been described since then, but it had usually been assumed that the interference was due to occupation of cells by the first virus, leading to exclusion of the second virus. The interest of the present experiments is that the influenza virus remains in the lungs and cannot be demonstrated in the blood-stream, so that the resistance of the mice is probably due to interferon produced in the lungs and diffusing to the other organs via the blood-stream. An analogous explanation has been put forward to account for the observation of Burnet and Fraser (1952) that chick embryos infected with influenza virus in the allantoic cavity became resistant to the lethal action of the neurotropic influenza virus strain NWS. Grossberg, Hook, and Wagner (1960) found that a similar resistance could be induced by inoculating interferon into the allantoic cavity, and they suggested that the "interference at a distance" described by Burnet and Fraser was due to the production of interferon in the allantoic cavity as a result of infection with the influenza virus, followed by diffusion of the interferon into the chick embryo.

The present experiments do not allow us to tell which would be the best route towards the goal of protecting man against virus infections, but they point to a number of interesting avenues of exploration.

Summary

Mice were protected to a slight but statistically highly significant extent against the lethal action of an encephalitis virus. Two different methods were used. The first was by intraperitoneal inoculation of interferon, and the second by active infection with influenza virus. The influenza virus grows in the mouse lung and protects the mice apparently by inducing production in the animals' lungs of interferon, which then diffuses into the blood-stream. It is suggested that interferon may protect the mice by inhibiting the growth of the encephalitis virus without impairing the animals' natural bodily defences.

We thank Dr. C. H. Andrewes, F.R.S., for his advice and criticism, and Mrs. Valerie Clay-Peters and Mr. V. G. Law for their valuable technical help.

REFERENCES

- Burnet, F. M., and Fraser, K. B. (1952). *Aust. J. exp. Biol. med. Sci.*, **30**, 447.
 Findlay, G. M., and MacCallum, F. O. (1937). *J. Path. Bact.*, **44**, 405.
 Grossberg, S., Hook, E. W., and Wagner, R. R. (1960). *Fed. Proc.*, **19**, 408.
 Henle, W., Henle, G., Deinhardt, F., and Bergs, V. V. (1959). *J. exp. Med.*, **110**, 525.
 Ho, M., and Enders, J. F. (1959). *Proc. nat. Acad. Sci. (Wash.)*, **45**, 385.
 Isaacs, A., and Burke, D. C. (1958). *Nature (Lond.)*, **182**, 1073.
 — and Hitchcock, G. (1960). *Lancet*, **2**, 69.
 — and Lindenmann, J. (1957). *Proc. roy. Soc. B.*, **147**, 258.
 — and Westwood, M. A. (1959). *Lancet*, **2**, 324.
 Porterfield, J. S. (1960). *Bull. Wld Hlth Org.*, **22**, 373.

Four times as many overseas students as there were 10 years ago are now in Britain. The estimated total for 1959-60 is 47,500. More than half were living and studying in the London area. These and other facts are contained in a new and expanded edition of a handbook, *Overseas Students in Britain*, published by the Standing Committee of the London Conference on Overseas Students, which represents about 70 organizations concerned with overseas students and has a secretariat provided by the British Council. The booklet may be obtained from the London Overseas Department of the British Council, 3 Hanover Street W.1. (Price 1s. 6d., post free.)

STUDIES WITH ¹³¹I TRIOLEIN IN SOUTH AFRICAN BANTU AND WHITE SUBJECTS, AND IN PATIENTS WITH MYOCARDIAL INFARCTION*

BY

J. METZ, M.D. B.Ch.

A. ANTONIS, Ph.D., A.R.I.C.

I. BERSOHN, M.B., B.Ch., B.Sc.

AND

D. HART

South African Institute for Medical Research,
Johannesburg

Recent studies on the levels of blood radioactivity after the oral administration of ¹³¹I triolein have demonstrated significant differences between normal subjects and patients with ischaemic heart disease (Likoff *et al.*, 1958; Sellar *et al.*, 1959). Likoff *et al.* estimated the radioactivity in whole blood and in the trichloroacetic acid precipitable fraction after the oral administration of ¹³¹I triolein to normal males and to males who had suffered a previous myocardial infarction. Significant differences in radioactivity at the peak time and at the 24-hour interval were demonstrated in the two groups. In a similar study, Sellar *et al.* found the blood radioactivity of normal subjects after the oral administration of ¹³¹I triolein to be much lower than in patients with ischaemic heart disease.

Plasma levels of radioactivity after the oral administration of ¹³¹I triolein probably represent a complex resultant of the rates of fat absorption from the bowel and of clearance from the blood. The results in ischaemic heart disease and control subjects would therefore appear to reflect metabolic differences in regard to one or the other mechanism, or to both.

There are well-recognized differences in the dietary and disease patterns in the South African White and Bantu populations. While the consumption of fat and the incidence of ischaemic heart disease in Whites are probably comparable to those of the people of Western Europe and the United States, the average Bantu diet contains little fat (Walker and Arvidsson, 1954), and myocardial infarction is rare. Although this rare incidence of ischaemic heart disease among the South African Bantu may be a reflection of constitutional differences, habitual dietary factors, especially the type and amount of fat consumed, may also be of significance.

Material and Methods

In the present study the absorption and plasma clearance of a test dose of ¹³¹I triolein was followed in Bantu and White subjects having no evidence of ischaemic heart disease, and in White patients convalescing from an attack of myocardial infarction.

Bantu Subjects.—29 male hospital patients aged 40 to 60, convalescing from diseases not generally affecting absorption from the gastro-intestinal tract, and with no evidence of ischaemic heart disease.

White Control Subjects.—30 male hospital patients aged 40 to 60. There was no evidence of any disease affecting gastro-intestinal absorption, or ischaemic heart disease.

*This study was made possible by a grant from the Atomic Energy Board, Pretoria, South Africa.

Myocardial Infarction.—20 white males aged 40 to 60, convalescing from a recent attack of myocardial infarction, diagnosed by the characteristic clinical picture and electrocardiographic changes.

^{131}I triolein obtained from the Radiochemical Centre, Amersham, England, was diluted with olive oil to a concentration of $50\ \mu\text{c.}/10\ \text{ml.}$ The $50\text{-}\mu\text{c.}$ dose, together with 50 ml. of milk, was given orally to the patient in the fasting state. Part of the milk was used to rinse out the container, and the rinsings were administered. Residual radioactivity in the container was negligible on assay. After receiving the dose the patient was permitted the usual hospital breakfast, and there were no further dietary restrictions during the test period. Thyroid uptake of ^{131}I was blocked by administering three drops of Lugol's iodine three times on the day prior to the test, and a further three drops together with the test dose of ^{131}I triolein. Venous blood was withdrawn at 4, 6, 8, 12, 24, and 36 hours after administration of the test dose, and placed in heparinized containers. The radioactivity of 3–5 ml. samples of plasma was determined in a well-type scintillation counter containing a thallium-activated sodium iodide crystal. The counting-time varied with the activity of the sample, but all samples were counted to within at least 2% accuracy. With each batch of ^{131}I triolein used, part of the solution was retained as a standard, a dilution of which was counted with each assay of plasma radioactivity. This obviated the necessity for corrections for radioactive decay. The percentage of administered radioactivity per 1,000 ml. of plasma was calculated.

Results

The mean percentages of administered radioactivity absorbed per litre of plasma in the three groups at different time intervals after the dose was fed are shown in Table I and are presented graphically in the Chart. Table II shows the percentage of subjects reaching their maximum percentage absorption before and after four hours.

TABLE I.—Plasma Radioactivity after Administration of ^{131}I Triolein to Bantu and White Control Subjects, and to Patients with Myocardial Infarction.

Group	No. of Subjects		Percentage of Administered Radioactivity per Litre Plasma					
			4 hrs.	6 hrs.	8 hrs.	12 hrs.	24 hrs.	36 hrs.
Bantu	29	Mean	2.94	2.64	2.58	1.86	1.16	0.60*
		S.D.	1.6	0.9	0.7	0.5	0.5	0.3
White controls	30	Mean	3.16	3.38	2.88	1.92	1.04	0.40
		S.D.	1.7	1.5	1.4	1.1	0.6	0.3
Myocardial infarction	20	Mean	3.90	4.14	3.32	2.28	1.22	0.54
		S.D.	1.9	1.8	1.1	0.9	0.6	0.5

* 21 subjects only.

TABLE II.—Percentage of Subjects Reaching Their Maximum Percentage Absorption Before and After Four Hours.

	Before 4 Hours	After 4 Hours
Bantu	58.6%	41.4%
White controls	46.66%	53.33%
Myocardial infarction	50.0%	50.0%

Bantu and White Control Groups.—The mean levels of plasma radioactivity in the Bantu patients were lower than those of the White control group at 4, 6, 8, and 12 hours, but were higher at 24 and 36 hours. Only the differences at 6 hours and 36 hours were statistically significant ($p < 0.05$).

White Control and Ischaemic Heart Disease Groups.—The mean levels of plasma radioactivity in the

ischaemic heart disease group were higher than those of the control group throughout the 36-hour period of study. The differences, however, were not statistically significant.

Bantu and Ischaemic Heart Disease Groups.—Mean plasma radioactivity in the ischaemic heart disease groups was higher than in the Bantu patients at 4, 6, 8, 12, and 24 hours, but lower at 36 hours. The differences at 6 and 8 hours were statistically significant ($p < 0.01$ and at 12 hours $p < 0.05$).

Bantu and White Groups.—59% of the Bantu reached their maximum percentage absorption under 4 hours, while for both White groups 46.66 to 53.33% did so. This difference is not statistically significant.

Plasma radioactivity after oral administration of ^{131}I triolein to White and Bantu control subjects, and to patients with myocardial infarction.

Discussion

In the present study the total plasma radioactivity only was assayed, since separate assay of the trichloroacetic acid precipitable fraction has been shown to yield little additional information (Likoff *et al.*, 1958; Seller *et al.*, 1959). Activity was expressed per litre of plasma, as this appears to be a more valid index of the concentration of the fat in the blood than that based on a calculated blood volume as an arbitrary percentage of body weight.

Recent studies confirm the validity of metabolic studies with iodinated fat. In dogs, ^{131}I triolein administered intravenously as an emulsion disappears at the same rate as natural fats (Van Handel and Zilversmit, 1958). In view of the great similarity in clearing ^{131}I triolein and the carrier fats of the artificial emulsion, it is unlikely that ^{131}I is removed from the circulation at a rate different from that of the carrier fat.

Bantu and White subjects with and without evidence of ischaemic heart disease showed differences in their plasma radioactivity curves. Although the mean observed peak of the Bantu group was at four hours after the administration of the fat load, that of the White groups was six hours after the load. Examination of the

individual maximum recorded percentage absorption of the subjects, however, does not permit any significance to be attached to these differences, since many subjects in all three groups appeared to reach their maximum before the first reading was taken—that is, before four hours. Furthermore, in some cases, while the highest recorded percentage was at six hours, it was not possible to assess whether the maximum occurred at some time between four and six hours or at some time between six and eight hours. The number of cases in all groups who reached their maximum recorded percentage by four hours, and the numbers that reached the maximum after four hours, have therefore been compared (Table II). While 59% of the Bantu reach the maximum percentage absorption under four hours, only 46.66 to 53.33% of the White groups did so. There is no difference between the White controls and the ischaemic heart disease patients. The 10% racial difference may or may not be real, but on the available sample size it is certainly *not* statistically significant.

While the mean levels of radioactivity of both White groups were significantly higher at six hours than that of the Bantu, the slopes of the ascending parts of the absorption curves were almost identical for all three groups (see Chart). The absorption curves which reflect a resultant of both plasma absorption and clearance rates indicate, therefore, that not only are the initial rates of absorption significantly greater than the rates of clearance, but that the rates of absorption are very similar in all three groups. Differences in the heights of the peaks of absorption, and the times at which they are reached, may thus be a reflection of differences in the rates of clearance. Since the peak values for the Bantu subjects tend to be not only earlier but also significantly lower than those of both White groups, it could therefore be inferred that the Bantu have significantly faster fat-clearance rates than the White subjects.

At 24 and 36 hours after the load, the plasma radioactivity levels of the Bantu were higher than those of the White controls. The reason for this phenomenon is not clear, but may be a further indication of faster clearance rates in the Bantu, since the higher residual levels may be due to earlier feed-back of labelled lipids. In fact, recent studies on the composition of plasma fatty acids during alimentary lipaemia by Dole *et al.* (1959) suggest that the fatty acid pattern of chylomicrons in plasma is stabilized by equilibration with the larger pool of tissue lipids, this constancy of the fatty acid patterns being due to rapid recycling of the lipid components. Faster clearance rates of ^{131}I radioactivity with consequently more rapid equilibration of plasma and tissue lipids would therefore lead to earlier feed-back to labelled lipids, the net result being measurably higher radioactivity levels in the plasma. These higher residual levels in the Bantu may also be the result of liver dysfunction, a possibility at present under investigation.

Since ischaemic heart disease is rare among the Bantu it seems apparent that the absence of ischaemic heart disease may be associated with fast fat-clearance rates, or, conversely, that ischaemic heart disease may be associated with slower (and possibly impaired) fat-clearance rates. If the Bantu could be considered as the true normals, ischaemic heart disease would therefore appear to be partly due to an impaired rate of removal of fat from the plasma, and this would offer a possible explanation for recent findings that ischaemic heart disease is

associated with raised fasting serum triglyceride levels (Albrink and Man, 1959; Antonis and Bersohn, 1960).

Summary

Plasma radioactivity over a period of 36 hours after the ingestion of ^{131}I triolein has been studied in groups of older (40 to 60 years) Bantu and White subjects without evidence of ischaemic heart disease, and in patients suffering from myocardial infarction.

The Bantu subjects, in whom the intake of animal fat and the incidence of myocardial infarction are both low, show lower initial levels of plasma radioactivity when White subjects with and without ischaemic heart disease, but higher levels at 36 hours. The interpretation of these findings is discussed.

It is suggested that ischaemic heart disease may be partly due to an impaired rate of removal of fat from the plasma.

We thank the Director of the South African Institute for Medical Research for his interest in this study.

REFERENCES

- Albrink, M. J., and Man, E. B. (1959). *A.M.A. Arch. int. Med.*, **103**, 4.
 Antonis, A., and Bersohn, I. (1960). *Lancet*, **1**, 998.
 Dole, V. P., James, A. T., Webb, J. P. W., Rizack, M. A., and Sturman, M. F. (1959). *J. clin. Invest.*, **38**, 1544.
 Likoff, W., Berkowitz, D., Woldow, A., Jacobs, A. G., and Sklaroff, D. M. (1958). *Circulation*, **18**, 1118.
 Seller, R. H., Brachfeld, J., Sandberg, H., and Bellet, S. (1959). *Amer. J. Med.*, **27**, 231.
 Van Handel, E., and Zilversmit, D. B. (1958). *J. Lab. clin. Med.*, **52**, 831.
 Walker, A. R. P., and Arvidsson, U. B. (1954). *J. clin. Invest.*, **33**, 1358.

POLIOMYELITIS EPIDEMIC IN MAURITIUS AND THE EFFECT OF VACCINATION

BY

B. TEELOCK, M.B., Ch.B., D.P.H., D.T.M.&H.
Medical and Health Department, Mauritius

During June–September, 1959, a poliomyelitis outbreak occurred in Mauritius, 97 paralytic cases being admitted to the Princess Margaret Orthopaedic Centre.

Sporadic cases of poliomyelitis have been known in the island since a recognized outbreak in 1912. The disease became notifiable in 1925. There was a major epidemic in 1945, which was described by Seddon, Hawes, and Raffray (1946) and by McFarlan, Dick, and Seddon (1946). The attack rate in 1945 of about 2 per 1,000 was comparable with the 1916 outbreak in New York and the 1942–3 outbreak in Malta. In 1945, when Seddon, Dick, and McFarlan were in the island, they found a considerable number of old cases—one of them dating from 1891.

Another outbreak lasted from November, 1948, to February, 1949, and another one from March to June, 1952. Since then there have been a few cases every year except in 1957 and 1958, when no cases were notified.

State of Immunity of the Child Population before the Present Outbreak

In 1955 a polio immunity survey was carried out in the island with the assistance of Dr. J. H. S. Gear. The results of the tests made on 80 children under 10 years are shown in Table I.