allele frequency between control subjects and patients with amyloid-forming diseases such as Creutzfeld-Jakob disease, familial amyloidotic polyneuropathy, and Down’s syndrome. Vascular dementia is a poorly defined disease and its diagnostic criteria are still a matter of debate.3

We examined e4 frequency in 19 patients with mixed and vascular dementia (mean age, 81.7 years; SD, 9.5) and in 76 patients with Alzheimer’s disease (mean age, 78.4 years; SD, 7.7)4 by using a phenotyping technique.5 Our results are different from those of Frisoni et al (Table). Several factors might account for the discrepancy. First, the two groups in our study were small and interpretation of the results should be made cautiously. Second, the populations of the two studies had different mean ages. Third, the difference in findings could derive from the heterogeneity of cerebrovascular diseases that led to vascular dementia.6 Moreover, other environmental or genetic risk factors such as diet and hypertension should be taken into account. Far more patients need to be studied and additional clinical, biological, and genetic information will be necessary to determine whether the apo E e4 allele is indeed a specific risk factor for Alzheimer’s disease.

References
TABLE 2. Biosynthesis of Collagen in Cultured Skin Fibroblasts From Patients With Carotid Dissection and Controls

<table>
<thead>
<tr>
<th>Clinical Data</th>
<th>Procollagen Production*</th>
<th>Intracellular Hydroxyproline, †</th>
<th>Procollagen Type III/Type I, ×100‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell Line</td>
<td>(dpm/μg cell protein)</td>
<td>Percentage intracellular</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>[3H]hydroxyproline.</td>
<td></td>
</tr>
<tr>
<td>CD 3</td>
<td>412</td>
<td>13</td>
<td>14</td>
</tr>
<tr>
<td>CD 4</td>
<td>356</td>
<td>16</td>
<td>19</td>
</tr>
<tr>
<td>CD 5 b</td>
<td>260</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>CD 8</td>
<td>248</td>
<td>15</td>
<td>16</td>
</tr>
<tr>
<td>CD 20</td>
<td>376</td>
<td>17</td>
<td>16</td>
</tr>
<tr>
<td>CD 27</td>
<td>507</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>Controls (n=7)</td>
<td>322 ± 104</td>
<td>16±2</td>
<td>18±3</td>
</tr>
</tbody>
</table>

Experiments were carried out in triplicate from one or two separate labeling experiments.
*Procollagen production is expressed as radioactivity (dpm [disintegrations per minute]) of macromolecular [3H]hydroxyproline in culture medium per μg cell protein after a 24-hour labeling with [1H]proline.
†Percentage of intracellular hydroxyproline is calculated as the ratio of intracellular radioactive hydroxyproline/total radioactive hydroxyproline in cell culture.
‡Ratio of type III to type I procollagen was calculated by electrophoresis on polyacrylamide slab gels.

The results suggest that either heritable predisposition is a risk factor for carotid dissection or carotid dissection is heritable in some cases. Furthermore, the familial aggregation of carotid dissection and cerebral aneurysm we observed suggests that these disorders may have common pathogenetic factors.

References
Familial aggregation of cervical artery dissection and cerebral aneurysm.
K Majamaa, H Portimojärvi, K A Sotaniemi and V V Myllylä

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