The Luangwa Valley Plague Outbreaks and their Significance in Relation to Savannah Plague in Central Africa

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Early in 1956 plague reappeared in the upper Luangwa Valley, Northern Rhodesia, after a lapse of nearly forty years. A strain of P. pestis was isolated from a patient from Lundazi, which proved to be glycerol-fermenting. The Luangwa Valley lies in the extreme north-east of the territory near the Nyasaland and Tanganyika borders. This is the most southerly point from which the continental strain of P. pestis has been demonstrated. From the evidence available it appears that the Luangwa Valley focus is of long standing, and that the recent outbreak was derived from a persistent local enzootic focus.

In July 1956 there was a small outbreak in the extreme south of Nyasaland at Port Herald on the Mozambique border. Previous outbreaks in Nyasaland occurred in 1916-18 and 1939. During the period 1950-57, outbreaks in Tanganyika occurred in the northern parts of the territory, far removed from the Luangwa Valley.

Although there have been two small outbreaks since 1950 in Barotseland, efforts to identify the strain were unavailing. The question whether the Barotseland focus is connected with South Africa or Central Africa has thus not yet been settled.

Luangwa Valley outbreaks

Outbreaks occurred at two centres: one, about 100 miles (160 km) north of Lundazi in the Tembwe area, which is near the scene of the 1917-18 epidemic, and the other some ten miles (16 km) south of Lundazi. (In this connexion it should be noted that the focus of the 1917-18 epidemic marked on the map (Fig. 1, in Davis a) as S14 E30 (lower Luangwa Valley) should be S11 E32: Tembwe School appears on the 1:1 000 000 map of Northern Rhodesia, 11°20'S, 32°57'E.)

Tembwe outbreak (February-March 1956). A report of suspected plague from the African orderly in charge of the rural health centre at Tembwe reached the Medical Officer at Lundazi hospital towards the end of February. Within three days the Medical Officer and his staff were at the scene of the outbreak. There had been five deaths, four persons had recovered, and five were still sick. The first case was taken ill on 12 February. Gram-negative organisms resembling P. pestis were seen microscopically in gland aspirates from fourteen of the first series of patients and in smears from a rat found dead in a hut. The villages, and particularly the grain bins, in the neighbourhood were found to be heavily infested with rats and fleas. The patients were treated with sulfadiazine and streptomycin, and later with tetracycline. Vaccination of the inhabitants of the worst affected villages was carried out. The village huts were sprayed with BHC, and 50% DDT powder was dusted into chicken houses and grain bins. Warfarin baits were put out. Patients continued to be admitted, one or two per day, to the temporary hospital at the dispensary until the third week in March, coming in from upwards of fifteen small villages in the neighbourhood. When the Health Department team left at the end of the month the villages were reported to be free of rats and fleas. The particulars of the cases are summarized in the accompanying table.

It will be noted that all the cases, except two, were in infants or children and that the majority had cervical buboes. All the patients responded well to treatment, the clinical impression being gained that the response to tetracycline was more rapid than that to streptomycin and sulfadiazine. On the whole the cases were mild, temperatures on admission rarely exceeding 100°F (37.8°C).

The rat examined for plague was identified provisionally as Rattus rattus. Specimens of fleas collected were identified by an entomologist as Xenopsylla brasiliensis. No other information was obtained on the rodent and flea fauna. It may be assumed, however, that the hut- and field-rat species will prove to be very similar to those found

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* Note submitted to the WHO Expert Committee on Plague, September 1958.

CLINICAL PARTICULARS OF THE TEMBE PLAGUE OUTBREAK

<table>
<thead>
<tr>
<th>Incidence</th>
<th>Site of buboes</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age-group (years)</td>
<td>Sex</td>
<td>No. cases</td>
</tr>
<tr>
<td>-----------</td>
<td>-----</td>
<td>---------</td>
</tr>
<tr>
<td>0-4</td>
<td>M</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>4</td>
</tr>
<tr>
<td>4-14</td>
<td>M</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>15</td>
</tr>
<tr>
<td>15-24</td>
<td>—</td>
<td>None</td>
</tr>
<tr>
<td>25-49</td>
<td>M</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>M</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>20</td>
</tr>
<tr>
<td>M+F</td>
<td>36</td>
<td>5</td>
</tr>
</tbody>
</table>

a Including three fatal cases
b One patient, first treated with streptomycin, failed to respond but recovered on tetracycline.
c Including one fatal case
d A fatal case
e Died one month after discharge from dispensary

in Nyasaland and southern Tanganyika, about which some information exists (see below).

Lundazi outbreaks (April-May 1956). On 20 April a man was admitted to Lundazi hospital from Mukomba’s village, 10 miles (16 km) south of Lundazi. He had a high temperature and a left axillary bubo. He recovered rapidly on oxytetracycline and penicillin. Inquiries in the village revealed the death of a 14-year-old girl at the end of March with axillary buboes. The village was heavily infested with rats (and fleas) but no mortality among them had been noticed.

On 27 and 30 May, two women from a neighbouring village were admitted to the hospital, both of whom died soon after admission. Blood-agar cultures were obtained, which were sent to the Pathological Laboratory, Lusaka.

After several passages through white mice and guinea-pigs a subculture was sent to the South African Institute for Medical Research, Johannesburg, for confirmation. The strain proved to be fully virulent and was glycerol-positive. The strain was later sent to the Institut Pasteur, Paris, and Dr Girard kindly confirmed the glycerol reaction and informed us that it also reduced nitrites and thus belonged to the Continental strain.

Plague in adjacent areas

Barotseland, western Northern Rhodesia. The focus in the Upper Zambesi Basin is considered to have been derived from the south. This view is based on epidemiological, faunistic and geographical grounds. On the other hand, a case could be made out for a central African connexion, as a few rodent and flea species which seem to be associated with plague in central Africa occur there and not in South Africa.

As noted above, the glycerol reaction of the Barotseland strain has not yet been determined. Two small outbreaks have occurred in the area since 1950. An attempt to obtain a strain from the last outbreak in Balovale district in 1954 was unavailing. c

Nyasaland. In the light of events in the Luangwa Valley, the history of plague in adjoining Nyasaland takes on added significance. As in the Luangwa Valley, there was an epidemic during 1916-18, which took place at Karonga in the north near the southern border of Tanganyika. No plague was thereafter recorded until 1939, when a small outbreak occurred in the south of the territory in the Shiré district. In July 1956, a further outbreak took place in the extreme south of the territory. P. pestis was identified microscopically, but a culture was not isolated. The glycerol reaction of the Nyasaland strain would show whether this southern Nyasaland focus was established during the present pandemic from coastal sources or whether it is traceable to the north.

In this last outbreak there were 13 cases with two deaths in a group of three villages on the Mozambique border near Port Herald. Energetic measures stamped out the disease in a short time. Mortality was observed among domestic rodents in the small group of villages concerned, but not among wild rodents in the vicinity. The Portuguese authorities on the other side of the border reported no unusual rodent mortality or sudden deaths on their side, and it would seem that the origin of the outbreak was localized.

Lamborn in the course of his investigations of the Shiré outbreak carried out a rodent-flea survey in and around the affected villages in the Neno area. He found that the domestic rat (Rattus rattus) and the multimammate mouse (Rattus natalis) in the village huts were carrying Xenopsylla brailiensis and Echidnophaga gallinacea. From wild rodents, the pouched mouse (Saccostomus campesris) and the gerbil (Tatera schinzi shirensis), he obtained the same species together with X. syngenis. This fauna appears to be characteristic of what is likely to be found in the upper Luangwa Valley.

Tanganyika. Since 1950 the plague situation in Tanganyika has remained much the same as in the past with one exception: plague was recorded in the Paré mountains to the south of Mount Kilimanjaro apparently for the first time. Elsewhere in Tanganyika the pattern was much the same as in previous years as far as the northern part of the territory is concerned—that is, there was a recrudescence in the Singida-Manyoni districts of the Central Province and in the Mwanza area at the south end of Lake Victoria in Lake Province. There have been no further outbreaks in the older foci in the south of the territory in the Iringa area.

A strain of P. pestis was isolated at the Medical Laboratory, Dar-es-Salaam from a patient from Same, Paré mountains, which acidified glycerol.

The check-list of Tanganyika mammals by Swynnerton & Hayman gives a very fair indication of the geographical distribution of the species of rodents which may be concerned in the perpetuation of plague in the territory, and is of particular value in that it contains a gazetteer of place names of collecting localities which makes it a simple matter to plot the distributions given of the rodent species.

The Tanganyika Medical Department has begun an investigation of the fleas and rodents in the plague area and some of the specimens of fleas have been identified at the South African Institute for Medical Research by Dr Botha de Meillon, from collections submitted by the entomologist, Mr J. G. Halcrow, assigned to the work.

The collections made at Suji in the Paré mountains, though on a small scale, indicate that the rodent-flea fauna resembles that of the Belgian Congo, Uganda and Kenya plague foci. The following list summarizes the findings to date:

Arvicanthis niloticus: Dinopsyllus longifrons lypusus, Xenopsylla brasiliensis, Leptopsylla a. aethiopica, Cienophthalmus cabirus
Rattus natalis: D. l. lypusus, X. brasiliensis, L. a. aethiopica
Rattus rattus: X. brasiliensis, C. cabirus

The Red Locust Control Service became interested in the rodents of Central Rukwa, southern Tanganyika, as a result of a “plague” of multimammate mice (Rattus natalis) observed in 1955. As a spare-time occupation a number of fleas and small mammals were collected by Dr I. A. D. Robertson and Mr C. F. Chapman and sent to Johannesburg for identification.

Central Rukwa lies to the east of the western Rift Valley escarpment, which appears to act as a faunal barrier to certain species. The following records of fleas from the Rukwa rodents are based on these collections:

Arvicanthis niloticus: Xenopsylla bantorum
Saccostomus campesris: X. syngenis, X. rubica
Tatera taborae: X. rubica

Lamborn, W. A. (1940). In: Nyasaland, Annual report of the Medical and Sanitation Department for 1939, Zomba, p. 27.

Rodents from which no fleas have been collected and which occur in the area are the domestic rat (*Rattus rattus*), the multimammate mouse (*R. natatorial*), the gerbil (*Tatera schinzi* near *nyasae*), the tree-rat (*Rattus pedulecs*) and tree-mouse *Dendromus* sp.

With certain exceptions, such as *Tatera taborae* and *Arvicanthis niloticus*, the above host-species are likely to be found in the Luangwa Valley, some 200 miles (320 km) to the south-east.

**Discussion**

The data available are inadequate to come to any definite conclusions as to the mechanism of persistence of these central African foci of savannah plague. The upper Luangwa Valley is bounded on the east by the Luangwa-Nyasa highlands and it is possible that the sub-mountain stretch of country provides the combination of factors which enables *P. pestis* to survive and to erupt from time to time, during periods of high host population density. The year 1956 saw several instances of a general increase in numbers of wild rodents in central Africa. The Rukwa increase in multimammate mice in 1955 has been mentioned. In the following year reports of unusual rodent numbers were fairly general in Tanganyika and rodent damage to crops in Nyasaland was reported in the *Rand Daily Mail* of 7 April 1956.

The general ecological pattern appears to be essentially similar to that of the other remaining plague foci in Africa and Madagascar. Resolution of the problem will only follow a critical assessment of the vector capacity of the flea species and the degree of resistance of the rodent hosts in these persistent foci, whose association with upland regions with a tropical climate tempered by altitude points to a controlling climatic factor.

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We are indebted to the Directors of Medical Services of Northern Rhodesia, Nyasaland and Tanganyika for permission to make use of the information upon which this report is based and for their encouragement; and to Dr I. A. D. Robertson, Mr. C. F. Chapman and Mr J. G. Halcrow for the collections of fleas and other information.

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**An Unusual Strain of *Pasteurella pestis* Isolated from a Fatal Human Case of Plague**

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The authors first called attention some time ago to the unusual nature of a recently isolated strain of *Pasteurella pestis*. Since that time this strain has been studied intensively in at least five laboratories. It seems appropriate to review briefly our reasons for urging such a study and to emphasize the importance of specific fluorescent antibody for *P. pestis* in calling attention to the aberrant characteristics of this organism.

In October 1957 the writers received from Dr J. V. Irons, Director of Laboratories of the Texas Department of Health, a culture of an organism identified as *Pasteurella pestis*. The culture had been isolated at necropsy from a four-year old girl who had been infected either in Colorado or in Texas. Our study of the culture revealed that it was typical of the plague bacillus in respect of cultural, staining and physiological characteristics, lysis by bacteriophage, and susceptibility to antibodies. However, serological investigation employing both agglutination and fluorescent antibody tests indicated that this strain, named "Bryans", was quite different from representative strains of other virulent cultures of *P. pestis*. Detailed comparisons were made with four strains: Alexander, 16/P, PKR-76 and Sackacs, the latter originating from a fatal human case of plague