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.

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POLIOMYELITIS EPIDEMIC IN MAURITIUS AND THE EFFECT OF VACCINATION

BY

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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.

TABLE I.—*Immunity Survey in Children up to 10 Years*

Age Group	Total	Type 1		Type 2		Type 3	
		+	—	+	—	+	—
1-5	37	21	16	23	14	27	10
6-10	43	35	8	38	5	29	14

In September, 1957, Canadian Salk vaccine was offered to all children aged between 1 and 5 years. In September, 1958, it was offered to all children between 1 and 2 years, and in April, 1959, also to all between 1 and 2 years. The routine was to give the first two injections at an interval of three to four weeks and the third injection after seven months. By the end of June, 1959 (the outbreak started while the vaccination campaign was still on but was nearing its end), (a) 43,395 children between 1 and 7 years had received three injections; (b) 35,603 children between 1 and 7 years had received two injections; (c) 20,330 children between 1 and 7 years had received one injection; and (d) 25,790 children between 1 and 7 years had not had the vaccine.

The Present Outbreak

The outbreak began on June 13, 1959. The first patient—a male child, aged 21 months, who had not had Salk vaccine—was admitted to hospital from the rural district of Pamplemousses, which is in the north of the island and adjoins the urban district of Port Louis. According to the mother, the child fell ill on June 13 with fever, was well the next day, and paralysis of the left lower limb developed on the 15th—that is, two days later. Previous to that, on May 4, the child was ill with slight fever, vomiting, and diarrhoea (seven or eight motions a day) necessitating admission to the local sugar estate hospital. He was discharged from hospital after eight days, apparently recovered, and, according to the mother, he remained well until the illness on June 13. His sisters, aged 3, 4, and 6 years, had received two injections of Salk vaccine in September–October, 1957, and were apparently well. No other case of illness occurred in the family during that time or the previous few weeks.

The second patient, a male child aged 10 months, was not admitted until July 10, though he had been ill since June 19. He came from Port Louis, the capital town, about 13 miles from the first case. The third patient, a girl aged 3 years, was admitted on June 25, having fallen ill on June 22; she came from the town of Beau Bassin, in the urban district of Plaines Wilhems, six miles from the second case. From then on, until September 12, 97 cases in all were admitted to the Princess Margaret Orthopaedic Centre from various parts of the island. The dates of onset of the illness and the ages of the patients are shown in Table II.

The youngest patient was 5½ months and the oldest 32 years. The number of cases in each of the nine districts is shown in Table III, and the island distribution of the cases is shown in the map.

It will be seen that no area of the country has escaped.

However, all the cases have been isolated individual ones, and no apparent connecting link has been found between them. One town alone (Vacoas), in the urban district of Plaines Wilhems, had 10 cases—that is, 10.3% of the total.

There being no isolation hospital for infectious diseases on the island, all cases were admitted to the orthopaedic centre under the care of the orthopaedic surgeons.

Diagnosis.—Lumbar puncture was done on the first few cases. Also stools and samples of blood from the first nine patients were sent for isolation of virus to Dr. J. H. S. Gear at the Poliomyelitis Research Foundation Laboratories, Johannesburg. Type 1 poliovirus was isolated from all the nine stool specimens; virus was not isolated from the specimens in baby mice.

Clinical Findings

In most cases the signs and/or symptoms at the onset were fever, headache, or pain in the neck, back, or one of the limbs. The majority of cases were mild. Six needed treatment in an iron lung, but only for a maximum of three weeks. There were no deaths. As to

TABLE II.—*Dates of Onset and Ages of Patients*

Date of Onset	No.	Age in Years										
		-6/12	6/12-	1-	2-	3-	4-	5-	6-	7-	8-	9-10+
June 7-13	1			1								
" 14-20	1		1			1						
" 21-27	1											
July 4	5		1		4							
July 5-11	10		2	3	3		1	1				
" 12-18	20		2	9	4	1		1				1
" 19-25	17	1	1	7	4	1	1				1	
" 26-												
Aug. 1	22		2	7	7	3		1	2			
Aug. 2-8	6		1	4	1							
" 9-15	7		1	1	3			1		1		
" 16-22	4			1		2						
" 23-29	0											
" 30-	1					1						
Sept. 5	1											
Sept. 6-12	2			2								
Total	97	1	11	35	26	9	3	3	2	1	2	0 4

TABLE III.—*Number of Cases by District*

District	Population (June, 1959)	No. of Cases
Port Louis (urban)	105,327	15
Pamplemousses	49,796	6
Rivière du Rempart	52,064	9
Flacq	71,315	11
Grand Port	64,180	11
Savanne	43,599	9
Plaines Wilhems (urban)	184,416	31
Moka	34,003	1
Black River	16,497	4

the site of paralysis, in 77.3% one or both lower limbs were affected, and in 27.9% the upper limb.

It is worth noting that before the outbreak was recognized, and even during the outbreak, errors in early diagnosis were encountered, and in 5 out of 80 cases analysed in detail the first diagnosis was acute appendicitis, acute rheumatism, osteomyelitis, suspected fracture of limb, and enteric fever respectively. These and other cases received injections of antibiotics or vitamins; but later, when paralysis had set in and the diagnosis was obvious, it was found that there was no real relationship between the site of injection and the site of subsequent paralysis.

Epidemiological Findings

A detailed analysis of 80 cases was made. No accurate estimate of the incubation period was possible. Very commonly, the mother of the child noticed only fever a day or two before paralysis set in. Typically, the paralysis was apparent when the fever had subsided and when the child could not use either one of the upper or lower limbs.

In spite of gross overcrowding in the households, and though in most cases before paralysis the patient had been sleeping with either one or both parents and/or with other children, there was not a single multiple case in the family. In the 1945 epidemic 24 multiple cases were seen out of 1,018 cases.

The attack rate in the different ages is shown in Table IV.

TABLE IV.—Attack Rate by Ages Per 100,000

0-	1-	2-	3-	4-	5-	6-	7-	8-	9-	10-	11-	15+
55.3	154.2	115.2	41.8	14.8	15.2	10.7	5.6	11.6	0	6.0	0	0.8

It will be seen that there are few adults and adolescents among the patients, and the term "infantile paralysis" is not a misnomer in this outbreak.

Course of the Outbreak

The weekly incidence of cases is shown in Fig. 1. It shows a rise from the third week, with a peak in the seventh to eighth week and with the outbreak over in 14 weeks.

The date of onset of the first case in each district is shown in Table V and the map.

TABLE V.—First Case in District

District	Date
Pamplemousses	June 13
Port Louis	" 19
Plaines Wilhems	" 22
Rivière du Rempart	" 28
Grand Port	July 7
Flacq	" 8
Black River	" 9
Savanne	" 20
Moka	August 9

FIG. 1.—Weekly incidence of poliomyelitis outbreak in 1959.

It will be seen that the only case in Moka district, in the central part of the island, occurred during the ninth to tenth weeks of the outbreak, and the first case in Savanne district (in the south of the island) occurred during the seventh week.

Preventive Measures

General.—No drastic measures which would increase panic were taken. Schools were not closed. They broke up for the normal August vacation on July 31 and remained closed until September 1. The school holiday camp, at the seaside, situated in the Pamplemousses district and receiving weekly batches of about 200 children aged between 10 and 16 years from all parts of the island, was allowed to run, but was under close medical supervision. The usual advice to avoid over-exertion, games, etc., was given to the public through radio and press. Advice was also given on the avoidance of injections, nose and throat operations, and sports meetings. Personal hygiene was encouraged, and the press and radio announced daily the number of cases notified. This helped to combat rumours.

Vaccination.—On August 14, Dr. P. D. Winter, of the Poliomyelitis Research Foundation, Johannesburg, arrived in the colony with Sabin type 1 poliovirus vaccine. After consultation, the Minister of Health decided upon a vaccination campaign with Sabin vaccine for all children between the ages of 6 months and 10 years. The Health Department opened vaccination centres, each in charge of a medical officer, and vaccination was begun on August 24—that is, 11 weeks after the outbreak started. There was a big response from the public, and in about 10 days some 195,000 children out of an estimated child population of 213,000 received Sabin live attenuated virus type 1.

Before the campaign began, samples of stools and blood from family groups, and blood from children under 6 years who had never received Salk vaccine, were taken at random from all parts of the island for virological studies in Johannesburg.

In all, 510 samples of blood and 202 specimens of stools were obtained before vaccination. Arrangements were made to collect post-vaccination blood and stools, at intervals, from the same individuals.

End of Outbreak.—Four cases were reported after the vaccination campaign started on August 24; these were the last cases of the outbreak.

Comparison with Previous Epidemics

The number of cases compared with those in the previous outbreaks, the months during which they occurred, the case rate, and the fatality rate are shown in Table VI and Fig. 2. It will be noted that there has been a diminished incidence and severity in each successive outbreak. The 1959 outbreak occurred during an unexpected time of the year—that is, the coldest months, as compared with all the previous ones, which occurred during the warmer months. Only the first major epidemic of 1945 had some relationship with a cyclone, as it started soon after a very violent cyclone had caused much damage.

FIG. 2.—Seasonal incidence in the four outbreaks.

The incidence of cases by district per 100,000 for these outbreaks is shown in Table VII, and the fluctuation within districts over the four outbreaks, standardized on

TABLE VI.—Incidence and Severity in the Four Outbreaks

Year	No. of Cases	Period	Case Rate per 100,000	Fatality Rate
1945	1,018	Feb.–April	240	About 6
1948–9	552	Nov.–Feb.	128	5.2
1952	284	March–June	56	1.0
1959	97	June–Sept.	15	Nil

TABLE VII.—Incidence of Cases by District Per 100,000

District	1945	1948–9	1952	1959
Port Louis (urban)	153.6	119.2	57.9	14.2
Pamplemousses	500.6	187.5	113.1	12.0
Rivière du Rempart	355.5	186.0	96.7	17.2
Flacq	185.4	217.9	65.0	15.4
Grand Port	285.6	112.1	79.6	17.1
Savanne	423.5	77.6	70.8	20.6
Plaines Wilhems (urban)	210.3	102.9	50.7	16.8
Moka	215.4	90.7	14.8	2.9
Black River	246.1	100.6	42.2	24.2

FIG. 3.—Fluctuations within district over the four outbreaks, standardized on that of 1945.

that of 1945, is shown in Fig. 3. In the present outbreak there seems to have been no correlation between the incidence of the disease and the density of population, and the incidence and distribution have been haphazard. However, the north of the island has been more generally affected in the different outbreaks.

In Table VIII the attack rates during the 1945 and 1952 epidemics are compared with those in the 1959 outbreak. These rates have been calculated only where accurate data were available. The tendency for older children to be affected, noticed in 1952 as compared with the 1945 and 1948–9 outbreaks, was not seen in the 1959 outbreak. In this outbreak 87.6% of the patients were under 5 years, and 95.8% under 10 years. The corresponding figures for the other epidemic years are shown in Table IX.

TABLE VIII.—Attack Rate Per 100,000 in the Different Ages in 1945, 1952, and 1959

Year	0–4	5–10	0–1	1–	2–	3–	4–	5–	6–	7–	8–	9–	10+
1945	1,070	Not available	400	1,490	1,230	1,230	1,300	1,190	860	340	170	80	Not available
1952	290	23.7	237	388	422	139	36						13
1959	92	8.8	55	154	115	41	14	15	10	5	11	0	0.94

TABLE IX.—Percentage of Cases by Age-group

Year	Under 5 Years	Under 10 Years
1945	64.5	95
1948–9	82.2	92.4
1952	77.7	83.4

The estimated population in the various ethnical groups in June, 1959, was: general population, 178,965; Indo-Mauritian, 420,107; Sino-Mauritian, 22,125. The attack rates per 100,000 population in the ethnical groups are shown in Table X, as well as the rates for 1948–9

TABLE X.—Attack Rate Per 100,000 in the Ethnical Groups in Three Successive Outbreaks

Year	General Population	Indo-Mauritian	Sino-Mauritian
1959	18.9	14.4	18.0
1948–9	72.3*	159.2	
1952	47.5*	77.3	

* Includes Sino-Mauritians.

and 1952. It will be seen that in 1959 the incidence was much the same in the various ethnical groups. In 1945 it was noted that the rate was higher among Sino-Mauritians (who are mostly shopkeepers) than in the other two groups.

As for the duration of the outbreak, the 1948–9 and the 1952 outbreaks, like the present one, also lasted about 14 weeks, with a peak in the 7th–8th weeks.

Influence of Salk Vaccine

The total estimated population on June 30, 1959, in the 1–7 age-group was 125,118. A total of 78 paralytic cases occurred in that age-group.

Among the 25,790 children in that age-group who were not vaccinated there were 72 paralytic cases, while there was only one paralytic case among the 43,395 children who had received three injections of the Salk vaccine. This paralytic case had received three injections in 1957–8. There were four paralytic cases among the 35,603 children who had received two injections of the vaccine: one had received the two injections in September–October, 1957, the second one in September–October, 1958, and the two others in May–June, 1959. In the latter two cases the children fell ill during the third week of July, 47 days and 38 days respectively after the second injection.

In the age group 1–7 years, therefore, the attack rate per 100,000 among those unvaccinated was 279.1 as compared with 2.3 among those who had received three injections. It is thus evident that Canadian Salk vaccine has offered considerable protection against paralytic poliomyelitis.

Influence of Sabin Vaccine

It will be noted (see Fig. 1) that by the time the vaccination campaign with Sabin vaccine had started—that is, the 11th week—the outbreak had practically come to an end.

Details of the four cases reported after the vaccination campaign started on August 24 are given in Table XI.

It will be seen that Case 1 was already ill when the mother took the child to be fed. In Case 2 also there was a suspicion whether the child was really well just before vaccination. The grandfather of the child had died post-operatively in the same house two weeks before the child fell ill, and there was crowding in the house for the funeral. In Cases 3 and 4 careful inquiry did not reveal any history of illness before vaccination. They were the last two cases of the outbreak.

TABLE XI.—Cases Reported After the Use of Sabin Vaccine

Case	Age	Date of Onset	Date of Feeding with Sabin	District
1	3 years	21/8/59	24/8/59	Port Louis
2	3 "	4/9/59	24/8/59	Pl. Wilhems
3	15 months	6/9/59	24/8/59	Savanne
4	21 "	6/9/59	24/8/59	"

How far the Sabin vaccine has influenced the course of the epidemic may be shown by the laboratory studies on blood and stools now in progress in Johannesburg. These studies will also help in establishing the amount of virus circulating in the community and the state of immunity of the population.

Summary

An outbreak of poliomyelitis due to a type 1 strain occurred in Mauritius during June–September, 1959, and lasted 14 weeks. There were 97 paralytic cases reported, and the attack rate was 15.6 per 100,000 population. There were no deaths.

The maximum incidence was in infancy, 75.5% of patients being under 3 years.

The outbreak occurred "out of season"—that is, in winter.

It started in one district and spread to all districts of the island, and the incidence and distribution within the districts were haphazard.

The incidence in those children aged 1–7 years who had received three injections of Salk vaccine was 2.3 per 100,000 and in those not vaccinated 279.1 per 100,000.

11 weeks after the outbreak started Sabin vaccine type 1 was given to about 195,000 children between the ages of 6 months and 10 years. There were no untoward effects after the vaccination.

I am grateful to Mr. P. Teckham, who was in charge of the orthopaedic centre during the outbreak, and his staff, for allowing me to study the patients, and to Mr. C. A. Bathfield, orthopaedic surgeon, for helpful criticism and advice. My thanks are due to Mr. L. Honoré, director of statistics, and to the Statistical Branch of the Medical and Health Department, for help with the statistical data. I am indebted to Dr. E. Losonczy, Director of Medical Services, for his encouragement and guidance and for permission to publish this report. This paper is intended to serve as a background to a subsequent report which will be published on the outbreak and on the large-scale use of Sabin type 1 vaccine.

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SERUM ENZYME ACTIVITY IN MUSCULAR DYSTROPHY*

BY

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1:6-Diphosphofructoaldolase is an intracellular glycolytic enzyme which is particularly abundant in muscle, and its function is specifically the reversible splitting of the molecule of D-fructose-1:6-diphosphate (F.D.P.) into equimolecular amounts of D-glyceraldehyde-3-phosphate (G-3-P.) and dihydroxyacetone phosphate (D.A.P.). It occurs in serum from normal individuals, where its activity may be altered by disease. The *transaminases* are also intracellular enzymes abundant in muscle; they operate the reversible exchange of α -amino groups, and also appear in the serum. The two transaminases examined here are serum glutamic oxalacetic transaminase (S.G.O.T.), which reversibly catalyses the conversion of aspartic and α -ketoglutaric acids to oxalacetic and glutamic acids, and serum glutamic pyruvic transaminase (S.G.P.T.), which similarly converts alanine and α -ketoglutaric acid to pyruvic and glutamic acids.

Warburg and Christian (1943) and Sibley and Lehninger (1949) reported the incidental finding of hyperaldolasaemia in cases of muscular dystrophy. Sibley later extended the scope of his investigations into the clinical significance of serum aldolase activity to include 880 patients suffering from a wide variety of complaints, among whom were five patients with primary myopathy (Sibley and Fleisher, 1954); he observed that while hyperaldolasaemia occurred in primary myopathy it did not do so in neurogenic muscular atrophy.

Meanwhile Schapira and his co-workers had begun a systematic study of several serum enzymes in progressive muscular dystrophy and related conditions, including aldolase (Schapira, Dreyfus, and Schapira, 1953) and S.G.O.T. and S.G.P.T. (Dreyfus and Schapira, 1955). All three enzymes were found to be elevated in the serum of patients with progressive muscular dystrophy but not in those with muscular atrophy due to disorders of the central or peripheral nervous system. Several workers have since confirmed these original findings (Jacob and Neuhaus, 1954; Beckmann, 1956; Aronson and Volk, 1956, 1957; Pearson, 1957; Evans and Baker, 1957; White and Hess, 1957; Rowland and Ross, 1958; White, 1959; Thompson and Vignos, 1959).

The assay of these serum enzymes has thus become an established procedure in the diagnosis of muscular dystrophy and in the clinical investigation of muscular weakness and atrophy of obscure origin. However,

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