variables were used in all analyses. Factorial ANOVA was used to calculate differences in means between subgroups. All ANOVAs were adjusted for use of antihypertensive medication. Missing data were evenly distributed over the geometric groups. Multiple regression analysis, correlation coefficients, and partial correlation coefficients were used to evaluate relationships between pairs of variables, adjusted for possible confounders. Squared variables and interaction terms between independent variables were tested in all models. Scatter plots were visually examined for other nonlinear associations. Relationships to LVMI but not to RWT were adjusted for use of antihypertensive medication, because subjects using antihypertensive medication had signif-
Numerically higher LVMIs but did not differ in RWT. Two-tailed significance values were given, with $P<0.05$ regarded as significant.

**Results**

**Relationships Between Components of the Insulin Resistance Syndrome and Left Ventricular Geometric Parameters**

Several components of the insulin resistance syndrome were significantly and directly related to RWT, such as 24-hour SBP and DBP, 24-hour heart rate, 2-hour glucose level and the AUC of glucose at the OGTT, fasting specific insulin and 32-33 split proinsulin, waist-to-hip ratio, body mass index, serum triglycerides, and NEFA, whereas clamp insulin sensitivity index was inversely related to RWT (Table 1). On the other hand, of the measured variables, only 24-hour SBP (directly), 24-hour heart rate, and 2-hour immunoreactive insulin level at the OGTT (inversely) were significantly related to LVMI (Table 1). Neither RWT nor LVMI was significantly related to serum total cholesterol, LDL cholesterol, HDL cholesterol, fasting plasma glucose, immunoreactive insulin, proinsulin, 2-hour immunoreactive insulin level, or the AUC of immunoreactive insulin at the OGTT.

In a subsample without valvular disease, controlling for ischemic heart disease, correlations between metabolic variables and RWT or LVMI were similar, but OGTT 2-hour immunoreactive insulin was also related to RWT ($r=0.10$, $P=0.03$), 24-hour DBP was related to LVMI ($r=0.11$, $P=0.02$), and the relationship between OGTT 2-hour immunoreactive insulin and LVMI lost significance ($P=0.052$). There was no relationship between RWT and LVMI ($r=-0.05$, $P=0.2$). Interaction terms or squared variables were not found to be significant in any model.

**Metabolic and Other Characteristics of Subjects With Various Left Ventricular Geometries**

The levels of several components of the insulin resistance syndrome—24-hour SBP and DBP, 24-hour heart rate, clamp insulin sensitivity index, 2-hour glucose level and the AUC of glucose at the OGTT, waist-to-hip ratio, and body mass index—differed significantly between the 4 left ventricular geometric groups (Table 2). There was no significant difference between the geometric groups regarding the levels of serum triglycerides, NEFA, total cholesterol, LDL cholesterol, HDL cholesterol, fasting plasma glucose, fasting immunoreactive insulin, specific insulin, proinsulin, 32-33 split proinsulin, 2-hour immunoreactive insulin level, or the AUC of immunoreactive insulin at the OGTT.

The values of 24-hour heart rate, waist-to-hip ratio, 2-hour glucose level, and the AUC of glucose at the OGTT were significantly higher and clamp insulin sensitivity index was significantly lower in the concentric remodeling geometry group compared with the group with normal left ventricular geometry. The 24-hour SBP and DBP were significantly higher in the concentric LVH group compared with the group with normal left ventricular geometry.

The difference in 24-hour heart rate between the groups remained significant ($P=0.004$) when adjusting simultaneously for the possible confounders ischemic heart disease, stroke index (mean, 38±8 mL/m²), and use of β-receptor blockers and any other antihypertensive medication. The 24-hour heart rate was still significantly higher in the concentric remodeling geometry group ($P=0.05$) and lower in the eccentric LVH group ($P=0.01$) than in the normal geometry group.

In a subsample without valvular disease, controlling for ischemic heart disease, differences between groups were similar except that waist-to-hip ratio also was significantly higher in the eccentric LVH group than in the normal group ($P=0.03$), whereas differences between groups regarding body mass index and OGTT AUC of glucose lost significance (both $P=0.07$).

**Discussion**

In the present large, population-based sample of elderly men of the same age, several components of the insulin resistance syndrome were found to be related to an increased left ventricular RWT and left ventricular concentric remodeling but less to LVH. These findings were somewhat unexpected, because some other studies have shown LVH to be related to the insulin resistance syndrome. However, other studies have shown insulin resistance determined by hyperinsulinemic euglycemic clamp or impaired glucose tolerance by an OGTT to be related to thick left ventricular walls rather than to LVH in accordance with the present study, although some studies have used the sum of posterior wall...
TABLE 2. Metabolic and Other Characteristics of Subjects With Various Left Ventricular Geometries

<table>
<thead>
<tr>
<th></th>
<th>Normal (n=262)</th>
<th>Concentric Remodeling (n=79)</th>
<th>Concentric LVH (n=40)</th>
<th>Eccentric LVH (n=94)</th>
<th>P for No Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMI, g/m²</td>
<td>123±16</td>
<td>120±19</td>
<td>169±14§</td>
<td>173±24§</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RWT</td>
<td>0.38±0.04</td>
<td>0.49±0.04§</td>
<td>0.50±0.04§</td>
<td>0.37±0.05</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>24-h SBP, mm Hg</td>
<td>135±16</td>
<td>138±15</td>
<td>147±17§</td>
<td>139±18</td>
<td>0.002</td>
</tr>
<tr>
<td>24-h DBP, mm Hg</td>
<td>76±8</td>
<td>77±7</td>
<td>82±8§</td>
<td>78±10</td>
<td>0.003</td>
</tr>
<tr>
<td>24-h heart rate, bpm</td>
<td>70±10</td>
<td>73±11*</td>
<td>71±12</td>
<td>66±10†</td>
<td>0.0006</td>
</tr>
<tr>
<td>Insulin sensitivity index, mg · min⁻¹ · kg⁻¹/(100 mU/L)</td>
<td>5.4±2.4</td>
<td>4.6±2.0†</td>
<td>4.7±2.3</td>
<td>5.3±2.7</td>
<td>0.03</td>
</tr>
<tr>
<td>Fasting plasma glucose, mmol/L</td>
<td>5.7±1.2</td>
<td>5.9±1.6</td>
<td>6.0±1.9</td>
<td>5.7±1.3</td>
<td>0.27</td>
</tr>
<tr>
<td>OGTT 2-h glucose, mmol/L</td>
<td>7.8±3.5</td>
<td>9.2±4.7†</td>
<td>8.7±4.8</td>
<td>8.0±3.8</td>
<td>0.03</td>
</tr>
<tr>
<td>OGTT AUC glucose</td>
<td>59±39</td>
<td>73±45†</td>
<td>69±42</td>
<td>62±39</td>
<td>0.04</td>
</tr>
<tr>
<td>Fasting immunoreactive insulin, mU/L</td>
<td>12±7</td>
<td>13±8</td>
<td>14±10</td>
<td>12±7</td>
<td>0.47</td>
</tr>
<tr>
<td>OGTT AUC immunoreactive insulin, mU/L</td>
<td>64±48</td>
<td>68±40</td>
<td>64±42</td>
<td>64±56</td>
<td>0.14</td>
</tr>
<tr>
<td>Specific insulin, pmol/L</td>
<td>44±38</td>
<td>55±44</td>
<td>52±31</td>
<td>49±28</td>
<td>0.06</td>
</tr>
<tr>
<td>Fasting proinsulin, pmol/L</td>
<td>7±6</td>
<td>8±7</td>
<td>12±22</td>
<td>8±6</td>
<td>0.63</td>
</tr>
<tr>
<td>Fasting 32-33 split proinsulin, pmol/L</td>
<td>9±9</td>
<td>10±9</td>
<td>15±22</td>
<td>11±8</td>
<td>0.26</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>0.93±0.05</td>
<td>0.96±0.06‡</td>
<td>0.94±0.04</td>
<td>0.95±0.06</td>
<td>0.005</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>25.7±3.0</td>
<td>26.9±3.6†</td>
<td>26.2±3.6</td>
<td>26.5±3.3</td>
<td>0.01</td>
</tr>
<tr>
<td>Serum triglycerides, mmol/L</td>
<td>1.33±0.66</td>
<td>1.49±1.13</td>
<td>1.57±0.78</td>
<td>1.55±0.80</td>
<td>0.08</td>
</tr>
<tr>
<td>Serum NEFA, mmol/L</td>
<td>0.48±0.18</td>
<td>0.54±0.18</td>
<td>0.50±0.19</td>
<td>0.49±0.20</td>
<td>0.08</td>
</tr>
<tr>
<td>Serum total cholesterol, mmol/L</td>
<td>5.76±0.99</td>
<td>5.94±0.97</td>
<td>5.79±0.82</td>
<td>5.78±0.95</td>
<td>0.53</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>3.84±0.88</td>
<td>4.01±0.90</td>
<td>3.83±0.74</td>
<td>3.86±0.85</td>
<td>0.48</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.32±0.35</td>
<td>1.30±0.33</td>
<td>1.23±0.33</td>
<td>1.24±0.36</td>
<td>0.32</td>
</tr>
<tr>
<td>Use of antihypertensive medication, % (n)</td>
<td>29 (75)</td>
<td>30 (24)</td>
<td>55 (22)</td>
<td>49 (46)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Isolated systolic hypertension, % (n)</td>
<td>16 (42)</td>
<td>16 (13)</td>
<td>23 (9)</td>
<td>33 (31)</td>
<td>0.007</td>
</tr>
</tbody>
</table>

Values are mean±SD, unadjusted. Analyses are adjusted for use of antihypertensive medication.

*P<0.05, †P<0.01, §P<0.001, ‡P<0.0001 for difference vs group with normal left ventricular geometry.

and interventricular septum thickness instead of RWT or LVMI.

Insulin sensitivity index derived from the hyperinsulinemic euglycemic clamp, glucose tolerance at an OGTT, and fasting levels of specific insulin and 32-33 split proinsulin were all related to left ventricular RWT but not to LVMI in the present study. Insulin resistance and the accompanying hyperinsulinemia have often been suggested as a central derangement in the insulin resistance syndrome, and insulin has been regarded as a trophic factor responsible for the development of cardiovascular hypertrophy. Recent studies in the rat have shown that chronic moderate hyperinsulinemia, while maintaining control of hormones with effects opposing insulin, was followed by a pronounced hypertrophy of cardiac ventricles. In view of the results from the present study, the trophic effect might in humans mainly influence ventricular wall thickness, leaving cavity dimensions largely unaffected. This is also illustrated by the finding that subjects with left ventricular concentric remodeling had lower insulin sensitivity index and impaired glucose tolerance compared with subjects with normal left ventricular geometry. These findings are in accordance with another population study of men in which the insulin levels tended to be higher in subjects with concentric remodeling and concentric LVH and glucose and insulin levels correlated with RWT but not with LVMI. The negative correlation between LVMI and 2-hour insulin in the present study may be explained by the insulin resistance and glucose intolerance of the concentric remodeling group. The values of several insulin sensitivity variables were similar for concentric remodeling and concentric LVH, although significant only for the former. This may be due to a small concentric LVH group but may also reflect a real difference. The only significant differences between groups were in LVMI and 24-hour SBP and DBP. When the 2 groups were combined, results similar to those for the concentric remodeling group were obtained. In a subsample without valvular disease, controlling for ischemic heart disease, most relationships between metabolic variables and RWT or LVMI and differences between geometric groups were the same as in the main analysis. The finding that specific insulin and 32-33 split proinsulin but not intact proinsulin or immunoreactive insulin were related to RWT in this study may indicate that the first two have a more pronounced hypertrophic effect on the cardiac myocytes. However, at the low plasma levels detected in the present study, 32-33 split proinsulin should probably be seen as a correlate of insulin resistance rather than as having any significant metabolic effects of its own.
were related to RWT and concentric remodeling probably reflects the higher precision of the glucose measurements.

LVH has formerly been proposed to partially be an adaptation to obesity, especially in women. In the same study, left ventricular mass indexed to body surface area was not correlated to body mass index in men. In the present study, waist-to-hip ratio and body mass index were related to RWT and not to LVMI and were highest in the concentric remodeling group.

Heart rate was significantly increased in the concentric remodeling group, in accordance with previous research. Tachycardia is proposed to be a reliable marker for an increased sympathetic activity in population studies. Raised plasma catecholamine levels have been found in subjects with LVH and have more specifically been related to an increased interventricular septum thickness. Further evidence that the sympathetic nervous system could affect left ventricular geometry comes from experimental studies in dogs in which repeated pressor episodes with elevated plasma norepinephrine levels or chronic infusion of norepinephrine resulted in LVH but did not induce a sustained elevation of blood pressure. The inverse relationship between LVMI and heart rate reflects the increased heart rate in the concentric remodeling group and decreased heart rate in the eccentric LVH group, the latter probably made up of both subjects with heart failure and subjects with a physiological LVH caused by exercise, with low sympathetic activity and normal metabolic status. An inverse relationship between LVMI and heart rate in men has been found in another population-based study.

A deranged microcirculation with vascular hypertrophy and rarefaction of skeletal muscle blood vessels has been proposed as a central pathology in hypertension, insulin resistance, and dyslipidemia. This leads to an increased peripheral resistance, which is believed to be of pathogenetic importance for left ventricular concentric remodeling. Vascular rarefaction and insulin resistance have been associated with sympathetic hyperactivity, illustrated by the increased heart rate often found in subjects with the insulin resistance syndrome.

Left ventricular wall thickness and the prevalence of left ventricular concentric remodeling increase with age. This is mainly due to cardiac myocyte hypertrophy, for which an increased stroke work resulting from increased arterial stiffness has been proposed to be an etiological factor. Because insulin sensitivity also decreases with age, the described association between the insulin resistance syndrome and the growth of left ventricular walls could in part be a consequence of aging, a process proceeding faster in some subjects prone to both insulin resistance and cardiac remodeling.

This study has limited generalizability to women and other ethnic and age groups. Although a study in a younger population of men points toward the same conclusion, further studies in other groups are needed. The present cohort has been closely followed for 20 years and may therefore be healthier than average Swedish 70-year-old men. Thus, the associations found in this study may be weaker than in the general population. Confounding factors other than those adjusted for may also exist. Because many analyses were made, some chance associations may have been found. However, all components of the insulin resistance syndrome behaved in the same way in this study except for the cholesterol measurements. This might be due to chance or limits of the linear model but may also indicate that cardiac geometry is associated more with glucose and fatty acid metabolism than cholesterol metabolism.

In conclusion, several components of the insulin resistance syndrome were significantly related to thick left ventricular walls and concentric remodeling but less to LVH in this population-based sample of elderly men.

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