Adenylate Kinase and Malate Dehydrogenase in Four Malaysian Racial Groups*

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Summary. Four Malaysian racial groups were typed for red cell adenylate kinase: 324 Malays, 300 Chinese, 256 Indians, and 483 West Malaysian Aborigines. The AK2 gene frequencies found were 0.015, 0.0, 0.086, and 0.013, respectively. All 244 Malays, 170 Chinese, 153 Indians, and 132 West Malaysian Aborigines examined had the common cytoplasmic malate dehydrogenase phenotype.

Zusammenfassung. Vier Rassengruppen aus Malaysia wurden auf Adenylatkinase-Varianten hin untersucht: 324 Malayen, 300 Chinesen, 256 Inder und 483 Eingeborene von West-Malaysia. Die Genhäufigkeiten des Allels AK2 waren: 0,015, 0,0, 0,086 und 0,013. Alle 244 Malayen, 170 Chinesen, 153 Inder und 132 west-malaysischen Eingeborenen, die daraufhin untersucht werden konnten, hatten den häufigen Phänotyp der cytoplasmatischen Malat-Dehydrogenase.

Fildes and Harris (1966) first reported electrophoretically-detectable genetic variation of adenylate kinase (AK) in human erythrocytes. The three phenotypes designated as AK 1, AK 2—1, and AK 2 subsequently were extended by Bowman et al. (1967) with AK 3—1 and AK 4—1. Davidson and Cortner (1967) first reported a genetic variant of cytoplasmic malate dehydrogenase (S-MDH). They found one heterozygous Negro in a survey of 1470 US Negroes and 1440 US whites, and Blake et al. (1970) found 10 persons in New Guinea to have a new variant, of whom 6 were related. In this paper we report AK and S-MDH findings in Aborigines, Malays, Chinese, and Indians of West Malaysia.

Materials and Methods

Haemolysate were prepared in the standard way from venous blood obtained from healthy persons (blood donors and employees) and patients from the General Hospital. 324 were Malays (293 healthy persons, 31 patients), 300 were Chinese (151 healthy persons, 149 patients), and 256 were Indians (188 healthy persons, 68 patients). Haemolysates were prepared according to Scott (1970) of blood collected in capillary tubes from finger punctures of 483 Aborigines of all ages admitted to the Ulu Gombak Hospital near Kuala Lumpur, which serves Aborigines from all of West Malaysia. Known relatives were excluded from the data.

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in this report. We verified that identical phenotypes were obtained with both methods of
blood sampling and haemolysate preparation. 244 Malays (213 healthy persons, 31 patients),
170 Chinese (83 healthy persons, 87 patients), 153 Indians (40 healthy persons, 113 patients)
and 132 West Malaysian Aborigines patients were also examined for red cell S-MDH pheno-
types. For the study of AK phenotypes we followed the electrophoretic method of Radam
and Strauch (1968) and the staining method of Fildes and Harris (1966), and for the study
of S-MDH we followed the method of Davidson and Cortner (1967).

Results and Discussion

All persons examined for S-MDH had the common S-MDH phenotype. Table 1
shows the results of the AK typing. No significant differences in gene frequencies
between patients and healthy persons were found. The AK² allele frequency is
highest in Indians and lowest in Chinese, and intermediate in Malays and Abo-
rigines. The AK² gene frequency of Malaysian Indians corresponds with other
reports of frequencies in Indians, ranging from 0.081 for Madras Brahmins to 0.130
for English Pakistanis (Ananthakrishnan and Kirk, 1969; Chan, in press; Das et al.,
1970; Rapley et al., 1968). Bajatzadeh et al. (1969) also found an AK² frequency of
0.127 in the Irish. The AK² frequency for Malays has been reported by Chan (in
press) to be 0.019 for Malaysians, and by Gordon et al. (1966) to be 0.035 for Malays
in South Africa. Only one AK 2—1 was found in the 645 Chinese thus far typed
for AK (Shih et al., 1968; Shih and Hsia, 1969; Chan, in press). All 662 Japanese
reported were AK 1 (Omoto and Harada, 1970). From these preceding reports and
from Giblett (1969, Table 15.1), it can be inferred that the AK² allele is less com-
mon than 0.01 in pure Africans and South American Indians, as well as in Chinese
and Japanese. However, it seems that not all Mongoloid populations have such
a low frequency, as Bajatzadeh et al. (1969) surprisingly found an AK² allele fre-
quency of 0.067 in Koreans.

Table 1. Types of adenylate kinase in West Malaysians

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>Phenotype</th>
<th>AK² gene frequency</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2—1</td>
</tr>
<tr>
<td>Aborigine patients</td>
<td>483</td>
<td>470</td>
<td>13</td>
</tr>
<tr>
<td>Malay patients</td>
<td>31</td>
<td>30</td>
<td>1</td>
</tr>
<tr>
<td>healthy persons</td>
<td>293</td>
<td>284</td>
<td>9</td>
</tr>
<tr>
<td>total</td>
<td>324</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chinese patients</td>
<td>149</td>
<td>149</td>
<td>0</td>
</tr>
<tr>
<td>healthy persons</td>
<td>151</td>
<td>151</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>300</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indian patients</td>
<td>68</td>
<td>57</td>
<td>11</td>
</tr>
<tr>
<td>healthy persons</td>
<td>188</td>
<td>155</td>
<td>33</td>
</tr>
<tr>
<td>total</td>
<td>256</td>
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