

Table 1

		Histamine value ($\mu\text{g/g}$ wet tissue)	Mast cell count
Bullfrog	Mesentery	0.19 \pm 0.06	+ + + +
	Tongue	0.04 \pm 0.01	+ + +
	Liver	0.07 \pm 0.01	-
Rat	Mesentery	24.8 \pm 4.4	+ +
	Abdominal skin	29.8 \pm 3.1	+ +

Preliminary results obtained in the course of a comparative study of various animals in our laboratory suggest that mast cells are free of histamine throughout the teleosts and amphibia and that it is not present below the level of the reptilia.

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¹ Fujita, T., and Takaya, K., *Z. Zellforsch.*, **75**, 160 (1966).

² Arvy, L., *C.R. Soc. Biol. Paris*, **149**, 528 (1955).

³ Arvy, L., *C.R. Assoc. Anat.*, **42**, Reunion, 217 (1955).

⁴ Code, C. F., *J. Physiol.*, **89**, 257 (1937).

⁵ Juhlin, L., and Shelley, W., *J. Histochem. Cytochem.*, **14**, 525 (1966).

⁶ Falk, B., *Acta Physiol. Scand.*, **56**, suppl. 197, 1 (1962).

⁷ Padawer, J., *Z. Zellforsch.*, **75**, 178 (1966).

GENETICS

Haptoglobins, Transferrins and Serum Gamma-globulin Types in Malayan Aborigines

THE Malayan aborigines form a distinct ethnic group which is socially and anthropologically different from the three main population groups in Malaya—the Malays, the Chinese and the Indians. The aborigines live mostly in the jungle, follow their own customs and beliefs and have little contact with non-aborigines. They are plagued by diseases, the most common of which are malaria and tuberculosis. Genetically the aborigines are of particular interest. Studies have revealed high frequencies in this population of certain haematological abnormalities: haemoglobin E ranges from 8 to 50 per cent in different groups¹, glucose-6-phosphate (G-6-P) dehydrogenase deficiency from 8 to 23 per cent¹, and hereditary ovalocytosis is found in 12.3 per cent of aborigines examined². These high frequencies may be due to malaria or inbreeding or both. We now report the results of a preliminary study of haptoglobins, transferrins and serum gamma-globulin types in Malayan aborigines.

Blood samples obtained from various aborigine groups were frozen and shipped by air to San Francisco. Haptoglobin types were determined by starch-gel electrophoresis by the method of Smithies³. A highly sensitive benzidine solution was used to stain the haemoglobin-haptoglobin complex. In those instances when haptoglobin was not detected, the serum sample was run three times before ahaptoglobinaemia was diagnosed. Transferrin types were determined from the same starch gel by the autoradiographic method of Giblett *et al.*⁴, in which they are identified by the use of radioactive iron-59. At a later stage a method of micropurification, followed by electrophoresis, was also employed⁵. Typing for hereditary gamma-globulin (Gm) groups was performed by the inhibition of agglutination reactions using standard tube systems described by Fudenberg and Kunkel⁶.

Blood from 266 Malayan aborigines, 72 jungle fighters and 194 villagers and aboriginal hospital personnel was examined for haptoglobins. The aborigines were from different jungle areas, and 64 villagers had to be excluded from the study because they were related to persons included. Type 1-1 was found in six aborigines, 2-1 in

79, and 2-2 in 101, and haptoglobin was not detectable in sixteen. The gene frequencies for Hp¹ and Hp² were respectively 0.24 and 0.76. The frequency for haemoglobin E in the whole group was 34.0 per cent, for G-6-P dehydrogenase deficiency, 15.7 per cent, and for ovalocytosis, 12.0 per cent. Of the sixteen haptoglobin-negative persons, six carried haemoglobin E and ten did not; two were deficient in G-6-P dehydrogenase and fourteen had normal amounts in the erythrocytes; two had ovalocytosis and fourteen had not; one had malarial parasites and fifteen were uninfected at the time of study. From these data, malaria, haemoglobin E and G-6-P dehydrogenase deficiency did not seem to be the cause of the high frequency of haptoglobin-negative persons in this population group.

The same serum samples were also studied for transferrins. Among 202 sera from unrelated subjects, 196 contained only Tf C and six contained Tf CD. The Tf D resembled Tf D_{Chi} and Tf D₁. The exact identity of the Tf D is being studied at the Department of Zoology, University of Texas, Austin. The Tf D_{Chi} has been found to differ in amino-acid substitution from that of Tf D₁ (ref. 7).

The attention of population geneticists has recently been attracted to the examination of hereditary gamma-globulins. The Gm types, because of their typical Mendelian inheritance, have proved useful in both anthropological and genetic studies, and the frequencies of different phenotypes vary greatly from one population to another. The systems for the detection of hereditary Gm types have steadily increased in number and, like the blood group system, they have become more and more complex. We examined various Gm factors in the sera from Malayan aborigines, and results were as follows: results of tests for Gm (a) and (b) on 147 sera from unrelated persons were 99 per cent positive; of 72 tested for Gm f, 97 per cent were positive. Gm x was uniformly negative in 107 sera from unrelated persons, and negative results were also uniformly obtained for Gm-like (Gm-c) activity in 131 sera. This pattern differs from the patterns found in Caucasians, Negroes, Australian aborigines, Japanese and Chinese.

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¹ Lie-Injo, L. E., and Chin, J., *Nature*, **204**, 291 (1964).

² Lie-Injo, L. E., *Nature*, **208**, 1329 (1965).

³ Smithies, O., *Biochem. J.*, **71**, 585 (1959).

⁴ Giblett, E. R., Hickman, C. G., and Smithies, O., *Nature*, **183**, 1589 (1959).

⁵ Matson, G. A., Sutton, H. E., Swanson, J., Robinson, A. R., and Santlana, A., *Amer. J. Phys. Anthropol.*, **24**, 51 (1966).

⁶ Fudenberg, H. H., and Kunkel, H. G., *J. Exp. Med.*, **114**, 257 (1961).

⁷ Wang, An-chuan, and Sutton, H. E., *Science*, **156**, 936 (1967).