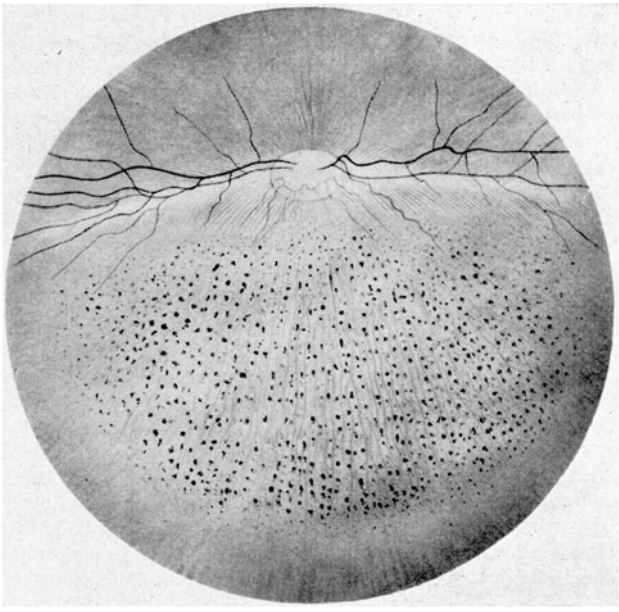

Fig. 1. Dégénérescence rétinienne expérimentale. Intoxication par le Fluorure Na (50 mg/kg, 2 injections).
Aspect du fond de l'œil après 3 semaines.

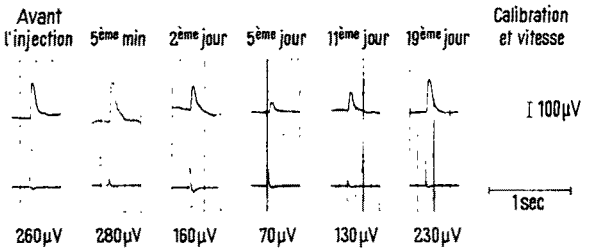

Fig. 2. Évolution de l'ERG dans les minutes qui suivent l'injection

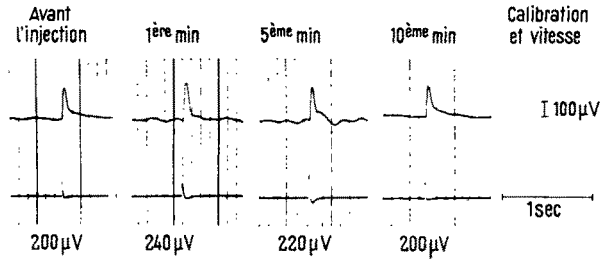

Fig. 3. Évolution de l'ERG dans les jours qui suivent l'injection. Diminution régulière du potentiel b pendant les 5 premiers jours, puis retour à la normale.

b) Quand l'œdème a disparu, la désintégration du neuro-épithélium se poursuit. A noter qu'elle reste localisée à la région sous-papillaire et respecte la périphérie. Il se forme des adhérences entre neuroépithélium et épithélium pigmenté, donnant un aspect d'arcades. L'épithélium pigmenté se modifie également: alternance de prolifération et raréfaction.

Pas de phénomènes inflammatoires infiltratifs ou exsudatifs. Les couches internes sont respectées.

Zusammenfassung: Die Einspritzung von NaFl beim Kaninchen verursacht Netzhautveränderungen, welche der *Retinitis pigmentosa* ähnlich (Fundus, ERG, Histologie), aber auf einen Bezirk beschränkt sind.

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The Incidence of Erythrocyte Glucose-6-Phosphate Dehydrogenase Deficiency in Singapore

Susceptibility to drug induced haemolytic anaemia is known to be associated with deficiency of glucose-6-phosphate dehydrogenase in erythrocytes (Carson, Flanagan, Ickes, and Alving[1]; Szeinberg, Sheba, and Adam[2]). This defect is common in American Negroes (Gross, Hurwitz, and Marks[3]) and amongst non-Askhenazic Jews (Szeinberg and Sheba[4]). Beutler, Yeh, and Necheles[5] recently studied 76 Chinese subjects by the glutathione stability test and suggested that the incidence of this defect is low in Chinese. However, several reports of haemolytic anaemia following ingestion of fava beans have appeared from China (Du[6]; Wang and Wang[7]; Chu, Ni, and Yang[8]). Chang[9] has reported on a clinical study of 228 cases of favism, while Bernard[10] has stated that: 'Favism is responsible for severe haemolytic anaemias with haemoglobinuria in many regions of China, mostly around Hang Chow'.

Between January and December 1959 an attempt was made to assess the frequency of this defect in Singapore. Blood samples were tested for glucose-6-phosphate dehydrogenase activity by the method devised by Professor A. G. Motulsky and Dr. J. M. Campbell of the University of Washington in which the dye brilliant cresyl blue is used as an indicator of the reaction:

$$\text{Glucose-6-PO}_4 + \text{TPN} \rightleftharpoons \text{6-Phosphogluconic acid} + \text{TPNH}_2.$$

Under standard conditions, a time of decolourisation longer than 120 min was taken as definite evidence of absent or deficient enzyme activity. Decolourisation usually occurred between 30 and 70 min. The method has been described in detail (Vella[11]).

In all, 673 blood samples from adult male blood donors were tested. The results found in this group of people are summarised in Table I. An enzyme deficiency was found in 2.50% of Chinese, 3.33% of Indian, 37.50% of Jewish and 0.65% of Malay subjects. The Jewish subjects had originated from Iraq and Persia. No instance of enzyme

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[9] Chang Hsiao-Hsiang, Chinese J. Pediat. *10*, 63 (1959).
[10] J. Bernard, Blood *14*, 605 (1959).
[11] F. Vella, Med. J. Malaya *13*, 298 (1959).

Tab. I. Incidence of glucose-6-phosphate dehydrogenase deficiency in Singapore

Ethnic group	Number studied	Number deficient	%
Chinese	240	6	2.50
Malay	155	1	0.65
Indian	132	4	3.33
European	80	—	—
Eurasian	27	1	—
Jew	16	6	37.50
Nepalese	14	—	—
Arab	4	—	—
Filipino	3	—	—
Turk	1	—	—
Puerto-Rican	1	1	—
Total	673	19	2.82

Tab. II. Incidence of glucose-6-phosphate dehydrogenase deficiency in Chinese hospital patients in Singapore

Age group	Number studied	Enzyme activity absent	%
Birth –7 days	29	13	44.89
8 days –4 weeks	22	8	36.35
5 weeks –6 months	49	8	16.32
7 months –12 months	10	—	—
13 months–10 years	43	6	13.95
11 years –75 years	44	9	20.45
Total	197	44	22.33

Tab. III. Incidence of glucose-6-phosphate dehydrogenase deficiency in non-Chinese hospital patients in Singapore

Age group	Indians		Malays		Nepalese		Jews	
	a	b	a	b	a	b	a	b
Birth –7 days	3	–	4	3	–	–	–	–
8 days –4 weeks	2	1	1	–	–	–	–	–
5 weeks –6 months	4	–	3	–	–	–	1	1
7 months –12 months	1	–	–	–	–	–	–	–
13 months–10 years	6	–	5	–	1	–	1	1
11 years –75 years	1	–	10	–	2	–	1	1
Total	17	1	23	3	3	–	3	3

[a] number studied. [b] number deficient.

deficiency was found amongst European 'white', Nepalese Gurkha, Arab, Filipino and Turkish subjects and only single instances amongst Eurasians and in a Puerto Rican.

A similar study, though on a smaller number of blood samples obtained from the umbilicial cord, revealed an enzyme deficiency in only two otherwise normal Chinese infants (VELLA[11]). The absence of an association between this enzyme defect and the presence of abnormal haemoglobins or the thalassaemia abnormality in erythrocytes has been demonstrated (VELLA[12]).

During the same period, 243 blood samples from hospital patients of both sexes were also tested for enzyme deficiency. These samples were obtained from individuals suffering from severe neo-natal jaundice, severe anaemia, haemolytic jaundice or haemoglobinuria. The material studied and the results obtained are summarised in Tables II and III. Of 51 Chinese infants hospitalised during the first four weeks of life 21 (i.e. 41.18%) were enzyme deficient, and in these the diagnosis that had been made was that of 'severe unexplained neo-natal jaundice' or 'severe unexplained neo-natal jaundice with kernicterus' (SMITH and VELLA[13]).

The same enzymatic defect was found in two Italian infants suffering from kernicterus (SEGNI[14]). Of 5 Malay infants studied during the same period of life, 3 were enzyme deficient and in them the clinical diagnosis was the same as that in the Chinese infants (WEATHERALL[15]). Amongst 146 Chinese patients tested after the first week of life, 23 (i.e. 15.75%) were enzyme deficient. In at least 7 of these instances, a haemolytic episode had followed the taking of Chinese medicines or western drugs. Of the remaining 41 samples, three had come from Jewish subjects and all three were enzyme deficient. A full report on one of these Jewish subjects has been published (VELLA and PHOON[16]).

There is no doubt that, contrary to the results of BEUTLER et al.[5], deficiency of glucose-6-phosphate dehydrogenase in erythrocytes is far from rare amongst Chinese subjects and in fact is present in a significant number of Chinese infants in whom a kernicterus syndrome develops in absence of any demonstrable maternal-foetal blood incompatibility, and in Chinese patients suffering from episodes of haemolysis. The Chinese subjects of Singapore and the Federation of Malaya emanated mainly from the southern provinces of China, while the Indians came mainly from the southern parts of India. Extensive surveys in various regions should help to elucidate the real incidence of this enzyme defect in south-east Asia.

Résumé. Chez des anémiques de sexe masculin étudiés à Singapour, l'auteur a constaté l'absence ou la déficience de l'activité de la glucose-6-phosphate déhydrogénase érythrocytaire dans les proportions suivantes: 2,5% sur 240 Chinois, 3,33% sur 132 Indiens, 37,5% sur 16 Israélites et 0,65% sur 155 Malais. L'absence de cette activité enzymatique était fréquente chez les malades chinois, malais et israélites hospitalisés pour anémie grave, ictère grave des nouveau-nés, anémie hémolytique ou hémoglobinurie.

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Department of Biochemistry, University of Khartoum (Sudan), January 12, 1961.

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[17] *Acknowledgements.* The work reported here was carried out while the author was a Lecturer in the Department of Biochemistry, University of Malaya in Singapore.
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