of diabetes and hypertension. We are concerned that the demographic features of the patient and control groups are not homogeneous, and may explain some of the differences they found.

We examined differences between the patients and control subjects using the numbers from Table 1 (although the number of patients with risk factors differs slightly from that in "Subjects and Methods"). Eighty-nine of 153 (58%) poststroke patients were hypertensive, compared with 14 of 69 control subjects (20%); this difference is very significant (P<.001, \( \chi^2 \) test). A similarly high proportion—32 of 153 (21%)—of patients were diabetic, compared with 0 of 69 control subjects (P<.001, \( \chi^2 \) test). Both hypertension and diabetes have been associated with elevated levels of PAI-1. We suggest that the elevation in coagulation factors in poststroke patients in this study may be due to certain stroke risk factors.

Although there are no statistically significant differences between the treatment groups, the numbers are small and the groups are not homogeneous. Differences in cholesterol and triglyceride levels may also contribute to differences in levels of coagulation-fibrinolytic markers.4,5 In addition, we do not know why patients were assigned to receive aspirin, ticlopidine, or no treatment. We would be cautious about drawing the conclusion that antiplatelet medication lowers PAI-1 levels on the basis of this nonrandomized study.

\[ \text{References} \]

of aspirin and ticlopidine before continuing long-term treatment. Our patients, however, were homogeneous in terms of infarct size and clinical severity.

As Dr Öztürk and colleagues state, further studies based on a large number of patients (with more appropriate controls) are necessary to determine the significance of the coagulation-fibrinolysis markers for stroke and stroke recurrence and the effects of antiplatelet medication on the markers.

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Reference

Apolipoprotein E ε4 Allele Frequency in Vascular Dementia and Alzheimer’s Disease

To the Editor:
The issue of apolipoprotein E (apo E) polymorphism has been receiving a great deal of attention in the neurological literature. It has been suggested in Stroke that the frequent ε3/ε4 phenotype may protect against vascular morbidity and that the ε4 allele might be a predisposing genetic marker for ischemic cerebrovascular disease. However, the ε4 allele has recently been reported to also be more frequent in patients with sporadic Alzheimer’s disease (40% in patients with Alzheimer’s disease compared with 10% to 16% of control subjects), raising questions about the specificity of ε4 as a risk factor for cerebrovascular or Alzheimer’s disease.

We assessed apo E allele frequencies in 51 elderly control subjects, 23 subjects with vascular dementia, and 93 patients with Alzheimer’s disease. Apo E phenotype was determined by isoelectric focusing in immobilized pH gradients and immunodetection by sheep anti-apo E antibodies. Frequency of the ε4 allele is higher in patients with Alzheimer’s disease (40%) compared with age-matched control subjects,2;3 raising questions about the specificity of the ε4 allele as a risk factor for cerebrovascular or Alzheimer’s disease.

We assessed apo E allele frequencies in 51 elderly control subjects, 23 subjects with vascular dementia, and 93 patients with Alzheimer’s disease. Apo E phenotype was determined by isoelectric focusing in immobilized pH gradients and immunodetection by sheep anti-apo E antibodies. Frequency of the ε4 allele is higher in patients with Alzheimer’s disease (40%) compared with age-matched control subjects, raising questions about the specificity of the ε4 allele as a risk factor for cerebrovascular or Alzheimer’s disease.

The association of the apo E allele with vascular disease has been repeatedly reported, and it should not be surprising that it appears more frequently in patients with vascular dementia than in control subjects. However, this casts doubt on its specificity as a risk factor for Alzheimer’s disease.

A unifying explanation of the coagulation-fibrinolysis markers for stroke and stroke recurrence and the effects of antiplatelet medication on the markers.

Letters to the Editor

Frequency of the Apo E Allele in Selected Groups

<table>
<thead>
<tr>
<th>Population</th>
<th>n</th>
<th>No. of Alleles</th>
<th>Age±</th>
<th>ε2§</th>
<th>ε3§</th>
<th>ε4§</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elderly</td>
<td>51</td>
<td>102</td>
<td>69.2 (3.6)</td>
<td>.10 (.02)</td>
<td>.72 (.05)</td>
<td>.18 (.04)</td>
</tr>
<tr>
<td>Young†</td>
<td></td>
<td>704</td>
<td>35.2 (5.7)</td>
<td>.04 (.00)</td>
<td>.86 (.02)</td>
<td>.10 (.02)</td>
</tr>
<tr>
<td>Patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td>93</td>
<td>186</td>
<td>73.6 (8.2)</td>
<td>.02 (.01)</td>
<td>.53 (.03)</td>
<td>.45 (.03) §</td>
</tr>
<tr>
<td>Vascular dementia</td>
<td>23</td>
<td>48</td>
<td>77.0 (7.5)</td>
<td>.00 (.00)</td>
<td>.54 (.07)</td>
<td>.46 (.07) §</td>
</tr>
</tbody>
</table>

*Mean (standard deviation); †Frequency (standard error).
§Data taken from Gabellini et al. §
$P<.0005$ on z-test for difference with controls.
Coagulation-fibrinolysis abnormalities in poststroke patients.
S Oztürk, D Bruck and W M Feinberg

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