Restriction Fragment Length Polymorphism of the Apoprotein A-I–C-III Gene Cluster in Control and Stroke-Prone White and Black Subjects: Racial Differences

R. Kasturi, PhD; F.M. Yatsu, MD; R. Alam, PhD; and S. Rogers, BS

**Background and Purpose:** The presence of known restriction fragment length polymorphisms in the apoprotein A-I–C-III gene cluster, which encodes their respective apoproteins, was investigated using the restriction enzymes Sac I and Pst I to determine the potential role of genetic variations for stroke risk in an American population.

**Methods:** Ninety-eight subjects (70 white, 28 black subjects), both normal controls with no carotid stenosis and those with carotid stenosis believed at risk for stroke, defined as showing stenosis focally or diffusely at that site, composed the study population.

**Results:** Sac I polymorphic S2 allele frequency was higher in stroke-risk groups, whereas Pst I polymorphic P2 allele frequency was similar in control and stroke-risk groups. Significantly higher levels of serum cholesterol, triglycerides, and low density lipoprotein ($p<0.05$) and significantly lower levels of high density lipoprotein ($p<0.05$) were observed in stroke-risk groups with diffuse stenosis. Results of our study with the two racial groups show the following: the frequency of Sac I polymorphism was significantly higher in American black compared with American white subjects ($\chi^2=3.92, p<0.05$). Among serum lipids, triglycerides were significantly higher in white compared with black subjects ($p<0.05$). In white subjects, carotid artery stenosis was associated with significantly elevated total cholesterol and low density lipoprotein ($p<0.01$) but not with Sac I polymorphism. In black subjects the converse was observed, namely, the Sac I polymorphic S2 allele seemed to be associated with carotid bifurcation stenosis but did not reach statistical significance because of the small number of subjects. In addition, Sac I polymorphism did not correlate with any lipid profile. Pst I polymorphism was not associated with any lipid profile or carotid artery stenosis abnormalities.

**Conclusions:** Our results indicate that carotid artery stenosis identifies white subjects with increased plasma total cholesterol and low density lipoprotein, an atherogenic profile, but not with Sac I polymorphism. These findings suggest that carotid bifurcation stenosis in white subjects is associated with an atherogenic lipid profile but not with apoprotein A-I–C-III restriction fragment length polymorphisms. In black subjects, Sac I polymorphism seems to identify those individuals with significant carotid stenosis, a necessary precursor to atherothrombotic brain infarction, but not those with elevated total cholesterol, elevated low density lipoprotein, and/or reduced high density lipoprotein. These results suggest that Sac I polymorphism may identify black subjects at increased risk for atherothrombotic brain infarctions. (Stroke 1992;23:1257–1264)

**KEY WORDS** • racial differences • genetics • cholesterol • lipoproteins, HDL

A combination of factors conspires in the development of atherosclerosis and includes hypercholesterolemia, smoking, hypertension, and diabetes mellitus. An important association between abnormal lipid metabolism and atherosclerosis leading to strokes or atherothrombotic brain infarctions (ABI) and coronary heart disease (CHD) has been made in several studies. Various proteins are involved in lipid metabolism functioning as lipid-binding proteins, enzymes, ligands, and receptors. Any alteration in their structure and function may affect lipid metabolism in provoking the atherosclerotic process. If these protein defects causing premature atherosclerosis are genetic, identification of individuals harboring these genes should provide a potential means of disease prevention by instituting appropriate interventional therapies, such as serum cholesterol reduction.

To evaluate the specificity of genetic factors in atherosclerosis, particularly in causing ABIs, genetic variations of candidate gene(s) must be determined directly in an individual's DNA by using gene-specific DNA probes, even in the absence of identifiable phenotypic variations. Changes in nucleotide sequences of DNA can be detected by alterations in the sizes of distinct DNA
fragments generated by specific restriction endonucleases when hybridized with gene-specific probes. With these restriction fragment length polymorphisms (RFLPs), it is possible to analyze genetic variations associated with various pathological conditions. Variations in the hybridization pattern of DNA to the target probe identify different alleles, which can be used to determine the allele frequency associated with a particular disease compared with the frequency in the general population or in families.

Lipid profile studies with CHD have shown a more or less consistent pattern of increased plasma low density lipoprotein (LDL) and reduced plasma high density lipoprotein (HDL). Furthermore, reduced HDL alone and its major apoprotein, apolipoprotein (apo) A-I, correlate with premature CHD. Several HDL and apo A-I deficiency states have been described, resulting from different genetic alterations; the most common deficiency state is an autosomal dominant disorder resulting in familial hypoalphalipoproteinemia, which is associated with premature CHD and stroke. Other apo A-I deficiency states have been described, resulting from increased risk for ABIs, significant carotid bifurcation stenosis, and lipid abnormalities, such as elevated total cholesterol, increased LDL, and/or reduced HDL.

The objective of our study was to determine whether genetic alterations in the apo A-I-C-III gene cluster can identify subjects with asymptomatic carotid stenosis who are at increased risk for progression of stenosis and ABIs. We investigated the occurrence of known RFLPs in the apo A-I-C-III gene cluster, particularly with the restriction enzymes Sac I and Pst I, as markers for increased stroke risk, reflected in the occurrence of ABIs, significant carotid bifurcation stenosis, and lipid abnormalities, such as elevated total cholesterol, increased LDL, and/or reduced HDL.

Subjects and Methods

Subjects

Both control and stroke-risk subjects were obtained from the local population through groups affiliated with churches and other organizations. Ninety-eight subjects (70 white, 28 black subjects; 50 men, 48 women) were included in the study. Ages ranged from 45 to 80 years; 80% of the subjects were 45–70 years of age. Carotid ultrasound and lipid analysis were performed for each subject. The 98 study subjects were classified into control and stroke-risk groups based on the presence of carotid artery stenosis. Subjects exhibiting <30% carotid artery stenosis were used as the control group; subjects displaying >30% carotid artery stenosis were classified as stroke-risk subjects. Stroke-risk subjects were divided into groups 1 and 2 based on the degree of stenosis. Subjects having >30% carotid artery stenosis in one or more locations were classified as stroke-risk group 1. Subjects exhibiting >30% stenosis in more than three locations were indicated as stroke-risk group 2. Stroke-risk group 1, therefore, included all the subjects of stroke-risk group 2. Some of the subjects in group 2 had suffered strokes.

Carotid Ultrasound

A diasonics duplex B-mode imager with pulse Doppler real-time spectral analysis was used in this study (model DRF-400 RV, Diasonics, Milpitas, Calif.). Measurement of percent stenosis was performed as described by Grotta et al. Carotid ultrasound measurements were obtained at three different positions: distal common, bulb, and proximal internal in both the carotid arteries (six measurements).

Lipid Analysis

Total serum cholesterol, triglycerides, LDL, and HDL measurements were performed according to the standardized techniques followed by the Lipid Research Clinics of the National Institutes of Health.

DNA Analysis

Chromosomal DNA was isolated from peripheral blood lymphocytes as described by Baas et al and Blin and Stafford. To 10 ml blood collected in the presence of ethylenediaminetetraacetic acid (EDTA), 30 ml of lysis buffer containing 155 mM NaCl, 10 mM NH4HCO3, and 0.1 mM EDTA and 1.1 ml 3.8% sodium citrate were added, and the suspension was incubated on ice for 15 minutes with occasional shaking. Then the sample was centrifuged at 3000g for 10 minutes at 4°C. The pellet was washed once with 5 ml lysis buffer and suspended in 4.5 ml of 75 mM NaCl–25 mM EDTA, pH
FIGURE 1. Restriction map of apolipoprotein (APO) A-I and C-III region showing Sac I and Pst I sites and expected DNA fragment sizes after hybridization with apo A-I probe. Common and variant alleles of Sac I and Pst I are designated as S1, S2 and P1, P2, respectively. Polymorphic sites are indicated by asterisk.

8.0. To the homogenous suspension, 0.5 ml of 10 mg/ml proteinase K and 0.2 ml of 20% sodium dodecyl sulfate (SDS) were added, and the suspension was incubated overnight on a shaker at 37°C. The suspension was extracted two times with an equal volume of phenol saturated with TE buffer (10 mM tris(hydroxymethyl)aminomethane [Tris] [pH 7.8] and 1 mM EDTA) followed by two chloroform extractions. The aqueous phase was centrifuged for 15 minutes at 5000g to remove any undissolved material. DNA was precipitated from the aqueous phase by adding two volumes of ethanol and washed with 70% ethanol followed by absolute ethanol. DNA precipitate was then placed in TE buffer and allowed to dissolve.

Southern Analysis

Approximately 10–20 μg DNA was digested with 2 units of Sac I or Pst I/μg DNA for approximately 16–20 hours according to the manufacturer’s suggestion. The digested DNA was separated electrophoretically on 1% agarose gel. The DNA fragments were transferred to nitrocellulose membrane according to Southern blotting procedure. The filters were prehybridized overnight at 65°C in 6× standard saline citrate (SSC), 50 mM Tris (pH 7.5), 1 mM EDTA, and 4×Denhardt’s solution. Hybridization was performed by incubating the filters in a solution containing 10^6 cpm/ml of phosphorus-32-labeled 2.2-kb apo A-I gene probe, 3×SSC, 25 mM Tris (pH 7.5), 0.5 mM EDTA, and 2×Denhardt’s solution. The filters were washed two times each in 3×SSC/0.2% SDS and 1.5×SSC/0.2% SDS at 65°C. The final wash was performed using 2×SSC at room temperature. Each wash lasted 15 minutes. Autoradiography was established using a Dupont Corenex intensifying screen at −70°C.

FIGURE 2. Southern blots showing hybridization pattern of Sac I-digested DNA probed with apolipoprotein A-I. Left panel: DNA from individuals with common Sac I allele, generating 5.7-kb and 4.5-kb DNA fragments. Right panel: DNA from subjects with polymorphic Sac I allele, exhibiting 3.2-kb polymorphic fragment in addition to 5.7- and 4.5-kb fragments.
Apolipoprotein A-I Probe

A 2.2-kb apo A-I gene was obtained by digesting the vector (pSV2 gpt) with EcoRI. The 2.2-kb fragment was separated on low-melt agarose gel and purified. For Southern hybridization the 2.2-kb fragment was nick-translated using 32P deoxycytidine triphosphate. The apo A-I gene probe was provided by Dr. S. Karathanasis, Harvard Medical School, Boston.

Statistical Analysis

Mean cholesterol, triglycerides, LDL, and HDL values of different groups were compared using Student’s t test analysis. The analysis of genotype frequencies was performed using \( \chi^2 \) analysis. Data are expressed as mean ± SD.

Results

We analyzed 98 subjects for apo A-I gene polymorphism using a 2.2-kb apo A-I gene probe. Figure 1 shows the restriction map of apo A-I and C-III gene regions. When the apo A-I gene was used as a hybridization probe, two types of alleles were detected in Sac I restriction endonuclease-digested DNA samples. The common allele generated invariant 5.7-kb and 4.5-kb gene fragments (Figure 1 and Figure 2, left panel), whereas the uncommon allele produced invariant 5.7-kb and 3.2-kb polymorphic (S2) DNA fragments either with or without the 4.5-kb fragment (S1) (Figure 1 and Figure 2, right panel). The uncommon, polymorphic Sac I allele, S2, arises from the presence of an additional Sac I recognition site located in or close to the 3' noncoding region of the apo C-III gene and is about 2.7 kb downstream of the apo A-I gene. Using Pst I endonuclease, it is shown that the apo A-I probe hybridizes to a common allele (P1) of 2.2 kb and an uncommon allele (P2) of 3.3 kb (Figures 1 and 3). The uncommon P2 allele site is at the 3' end of the apo A-I gene, about 300 bp to the apo A-I gene.

The genotype and allele frequencies of the polymorphisms detected by Sac I and Pst I restriction endonucleases are shown in Table 1. The genotype frequency of Sac I polymorphism was high in both stroke-risk groups compared with the control group. However, the difference was not statistically significant. On the other hand, the genotype frequency of Pst I polymorphism was almost similar in the control and stroke-risk groups. A comparison of S2 and P2 allele frequencies in the control and two stroke-risk groups revealed that S2 allele frequency was higher in both stroke-risk groups (0.14 and 0.1 in groups 1 and 2, respectively) compared with the control group (0.06). The allele frequency of polymorphic Pst I was almost similar in the control and stroke-risk groups.

The data were analyzed further to determine the possible correlations between the genotype expressed and the lipoprotein phenotype exhibited in the three different groups (Table 2). The information displayed in Table 2 illustrates that there was no significant difference in the total serum cholesterol between all three groups. However, stroke-risk group 2 exhibited significantly higher levels of triglycerides and LDL compared with the control group and stroke-risk group 1 (\( p<0.05 \)). Furthermore, both stroke-risk groups showed significantly lower levels of HDL (\( p<0.05 \)).

An analysis was performed to determine whether race-oriented differences existed in regard to apo A-I genotype and serum lipid components. When the frequency of Sac I polymorphism was compared between American white (\( n=70 \)) and black (\( n=28 \)) subjects, the frequency of Sac I polymorphism was significantly higher in black subjects (\( \chi^2=3.92, p<0.05 \)). The numbers of white and black subjects exhibiting Sac I polymorphism were 12 of 70 and 10 of 28, respectively. Among the serum lipids, all lipids except triglycerides were similar in both racial groups. In white subjects, the level of serum triglycerides was significantly higher (150±66 mg/dl) than in black subjects (121±50 mg/dl) (\( p<0.05 \)). Further, an analysis was carried out within each racial group. Both American white and black subjects were classified into three groups: control and stroke-risk groups 1 and 2 (Table 3). As in the population study, these racial groups were classified based on the degree of carotid artery stenosis. The control group included subjects with <30% stenosis. Stroke-risk group 1 consisted of subjects with >30% stenosis in one or more locations. Stroke-risk group 2 consisted of subjects with severe stenosis, having >30% stenosis in more than three locations. In both white and black groups there was increased frequency of Sac I polymorphism in both
stroke-risk groups. Approximately 17%, 50%, and 45% of black subjects in the control group and stroke-risk groups 1 and 2, respectively, exhibited Sac I polymorphism. However, when compared with control subjects, Sac I polymorphism did not reach the significance level because of the small number of subjects. Similarly, the allele frequency of Sac I polymorphism in black subjects was higher in the stroke-risk groups (0.25 and 0.22 in groups 1 and 2, respectively) compared with the control group (0.08). But there were no significant differences in serum lipids between the control and stroke-risk groups.

In the American white population approximately 14%, 18%, and 20% of the control group and stroke-risk groups 1 and 2, respectively, displayed Sac I polymorphism. The frequency of Sac I polymorphism was high in both stroke-risk groups (0.10 in both groups) compared with the control group (0.07) but was not statistically significant. However, serum lipids were significantly different between the control and stroke-risk groups. Significantly higher levels of serum cholesterol and LDL existed in both stroke-risk groups compared with the control group (p<0.01).

Discussion

The subjects in our study were analyzed for carotid artery stenosis, apo A-I gene polymorphism, and serum lipids. The extent of stenosis was compared with the frequency of Sac I and Pst I RFLPs in the apo A-I-C-III gene cluster to assess their relation in identifying subjects who are susceptible to strokes. Comparison was also made to serum lipid phenotypes such as cholesterol, triglycerides, LDL, and HDL levels.

Our population study revealed that a relation exists between the extent of carotid stenosis, levels of serum triglycerides, LDL, HDL, and Sac I polymorphism. The frequency of Sac I polymorphism was higher in the stroke-risk group displaying significant carotid stenosis (0.10) compared with the control group (0.06), although the difference was not statistically significant. In addition, the stroke-risk group comprising subjects with diffuse carotid artery stenosis (stroke-risk group 2) exhibited significantly higher levels of serum triglycerides and LDL (p<0.05) and significantly lower levels of serum HDL (p<0.05). These observations indicate a possible association between Sac I polymorphism and serum lipids in the stroke-risk group displaying diffuse carotid stenosis. We also observed an inverse relation between triglycerides and HDL. A similar inverse relation has been reported for ABI's and CHD. 

Several studies indicate that Sac I polymorphism in the apo A-I-C-III gene cluster is associated with hypertriglyceridemia. suggesting that the polymorphic site may be present in such a location to alter the structure, function, or expression of apo C-III, which could be etiologic in hypertriglyceridemia. Apo C-III is a component of chylomicrons, very low density lipoprotein (VLDL), and HDL. The physiological role of apo C-III is not clear, but it has been suggested that it may modulate lipoprotein lipase activity by interfering with apo C-II-induced activation of lipase.

Our population study revealed virtually identical variant Pst I allele frequency in both the control and stroke-risk groups. Several investigators have observed variations in the frequency of the Sac I and Pst I polymorphic allele between racial groups. Analysis of our subjects based on their racial back-

### Table 1: Genotype Distribution and Relative Allele Frequencies of Apoprotein A-I-C-III Restriction Fragment Length Polymorphisms Detected With Sac I and Pst I in Control and Stroke-Risk Subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Subjects (n)</th>
<th>Genotype</th>
<th>Allele frequencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sac I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>33</td>
<td>S1S1</td>
<td>5752</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>57±4.5 kb</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>52±3.2 kb</td>
</tr>
<tr>
<td>Stroke-risk 1</td>
<td>65</td>
<td>47</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.86</td>
</tr>
<tr>
<td>Stroke-risk 2</td>
<td>35</td>
<td>28</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.90</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.10</td>
</tr>
<tr>
<td>Pst I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>33</td>
<td>P1P1</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.95</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.05</td>
</tr>
<tr>
<td>Stroke-risk 1</td>
<td>65</td>
<td>60</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.96</td>
</tr>
<tr>
<td>Stroke-risk 2</td>
<td>35</td>
<td>33</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.97</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.03</td>
</tr>
</tbody>
</table>

*Number of subjects with and without S2 or P2 allele.

Stroke-risk group 1, subjects with >30% carotid artery stenosis in one or more locations; stroke-risk group 2, subjects with >30% stenosis in more than three locations.

### Table 2: Serum Lipid Components of Control and Stroke-Risk Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Subjects (n)</th>
<th>Lipids (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Cholesterol</td>
</tr>
<tr>
<td>Control</td>
<td>33</td>
<td>219±42</td>
</tr>
<tr>
<td>Stroke-risk 1</td>
<td>65</td>
<td>228±41</td>
</tr>
<tr>
<td>Stroke-risk 2</td>
<td>35</td>
<td>234±45</td>
</tr>
</tbody>
</table>

Values are mean±SD. LDL, low density lipoprotein; HDL, high density lipoprotein; stroke-risk group 1, subjects with >30% carotid artery stenosis in one or more locations; stroke-risk group 2, subjects with >30% stenosis in more than three locations.

*p<0.05, comparison of control vs. stroke-risk group by Student’s t test.

**Kasturi et al** Apoprotein A-I-C-III Gene Cluster in White and Black Subjects 1261
grounds resulted in the emergence of certain differences regarding Sac I polymorphism and serum lipids. The frequency of Sac I polymorphism was significantly higher in black compared with white subjects ($\chi^2 = 3.92, p < 0.05$). Serum triglyceride content was significantly higher in white than in black subjects ($p < 0.05$). None of the other serum lipids showed significant differences between the two racial groups. Similarly, several other investigators observed variations in the frequency of the Sac I polymorphic allele between different racial groups.28-29 If the polymorphic allele is linked to a polymorphic allele in the black stroke-risk group, with a higher frequency of Sac I polymorphism. However, no association with the serum lipid components except HDL were lower in black subjects. The difference in the frequency of the polymorphic allele among various racial groups may be due to selection pressure for or against the allele or simply due to neutral drift.

In our study, risk for stroke was inferred by the degree of carotid artery stenosis as determined by ultrasonic measurements. We analyzed white and black study subjects to determine whether an association exists between carotid artery stenosis, serum lipids, and Sac I polymorphic genotype. From our study, a strong relation was apparent between LDL, total cholesterol, and carotid artery stenosis in white subjects, but not with Sac I polymorphism. However, no association with serum lipids was present in black subjects. In fact, all the serum lipid components except HDL were lower in the black stroke-risk group, with a higher frequency of polymorphic Sac I genotype compared with control subjects.

Mortality due to hypertension, hypertensive heart disease, and stroke as a direct complication of hypertension is several times higher in the American black than in the American white population.65,66 The reported coronary heart disease mortality, on the other hand, is lower in black men but higher in black women.65,66 The decreased rates of CHD and myocardial infarction in black men prompted the search for mechanisms explaining this apparent vascular paradox. One such mechanism could be the higher relative levels of antiatherogenic HDL-cholesterol and lower atherogenic LDL-cholesterol compared with white men. Several studies show higher HDL-cholesterol and lower LDL- and VLDL-cholesterol in black compared with white subjects,67-68 and the differences are larger in men than in women.69 Differences in HDL-cholesterol and complementary differences in VLDL- and LDL-cholesterol have been observed in several studies. Understanding the contribution of genetic and environmental factors to the elevated HDL-cholesterol of black men and associated protection against coronary artery disease risk may clarify further the protective, antiatherogenic nature of HDL. However, the increased frequency of Sac I polymorphic allele in the black stroke-risk group suggests that stroke risk due to carotid stenosis results from other risk factors but does not exclude abnormal metabolism of LDL, HDL, and triglycerides.

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**TABLE 3. Comparison Between Control and Stroke-Risk Groups of White and Black Americans**

<table>
<thead>
<tr>
<th>Group</th>
<th>Subjects (n)</th>
<th>Allele frequency</th>
<th>Lipids (mg/dl)</th>
<th>TC</th>
<th>TG</th>
<th>LDL</th>
<th>HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>White Control</td>
<td>21</td>
<td>0.93, 0.07</td>
<td></td>
<td>204±31</td>
<td>135±64</td>
<td>122±29</td>
<td>52±16</td>
</tr>
<tr>
<td>Stroke-risk 1</td>
<td>49</td>
<td>0.90, 0.10</td>
<td></td>
<td>235±41*</td>
<td>157±66</td>
<td>154±38*</td>
<td>48±11</td>
</tr>
<tr>
<td>Stroke-risk 2</td>
<td>29</td>
<td>0.90, 0.10</td>
<td></td>
<td>239±41*</td>
<td>168±67</td>
<td>156±39*</td>
<td>47±11</td>
</tr>
<tr>
<td>Black Control</td>
<td>12</td>
<td>0.92, 0.08</td>
<td></td>
<td>233±38</td>
<td>142±58</td>
<td>153±29</td>
<td>52±18</td>
</tr>
<tr>
<td>Stroke-risk 1</td>
<td>16</td>
<td>0.75, 0.25</td>
<td></td>
<td>212±43</td>
<td>117±42</td>
<td>139±37</td>
<td>50±14</td>
</tr>
<tr>
<td>Stroke-risk 2</td>
<td>7</td>
<td>0.78, 0.22</td>
<td></td>
<td>210±32</td>
<td>118±37</td>
<td>135±26</td>
<td>51±16</td>
</tr>
</tbody>
</table>

Values are mean±SD. TC, total cholesterol; TG, triglycerides; LDL, low density lipoprotein; HDL, high density lipoprotein; stroke-risk group 1, subjects with >30% carotid artery stenosis in one or more locations; stroke-risk group 2, subjects with >30% stenosis in more than three locations.

*p<0.01, comparison of control vs. stroke-risk group 1 or 2 by Student's t test.
Kasturi et al  Approtein A-I-C-III Gene Cluster in White and Black Subjects


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