Table 1

Phenotype	No.	Percentage
$Gm(a+x+)$	201	24·2
$Gm(a+x-)$	272	32·7
$Gm(a-x-)$	358	43·0
$Gm(a-x+)$	1	0·1
Total	832	100·0

$Gm(a - x + b +)$ in 3 out of 7 sibs in the F_1 generation and in the serum of the single representative of the F_2 generation. This may well be explained by the assumption of a rare gene—or closely linked gene combination—*bx*, beside the frequent gene *ax*, the genotype of the father of the proband being *ax/bx*.

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Glucose-6-Phosphate Dehydrogenase Activity in Papuans

During a recent investigation into hæmolytic and other anæmias in Papuans the glucose-6-phosphate dehydrogenase (G-6-P D) activity of erythrocytes was estimated in 45 cases. Blood was collected into heparin, and on several occasions into acid–citrate dextrose solution, and dispatched by air to Melbourne, arriving the same day. The G-6-P D activity was estimated in the Biochemistry Department of the Peter MacCallum Clinic, Melbourne, by the method described by Hsia[1]. By this method normal readings are 150–210 units/100 ml. The enzyme appears to be relatively stable for at least a week, provided the cells remain intact. The highest values were obtained in cases of thalassæmia major, acute hæmolytic anæmia of undetermined cause, and congenital hæmolytic anæmia of unknown cause. There was no significant difference in the results obtained, whether A.C.D. solution or heparin was used as an anti-coagulant. The twelve subjects used as controls all gave readings within the accepted range of normality.

Three examples of deficient activity were found. The first was a Papuan adult male, who denied any possibility of racial admixture and appeared to be a typical Papuan from the Rigo district, a few miles from Port Moresby. He was suffering from an attack of hæmolytic anæmia of undetermined cause when first seen on June 12, 1960. Electrophoresis of the hæmoglobin showed no abnormality and the alkaline-resistant hæmoglobin test and the direct Coombs test were both negative. No G-6-P D activity was found at this time. This result was not due to inhibitors, because the addition of blood from a case of thalassæmia major (518 units/100 ml.) gave the expected values. About two months later 8 units/100 ml. of enzyme activity were found and in a further six months the result was 11 units/100 ml. Although the patient was given 30 mgm. of 'Primaquine' daily for 10 days no evidence of hæmolysis was obtained. He stated that his brother had died one year previously from an attack of what seemed to be hæmolytic anæmia. He denied that either of them had eaten beans or taken any drug apart from 'Camoquin' at any time.

The two other patients with low values of G-6-P D were children. One was suffering from thalassæmia major, for which his spleen had been removed. His G-6-P D values on three occasions were 56, 46 and 36 units/100 ml. The other child was suffering from anæmia apparently due to tuberculosis infection and the G-6-P D value was found to be 66 units/100 ml. It has not been possible to confirm this result.

It is probable that anæmia which is determined by genetical factors is not nearly as common in New Guinea as is anæmia due to parasitic and nutritional factors. However, the finding of thalassæmia[2] and deficiency in G-6-P D in the Territory suggests that further attention should be given to investigating genetical factors.

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VIROLOGY

Cyclic Events in the Viral Cycle

The polio virus is able to develop only within given limits of temperature. The simplest reason for this seems to be that low and high temperatures block viral development by altering the metabolism of the host cell. Yet mutants of the polio virus can be obtained which thrive at these low or high temperatures at which the original type is unable to multiply: the sensitivity of viral development to temperature is thus controlled by the genetic constitution of the virus. Moreover, it was discovered that one mutational event shifts the whole curve expressing the sensitivity of viral development to temperature towards the left or the right: psychrosensitivity decreases when thermosensitivity increases and vice versa[1].

The polio virus is able to develop normally if submitted to supra-optimal temperatures during the first part of the cycle only or to infra-optimal temperatures during the second part only. Infra-optimal temperatures, which do not block the second part, interfere with the primary phase whereas supra-optimal temperatures, which do not block the first part, interfere with the secondary phase. The polio virus is, in fact, able to complete its development at high and low temperatures both incompatible with viral multiplication, provided they alternate in the right sequence during one growth-cycle.

The effects of infra-optimal temperatures are increased by heavy water, the effects of supra-optimal temperatures decreased. In the presence of heavy water the whole curve expressing the sensitivity of viral development to temperature is shifted: heavy water decreases the apparent temperature[2].

The fact that a single mutation modifies at the same time in opposite directions the psychro- and