Plasminogen Activator Inhibitor-1 and Relations to Fatty Acid Composition in the Diet and in Serum Cholesterol Esters

Liisa Byberg, Annika Smedman, Bengt Vessby, Hans Lithell

Abstract—High plasminogen activator inhibitor (PAI)-1 levels and poor dietary fat quality are potential risk factors for cardiovascular disease. The aim was to investigate the cross-sectional associations between PAI-1 activity and dietary nutrient intake, focusing on fat quality, in a population-based study of 871 men aged 70 years. The relationship between PAI-1 and the fatty acid composition in serum cholesterol esters (n=381 men) was also studied. The estimated total fat intake was positively associated with PAI-1 activity. The intake of both monounsaturated and polyunsaturated fatty acids was positively associated with PAI-1 activity, whereas the intake of saturated fatty acids was not. In serum cholesterol esters, higher proportions of palmitoleic and dihomo-γ-linolenic acid, a lower proportion of linoleic acid, and reduced estimated Δ5-desaturase activity were associated with higher PAI-1 levels. These associations were confounded by factors representing the insulin resistance syndrome. PAI-1 activity was positively associated with γ-linolenic and arachidonic acid, independent of potential confounders. In conclusion, this study demonstrates that dietary intake of unsaturated fatty acids is positively associated with PAI-1 activity, whereas intake of saturated fatty acids is not. The associations present between PAI-1 activity and the fatty acid proportions in serum cholesterol esters are partly influenced by metabolic syndrome–related factors. (Arterioscler Thromb Vasc Biol. 2001;21:2086-2092.)

Key Words: plasminogen activator inhibitor-1 ■ fatty acid composition ■ diet ■ serum cholesterol esters ■ population-based study

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The Uppsala Longitudinal Study of Adult Men (ULSAM) has been described previously. In brief, all men born from 1920 to 1924 and
living in Uppsala in 1970 were invited to a health investigation in which 2322 participated (of the 2841 men invited). The men still alive and still living in the Uppsala region in 1991 (n=1681) were invited for a reexamination, in which 1221 men participated. The study was approved by the local ethics committee, and all men gave their informed consent.

The physical and metabolic examinations of the men at age 70 have been described in detail previously.14 Examinations included anthropometry (height, weight, and waist and hip circumferences), blood pressure, serum lipids (total, LDL and HDL cholesterol, and triglycerides), an oral glucose tolerance test, and a hyperinsulinemic euglycemic clamp test for determination of the insulin sensitivity index. The investigations took place in the morning after an overnight fast. A medical questionnaire was used to obtain information on current use of medicines and on the dietary and lifestyle habits. Marital status and educational level was retrieved from the Swedish census registry.

PAI-1 activity was analyzed in platelet-poor plasma from 914 of the 1221 men by use of an indirect enzymatic assay (Spectrolyse/P.L. PAI-1, Biopool AB). Blood samples were drawn in 5-mL evacuated tubes (Vacutainer, Becton Dickinson), which had been prefilled with 0.5 mL buffered 0.129 mol/L sodium citrate (pH 5.8), with the subject at rest in the supine position with minimal venous stasis. The tubes were put on ice until centrifugation at 4°C at 2000g for 10 minutes. The plasma was stored at −70°C until analysis.

An optically readable, precoded, 7-day food record was completed (Stata Corp). Skewed variables were transformed to reach normality and skewness of the dietary intake, before they were analyzed. The dietary intake of nutrients was divided by the total energy intake before analysis (grams per day per megajoules), although the crude intake (grams per day) is presented in the tables as arithmetic mean ± SD. The associations of variables with PAI-1 were investigated with pairwise or partial correlation analyses. The associations were adjusted for potential confounders suggested to influence the dietary habits, PAI-1 activity, or both. Confounders that were adjusted for were age, use of lipid-lowering or anti-hypertensive medicines, smoking, educational level, marital status, and physical activity. The associations were also adjusted for body mass index (BMI), waist/hip ratio, insulin sensitivity index, and serum triglyceride concentrations, which are components of the insulin resistance syndrome, and have been shown to be independently associated with PAI-1 activity.14 A value of P<0.05 was regarded as statistically significant.

### Results

The reported total energy intake per day was 7.3±1.9 MJ (mean±SD) and ranged between 2.3 and 19.4 MJ. To avoid confounding by extreme underreporting of the dietary intake,
the analyses were also performed when men with a reported energy intake <4.5 MJ were excluded (n=46). This exclusion did not change any of the results presented below. The total energy intake was inversely associated with PAI-1 activity (Table 3).

### Intake of Macronutrients

The reported intake of macronutrients, presented as grams per day, and the associations with PAI-1 activity are shown in Table 3. Carbohydrate intake and fiber intake were inversely associated with the activity of PAI-1. The intakes of protein and of alcohol were positively associated with PAI-1, as were the intakes of total dietary fat and of cholesterol. When the fat intake was divided into the intake of saturated, monounsaturated, and polyunsaturated fatty acids, there was no correlation between the intake of saturated fatty acids and PAI-1, whereas the intake of monounsaturated and also of polyunsaturated fatty acids was positively associated with PAI-1 activity.

Adjustment for potential confounders and metabolic variables (as described in Methods) did not alter the observed relationships, except for the association between fiber intake and PAI-1, which turned nonsignificant after adjustment for educational level or the level of physical activity.

### Dietary Fatty Acids

The reported intake of specific fatty acids and their associations with PAI-1 activity are presented in Table 4 as grams per day. PAI-1 activity was not associated with the dietary intake of any of the saturated fatty acids. The estimated

<table>
<thead>
<tr>
<th>Fatty Acids</th>
<th>Mean±SD</th>
<th>Range</th>
<th>E%</th>
<th>r</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>16:0</td>
<td>14.6±5.4</td>
<td>4.3–43.6</td>
<td></td>
<td>0.04</td>
<td>0.207</td>
</tr>
<tr>
<td>16:1 ω7</td>
<td>1.2±0.4</td>
<td>0.4–4.2</td>
<td></td>
<td>0.10</td>
<td>0.003</td>
</tr>
<tr>
<td>18:0</td>
<td>6.7±2.4</td>
<td>1.8–18.2</td>
<td></td>
<td>0.06</td>
<td>0.059</td>
</tr>
<tr>
<td>18:1 ω9</td>
<td>19.8±6.8</td>
<td>3.4–60.4</td>
<td></td>
<td>0.10</td>
<td>0.002</td>
</tr>
<tr>
<td>18:2 ω6</td>
<td>8.0±3.3</td>
<td>2.1–31.8</td>
<td></td>
<td>0.11</td>
<td>0.001</td>
</tr>
<tr>
<td>18:3 ω3</td>
<td>1.2±0.5</td>
<td>0.4–6.0</td>
<td></td>
<td>0.09</td>
<td>0.005</td>
</tr>
<tr>
<td>20:0</td>
<td>0.30±0.11</td>
<td>0.07–0.90</td>
<td>70.8</td>
<td>−0.001</td>
<td>0.981</td>
</tr>
<tr>
<td>20:4 ω6</td>
<td>0.13±0.06</td>
<td>0.02–0.45</td>
<td>12.1</td>
<td>0.12</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>20:5 ω3</td>
<td>0.09±0.07</td>
<td>0–0.47</td>
<td>6.7</td>
<td>0.03</td>
<td>0.342</td>
</tr>
<tr>
<td>22:5 ω3</td>
<td>0.04±0.02</td>
<td>0–0.16</td>
<td>5.1</td>
<td>0.11</td>
<td>0.002</td>
</tr>
<tr>
<td>22:6 ω3</td>
<td>0.22±0.13</td>
<td>0–0.91</td>
<td>2.7</td>
<td>0.13</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Energy-adjusted fatty acids were used in analyses: (g/d)–MJ. There were 871 men studied, except for 20:5 ω3 values (n=846), and 22:6 ω3 values (n=870).
intakes of the monounsaturated fatty acids (palmitoleic acid [16:1 \(\omega 7\)] and oleic acid [18:1 \(\omega 9\)]) were positively associated with PAI-1 activity. The intakes of the polyunsaturated \(\omega 6\) fatty acids (linoleic acid [18:2 \(\omega 6\)] and arachidonic acid [20:4 \(\omega 6\)]) and the \(\omega 3\) fatty acids (\(\alpha\)-linolenic acid [18:3 \(\omega 3\)] and docosapentaenoic acid [22:5 \(\omega 3\)]) were also positively associated with PAI-1 activity. PAI-1 activity was not associated with the intake of eicosapentaenoic acid (20:5 \(\omega 3\)) or docosahexaenoic acid (22:6 \(\omega 3\)); there was borderline significance. All associations were independent of the potential confounders and metabolic variables mentioned in Methods.

**Fatty Acid Composition in Serum Cholesterol Esters**

The fatty acid composition of the cholesterol esters in serum is presented in Table 5 as the percentage of the total amount of fatty acids analyzed. Some of the associations present between PAI-1 and the proportions of the cholesterol ester fatty acids (Table 5) are similar to (although in the opposite direction from) those present between the fatty acid composition and insulin sensitivity.\(^\text{18}\) Thus, the relative amounts of palmitic acid (16:0), palmitoleic acid (16:1 \(\omega 7\)), and dihomo-\(\gamma\)-linolenic acid (20:3 \(\omega 6\)) were positively associated with PAI-1 activity (16:0, borderline significance), and the relative amount of linoleic acid (18:2 \(\omega 6\)) was inversely associated with PAI-1, as was the estimated \(\Delta 5\)-desaturase activity. These associations were no longer significant when adjustments were made for BMI, waist/hip ratio, the insulin sensitivity index, and serum triglyceride levels. There were also positive associations between PAI-1 and \(\gamma\)-linolenic acid (18:3 \(\omega 6\)), arachidonic acid (20:4 \(\omega 6\)), and \(\Delta 6\)-desaturase activity. The \(\omega 3\) fatty acids eicosapentaenoic acid (20:5 \(\omega 3\)) and docosahexaenoic acid (22:6 \(\omega 3\)) were not significantly associated with PAI-1 levels, which was also true for the relative amounts of stearic acid (18:0), oleic acid (18:1 \(\omega 9\)), and \(\alpha\)-linolenic acid (18:3 \(\omega 3\)). The association between PAI-1 and arachidonic acid turned nonsignificant when adjustments were made for the metabolic variables mentioned above, whereas the relationships with eicosapentaenoic acid (20:5 \(\omega 3\)) and docosahexaenoic acid (22:6 \(\omega 3\)) became of significance (\(r=0.11, P=0.032\)) and of borderline significance (\(r=0.09, P=0.090\)), respectively. The strength of the positive association between PAI-1 and \(\Delta 6\)-desaturase activity was reduced after adjustment for metabolic confounders, but the association was still statistically significant. Adjustment for the other potential confounders did not change the associations observed.

**Pentadecanoic and Heptadecanoic Acid in Serum Cholesterol Esters and Phospholipids**

The proportion of pentadecanoic acid (15:0) in serum cholesterol esters, analyzed in a subgroup of 96 men, was inversely associated with PAI-1 activity (\(r=-0.20, P=0.050\)). The proportion of heptadecanoic acid (17:0) and the sum of the proportions of 15:0 and 17:0 in serum cholesterol esters were not significantly associated with PAI-1 activity (\(r=-0.08, P=0.545\) and \(r=-0.15, P=0.245\), respectively; \(n=64\)). The detectable proportions of pentadecanoic and heptadecanoic acid in serum lipids are very low (for 15:0 and 17:0 in serum cholesterol esters: mean\(\pm SD\) 0.21\(\pm 0.05\) [range 0.10 to 0.32] and mean\(\pm SD\) 0.10\(\pm 0.02\) [range 0.07 to 0.18], respectively). In fact, the proportion of heptadecanoic acid in serum cholesterol esters could be determined in only 64 of the 96 analyses. The proportion of heptadecanoic acid was higher in serum phospholipids than in the cholesterol esters (0.46\(\pm 0.09\) and 0.20 to 0.87, respectively; \(n=96\)), and there was also an inverse relationship with PAI-1 activity that was of borderline significance (\(r=-0.19, P=0.064\)). The proportion of pentadecanoic acid in serum phospholipids was similar to that in the cholesterol esters, and

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**TABLE 5. Fatty Acid Composition of Serum Cholesterol Esters and Association With PAI-1 Activity**

<table>
<thead>
<tr>
<th>Fatty Acids</th>
<th>Men, n</th>
<th>Mean(\pm SD)</th>
<th>Range</th>
<th>(r)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16:0</td>
<td>381</td>
<td>11.7(\pm 0.8)</td>
<td>9.9–14.3</td>
<td>0.10</td>
<td>0.061</td>
</tr>
<tr>
<td>16:1 (\omega 7)</td>
<td>381</td>
<td>3.7(\pm 1.2)</td>
<td>2.0–10.4</td>
<td>0.20</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>18:0</td>
<td>381</td>
<td>0.94(\pm 0.21)</td>
<td>0.48–1.82</td>
<td>−0.04</td>
<td>0.403</td>
</tr>
<tr>
<td>18:1 (\omega 9)</td>
<td>381</td>
<td>20.5(\pm 2.2)</td>
<td>15.1–29.8</td>
<td>0.07</td>
<td>0.167</td>
</tr>
<tr>
<td>18:2 (\omega 6)</td>
<td>381</td>
<td>52.1(\pm 4.3)</td>
<td>38.0–61.7</td>
<td>−0.18</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>18:3 (\omega 3)</td>
<td>381</td>
<td>0.84(\pm 0.19)</td>
<td>0.37–1.47</td>
<td>−0.07</td>
<td>0.189</td>
</tr>
<tr>
<td>18:4 (\omega 6)</td>
<td>379</td>
<td>0.65(\pm 0.27)</td>
<td>0.21–1.68</td>
<td>0.25</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>19:3 (\omega 6)</td>
<td>379</td>
<td>0.71(\pm 0.14)</td>
<td>0.38–1.31</td>
<td>0.30</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>20:3 (\omega 6)</td>
<td>379</td>
<td>5.7(\pm 1.1)</td>
<td>3.5–10.2</td>
<td>0.15</td>
<td>0.003</td>
</tr>
<tr>
<td>20:4 (\omega 6)</td>
<td>381</td>
<td>1.8(\pm 0.8)</td>
<td>0.6–5.6</td>
<td>0.08</td>
<td>0.099</td>
</tr>
<tr>
<td>22:3 (\omega 3)</td>
<td>368</td>
<td>0.99(\pm 0.24)</td>
<td>0.42–2.02</td>
<td>0.07</td>
<td>0.162</td>
</tr>
<tr>
<td>20:4 (\omega 6/20:3 (\omega 6)</td>
<td>379</td>
<td>5.6(\pm 1.9)</td>
<td>4.6–14.7</td>
<td>−0.15</td>
<td>0.004</td>
</tr>
<tr>
<td>18:3 (\omega 6/18:2 (\omega 6)</td>
<td>379</td>
<td>0.01(\pm 0.01)</td>
<td>0.00–0.04</td>
<td>0.26</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>18:1 (\omega 9/18:0)</td>
<td>381</td>
<td>22.7(\pm 5.2)</td>
<td>10.2–49.4</td>
<td>0.08</td>
<td>0.117</td>
</tr>
<tr>
<td>18:0/16:0</td>
<td>381</td>
<td>0.08(\pm 0.02)</td>
<td>0.04–0.16</td>
<td>−0.08</td>
<td>0.130</td>
</tr>
</tbody>
</table>

Fatty acids are presented in relative amounts (percentage of total fatty acids analyzed).
the inverse association with PAI-1 was also of borderline significance ($r=-0.18$, $P=0.076; n=95$). The sum of $15:0$ and $17:0$ in the serum phospholipids was inversely associated with PAI-1 activity ($r=-0.21$, $P=0.041; n=95$). The associations between the fatty acid composition in serum cholesterol esters (besides pentadecananoic and heptadecananoic acid) and PAI-1 activity were similar in this subgroup of 96 men and in the main group of 381 men (Table 5).

Discussion

In the present study, we demonstrate a clear positive association between the dietary intake of unsaturated fatty acids and the activity of PAI-1 when, at the same time, the intake of saturated fatty acids is unrelated to the PAI-1 levels. This pattern is present when the total amounts of saturated, monounsaturated, and polyunsaturated fatty acids and of separate fatty acids in the diet are studied.

These results are contradictory in the sense that high PAI-1 levels are regarded as a risk factor for cardiovascular disease and that high intake of unsaturated fatty acids is believed to be protective. The mechanisms behind the cross-sectional associations in the present study may be the increased production of PAI-1 by unsaturated fatty acids11–13 but not by saturated fatty acids,13 as observed in vitro.

This is the first population study to cross-sectionally investigate the fatty acid composition in the diet and in serum cholesterol esters in relation to PAI-1. In a recent European multicenter study,19 the concentration of PAI-1 antigen was not associated with the fatty acid composition of erythrocyte phospholipids, whereas there was an inverse association between tissue-type plasminogen activator (tPA) antigen concentrations and the proportion of total ω3 fatty acids.19 The tPA antigen concentration mainly reflects PAI-1 activity and not tPA activity (PAI-1 is the physiological inhibitor of tPA). The discrepant results between this multicenter study and the present study are probably mainly due to the different sites at which the fatty acid composition was analyzed, ie, erythrocyte phospholipids and serum cholesterol esters. The multicenter study also included a younger study population, and all presented relationships were adjusted for BMI, factors that could further contribute to the discrepant results.

In intervention studies, supplementation with long-chain ω3 fatty acids or increased fish intake has been observed to increase PAI-1 levels, in spite of a reducing effect on serum triglycerides.8,9 However, some studies investigating the effect of ω3 supplementation on PAI-1 levels could not find a difference between the ω3-supplementation group and the control group (see summary10). An ω6 fatty acid is often used as a placebo in the control group to compensate for the increased fat intake in the ω3 treatment group. The reason for the lack of difference between ω3 and ω6 treatment groups in some studies could be the increasing effect of not only ω3 but also ω6 unsaturated fatty acids on PAI-1 production seen in vitro.11–13 The observations in the present study, in which dietary intake of unsaturated fatty acids is positively associated with PAI-1 activity, also support this idea. The few studies demonstrating a reduction of PAI-1 after ω3 supplementation were methodologically questionable (there was lack of a control group and placebo,20 and PAI-1 was measured in serum21).

It has been hypothesized that the increasing effect of ω3 fatty acids on PAI-1 levels may be compensatory for the increased tendency to bleeding observed in Inuit populations naturally eating a diet rich in long-chain ω3 fatty acids.22 The risk of cardiovascular disease may not be increased with an increase in the PAI-1 levels, because unsaturated fatty acids are beneficial in other aspects.2

On the nutrient level, there was a strong positive association between protein intake and PAI-1 activity. Others have demonstrated positive associations of the intake of fat1, alcohol6,7 with PAI-1 and inverse associations of the intake of carbohydrates4 and fibers4,5 with PAI-1, associations also noted in the present study. However, fiber intake was not independent of physical activity or educational level in the present study. Explanations for the associations between PAI-1 and the intake of protein and between PAI-1 and the intake of carbohydrates remain to be discovered. However, these associations were not dependent of fat intake or the dietary fat quality.

Intervention studies comparing diets rich in saturated fat versus polyunsaturated fat have demonstrated that the saturated fat–rich diet gives the same serum cholesterol ester fatty acid pattern as that observed in insulin-resistant individuals.18 This specific fatty acid pattern includes higher proportions of palmitic, palmitoleic, and dihomo-γ-linolenic acid, whereas the proportion of linoleic acid and the estimated Δ5-desaturase activity are lower.18 In addition, the estimated Δ6-desaturase activity appears to be higher.18 The same fatty acids that are changed after the saturated fat diet and in insulin-resistant individuals are related to high PAI-1 activity in the present study. However, these associations seem to be confounded by factors associated with the insulin resistance syndrome, namely, BMI, waist/hip ratio, serum triglyceride concentrations, and insulin sensitivity. We have previously demonstrated that these factors are independently related to PAI-1 activity in the present cohort.14 The results suggest that the processes involved in the metabolism of dietary fatty acids included in the serum cholesterol esters somehow could be linked to the insulin-resistant state. It could also be that the dietary habits influence insulin resistance and the incorporation of fatty acids into serum cholesterol esters.

The proportions of fatty acids present in the serum cholesterol esters were not associated with PAI-1 activity in the same manner as the fatty acids in the diet. There are several possible reasons for this. The fatty acid composition of serum cholesterol esters mimics the dietary intake over the last couple of weeks.23 However, some fatty acids, such as myristic, palmitic, stearic, palmitoleic, and oleic acids, can also be produced endogenously, whereas linoleic and α-linolenic acids cannot be produced endogenously and must be ingested.24 The fatty acid composition in a tissue also depends on other factors, including the activity of the different fatty acid desaturases, elongation, retroconversion, and oxidation systems, which all act on the fatty acids ingested.24 Before ending up in the serum cholesterol esters, the fatty acids are also esterified, and the esterification process also affects the final composition of fatty acids.24 Other determinants of the fatty acid composition in body tissues or body lipid structures...
are degree of physical activity, genetic disposition, and (possibly) fetal malnutrition. The method for measuring the fatty acid composition of the serum gives the proportion of a certain fatty acid in relation to the total amount of fatty acids analyzed. Thus, when the proportion of one fatty acid increases, the proportions of the others decrease, and vice versa. Data on the fatty acid composition of the diet are the estimated absolute amounts from the reported dietary intake and, thus, are not dependent on each other in the same manner.

A specific fatty acid in serum cholesterol esters may originate from different foodstuffs in different types of diets. For example, in a Northern European diet, a large part of the proportion of oleic acid in serum cholesterol esters originates from the intake of foodstuffs rich in saturated fatty acids and, today, also from rapeseed oil. In a Mediterranean diet, the proportion of oleic acid largely originates from the intake of olive oil. Thus, the results concerning the relationships between the fatty acid composition in serum cholesterol esters and PAI-1 may not be applicable to all populations, but this remains to be studied.

The proportions of pentadecanoic (15:0) and heptadecanoic (17:0) acid in serum and adipose tissue have been demonstrated to be specific markers of the intake of milk fat in the present cohort and in other studies. These fatty acids are mainly produced by ruminants. The inverse associations between PAI-1 activity and these markers of milk fat intake are in line with the inverse associations between milk fat intake and metabolic cardiovascular risk factors presented earlier. The consumption of milk products has also been inversely associated with the levels of tPA antigen. As mentioned above, the tPA antigen concentration mainly reflects PAI-1 activity; therefore, the results are in line with the present study. There were no significant associations between PAI-1 and the other saturated fatty acids analyzed in serum cholesterol esters (ie, palmitic acid and stearic acid). Thus, the proportions of pentadecanoic and heptadecanoic acid may more truly mirror the dietary intake of dairy products. Endogenous production, desaturation, and elongation may have influenced the proportions of palmitic and stearic acid, which are also to a large extent present in foodstuffs other than dairy products.

Because the present study is observational, no conclusions regarding cause and effect can be made. However, the present study is unique in the sense that it is the largest population-based study of elderly men presented that includes the estimation of fatty acid composition in the diet and measurements of the fatty acid composition in serum cholesterol esters. The nutrient intake and the fatty acid composition of the diet were estimated from dietary records. This comprehensive dietary record is a validated method that is rarely used in large epidemiological studies. The relative nutrient intake has been observed to be similar in Swedish men and women, although the energy intake is generally higher among men. Because the associations regarding dietary intake were adjusted for total energy intake in the present study, the results may also be applicable in women, although this remains to be established.

In conclusion, the present study demonstrates that the dietary intake of unsaturated fatty acids is positively associated with PAI-1 activity, whereas the intake of saturated fatty acids is not. The associations present between PAI-1 activity and the fatty acid proportions in serum cholesterol esters are partly influenced by factors associated with the insulin resistance syndrome.

Acknowledgments

This work was supported by grants from the Ernfrors Fund for Diabetes Research, the Foundation for Geriatric Research, “Förenade Liv” Mutual Group Life Insurance Co, the Medical Faculty at Uppsala University, the Swedish Council for Planning and Coordination of Research (No. A19-562), the Swedish Diabetes Association, the Swedish Medical Research Council (No. 5446), the Swedish National Association Against Heart and Lung Disease, and the Trygg Hansa Research Fund.

References

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*Arterioscler Thromb Vasc Biol.* 2001;21:2086-2092
doi: 10.1161/hq1201.100224

*Arteriosclerosis, Thrombosis, and Vascular Biology* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1079-5642. Online ISSN: 1524-4636

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