Haptoglobin Phenotypes Among Egyptians*

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Serum haptoglobins have been found to be useful genetic markers. The distribution of the different haptoglobin genes was found to vary in different parts of the world (Giblett, 1961). Thus Orientals and Eskimos were found to have a high incidence of the gene Hp^2 and Negroes a high incidence of Hp^1. Caucasians in continental Europe, Australia, and the United States have revealed an intermediate distribution of these 2 genes as have also the Australian aborigines. In studying American Indian populations both Blumberg, Allison, and Garry (1959) and Sutton, Matson, Robinson, and Koucky (1960) pointed out an increasing gene frequency of Hp^1 as one goes south.

The present study aims primarily at describing the distribution of haptoglobin phenotypes among Egyptians, which has not been done before. Some observations on haptoglobin patterns in certain hereditary disease states are also included.

Methods

Haptoglobin typing was done by Smithies' method (1959) using starch-gel electrophoresis. Sera were studied from 219 normal adults (blood donors), from 66 patients with hereditary CNS diseases, and from 53 of their normal relatives. Statistical analysis was done using the chi-square test.

The results are shown in Tables I and II.

Discussion

The relative distribution of the three common haptoglobin phenotypes (Hp^1-1, Hp^2-1 + Hp^2-2 modified, Hp^2-2) among normal Egyptians is 41%, 34.24%, and 61-64%, respectively; (gene proportion being 0.2123 for Hp^1 and 0.7877 for Hp^2). This distribution simulates prevalent patterns among Oriental races. Thus Sutton et al. (1960), in a study of haptoglobin phenotypes among 200 Japanese blood donors, found 35% and 58%, respectively. This pattern contrasts with that prevalent among several other African populations where the Hp^1 gene is the commoner, and among Europeans where Hp^1 and Hp^2 genes are approximately equal (Giblett, 1961).

It is of interest that no significant difference is found between the haptoglobin phenotypes in upper and lower Egypt. Since Egypt has been frequently invaded by foreigners, e.g. Asians (Persians, Arabs, and Turks) as well as Europeans (Greek, Roman, French, and British), Egyptians have had several chances for gene admixture. Any such admixture would be generally greater in the population of Lower Egypt (from Cairo downstream, i.e. the Nile Delta), being the part of the country facing the invaders from North or across the Sinai Peninsula. Upper Egypt (upstream from Cairo) is surrounded on both sides by vast areas of desert and mountains. The Turk and Arab have apparently contributed most to any genetic exchange with Egyptians on a common religion basis, Islam. A detailed survey of haptoglobin patterns in the inhabitants of the Sinai Peninsula may clarify this.

TABLE I

HAPTOglobin PHENOTYPES AMONG THREE STUDY GROUPS (NORMAL SUBJECTS, SELECTED PATIENTS HAVING HEREDITARY DISEASES, AND THEIR NORMAL RELATIVES)

<table>
<thead>
<tr>
<th>Study Group</th>
<th>Haptoglobin Phenotype</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1-1</td>
<td>2-1</td>
</tr>
<tr>
<td>I. Normals (Upper Egypt)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>37</td>
</tr>
<tr>
<td>Total</td>
<td>9</td>
<td>75</td>
</tr>
<tr>
<td>II. Normal relatives of disease patients in group III (sibs and parents)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>21*</td>
</tr>
<tr>
<td></td>
<td>(0.08)</td>
<td>(0.4)</td>
</tr>
<tr>
<td>III. Patients with CNS heredofamilial diseases</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>29†</td>
</tr>
<tr>
<td></td>
<td>(0.13)</td>
<td>(0.46)</td>
</tr>
</tbody>
</table>

* Figures between brackets represent proportions of haptoglobin genotypes.
† Including 1 case Hp^1-1 Johnson type.
‡ Including 1 case Hp^1-1 modified.
Table II

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Gene Frequency (estimate)</th>
<th>Expected Proportions</th>
<th>Normal Group</th>
<th>Disease* Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Observed</td>
<td>Expected</td>
<td>Observed</td>
</tr>
<tr>
<td>2-2</td>
<td></td>
<td>0-6205</td>
<td>135</td>
<td>135-9</td>
</tr>
<tr>
<td></td>
<td>Hpl* = 0-7877</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-1</td>
<td></td>
<td>0-3345</td>
<td>75</td>
<td>73-3</td>
</tr>
<tr>
<td></td>
<td>Hpl* = 0-2123</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-1</td>
<td></td>
<td>0-0450</td>
<td>9</td>
<td>9-9</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>1-0000</td>
<td>219</td>
<td>219-1</td>
</tr>
</tbody>
</table>

Chi-square test

\[ \chi^2 = 0-0023; p > 0-9 \]

\[ \chi^2 = 18-2228; p < 0-0002 \]

* CNS Heredo-familial diseases.

The incidence of ahaaptoglobinemia in the present study (0-73% out of total normals) contrasts very much with its incidence among various populations of the African continent where variable percentages amounting to 41% in West Africa were reported (Allison, Blumberg, and Rees, 1958; Sutton, Neel, Livingston, Binson, Kunstadter, and Trombley, 1959; Harris, Robson, and Siniscalco, 1958; Giblett and Steinberg, 1960).

Summary

A study of hapoglobin phenotypes among Egyptians indicates a high prevalence of the gene Hp*. The Hp* gene was found in a somewhat higher incidence in a group of patients with hereditary diseases of the central nervous system.

References


globin phenotypes among pure Turk and Arab populations would aid in clarifying the evolution of the present hapoglobin phenotypes as well as other genetic markers among Egyptians.

The data in Table I show a somewhat higher incidence of Hp\~1 in the hereditary CNS disease group than in the normal subjects, the unaffected relatives showing an intermediate incidence. Although the difference between the relatives and the normal subjects is not statistically significant \((p > 0.2)\) the higher incidence in the hereditary disease group is statistically significant \((0.025 > p > 0.05)\).

The expected proportions of the 3 hapoglobin genotypes in the normal group are in accordance with the Hardy-Weinberg law tested by the chi-square method where the gene frequencies are Hp\* = 0.2123 and Hp\~2 = 0.7877. The value of \( \chi^2 (\chi^2 = 0.0023; p > 0.9) \) shows a very insignificant deviation between the observed and expected numbers. In the CNS heredo-familial group however, the frequencies are different \((\chi^2 = 18.2228, p < 0.0002)\), and the observed proportions deviate conspicuously from the expected (Table II).

The implication of the significant relative increase in incidence of Hp\~1 phenotype among patients with hereditary CNS disease compared to normal subjects cannot be explained by available data, yet the evident increase in consanguineous marriage in several generations of families of those patients may be a perpetuating factor for an originally accidental association between the Hp\* gene and the gene(s) responsible for the CNS disease. However, any selective influence of those genes on different hapoglobins remains speculative.
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doi: 10.1136/jmg.3.4.279

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