## GENETICS

### Phosphoglucomutase Frequencies in Habbanite Jews and Icelanders

Two variants of human red cell phosphoglucomutase (PGM), genetically determined and detectable by starch-gel electrophoresis, were discovered by Spencer, Hopkinson and Harris[1].

In most populations three common phenotypes are found, corresponding to the homozygotes and the heterozygotes for a single pair of allelic genes, $PGM_1^1$ and $PGM_1^2$. Hopkinson and Harris[2] have discovered a number of further, rare, phenotypes, some of which are dependent on alleles at a second locus, and have also published the frequencies of phenotypes and genes in seven different population groups from England, the East Mediterranean area and Africa.

We are carrying out extensive red cell and serum group investigations of blood specimens from Icelanders and from the Habbanite Jews. The results of these tests will be published in full elsewhere. It should be mentioned that there is a preponderance of males among the Icelanders and that numerous near relatives are included among the Habbanites. These points will be considered in detail when the results are published in full.

It is unnecessary to discuss here the origin of the Icelanders, although there is no complete general agreement on this; their red cell and serum group frequencies, in any case, fit well into the pattern of Western Europe. The Habbanite Jews were a group of about 500 persons forming a strict isolate in the Hadhramaut in Southern Arabia. They recently emigrated to Israel where the investigation is being carried out. Their red cell group frequencies differ considerably from those of any other Jewish populations investigated, with the exception of the Falashas of Ethiopia, for they show a high frequency of numerous genes which are particularly common in African populations, including $R_0$, (*cDe*), *V* (*ce*$^s$), *He* (*Henshaw*), *Fy* (amorph) and *Js*$^a$.

Specimens from both populations were collected into anticoagulant and sent as air freight to London for testing. Tests for phosphoglucomutase variants were performed as recommended by Spencer *et al.*[1]. Only the common variants were found. The results are set out in Table 1.

Table 1. INCIDENCE OF PGM PHENOTYPES IN ICELANDERS AND HABBANITE JEWS

| | No. tested | Phenotype numbers and frequencies PGM 1 | PGM 2–1 | PGM 2 |
|---|---|---|---|---|
| Icelanders | 129 | 88 (0·682) | 35 (0·271) | 6 (0·047) |
| Habbanites | 222 | 49 (0·221) | 93 (0·419) | 80 (0·360) |

Table 2. FREQUENCIES OF *PGM* GENES (AFTER HOPKINSON AND HARRIS[2])

| | $PGM_1^1$ | $PGM_1^2$ |
|---|---|---|
| "English" | 0·764 | 0·235 |
| Greek | 0·693 | 0·307 |
| Iraqi Jews | 0·674 | 0·326 |
| Turkish Cypriots | 0·698 | 0·300 |
| Negroes: | | |
| (*a*) Living in England | 0·786 | 0·214 |
| (*b*) Yoruba, Nigeria | 0·758 | 0·239 |
| (*c*) Bantu, S. Africa | 0·792 | 0·202 |
| Icelanders | 0·818 | 0·182 |
| Habbanites | 0·430 | 0·570 |

In Table 2 are shown the frequencies of the two common genes at the $PGM_1$ locus found by Hopkinson and Harris[2], together with those calculated from our own results. It will be seen that frequencies of the phosphoglucomutase variants have hitherto been found to vary little. The frequency of the gene $PGM_1^1$ lies between 0·758 and 0·792 in all English and African populations tested, and somewhat lower, between 0·674 and 0·698, in East Mediterranean populations, including Iraqi Jews. Icelanders are now found to have a $PGM_1^1$ frequency of 0·822 which is the largest yet reported, while that of the Habbanite Jews is 0·430 which is much smaller than any previously reported figure. This may be related to the somewhat small frequencies found in other East Mediterranean peoples; it is unlikely to be derived from Africa, although it would be desirable to compare data from East African populations. It may, on the other hand, be the result of isolation —genetic drift, or natural selection in a specialized environment. The proportion of homozygotes is somewhat greater than expected on a random mating basis, and this suggests a considerable degree of inbreeding, but the genotype frequencies for all the other red cell and serum group systems are, broadly speaking, intermediate between those found in the East Mediterranean area and in Africa, with no indication either of extreme gene frequencies or of an excess of homozygotes. Attempts are, however, being made to secure specimens for comparison from other populations which have lived for a long period in the Arabian peninsula.

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[1] Spencer, N., Hopkinson, D. A., and Harris, H., *Nature*, **204**, 742 (1964).
[2] Hopkinson, D. A., and Harris, H., *Ann. Hum. Genet.*, **30**, 167 (1966).

## MICROBIOLOGY

### Mobilization of Transduced Tetracycline Resistance by the Δ Transfer Factor in *Salmonella typhimurium* and *S. typhi*

IT has been shown that transferable drug resistance factors (R-factors) are composed of two basically independent entities[1–5]: (1) genetic determinants for drug resistance; (2) transfer factors, which are linked to the determinants to form complexes which are transferred from donor to recipient cells by conjugation. In the absence of a transfer factor, resistance determinants are transmitted only in a linear fashion. In the absence of resistance determinants, the transfer factors have no resistance to transfer.

We suggested earlier that loss of mobility of an R-factor results from loss of its transfer factor[1]. This hypothesis was confirmed with R-factors for ampicillin and for streptomycin–sulphonamide resistance which had lost their mobility during transfer to *Salmonella typhimurium* phage-type 36 (= type 36)[1,2]. The original mobility of these R-factors was known to depend on the Δ transfer factor[1]. The absence of Δ in the type 36 recipient lines was indicated not only by the non-transferability of the resistances, but also by the lack of the characteristic phage-restriction pattern produced by Δ (refs. 1 and 4). Moreover, Δ could be introduced into the strains at the same frequency as into "virgin" strains devoid of both resistance and Δ, whereas Δ entry into strains which already carry it is reduced by a factor of $10^{-1}$ or less. When the strains carrying immobile resistance determinants were infected with Δ, the resistances became transferable and the phage-restriction pattern characteristic of Δ appeared[1,2].

An R-factor may lose its transferability in three ways: (1) by segregation during multiplication of the host strain; (2) during transfer to a new host; (3) during transduction to a new host.

Our earlier observations suggested that the immobility of the resistance in each case probably resulted from loss of the transfer factor, and that the postulate that the R-factor had lost its transferability because it had become integrated into the chromosome[6–9] was erroneous. It must be remembered, however, that at the time this postulate was made the essential independence of the transfer factor and the determinants it carried had not