Rodents from which no fleas have been collected and which occur in the area are the domestic rat (*Rattus rattus*), the multimammate mouse (*R. natalensis*), the gerbil (*Tatera schinzi* (near) *nyasae*), the tree-rat (*Rattus paedulcus*) 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[f] 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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[f] Tanganyika (1957) *Annual report of the Medical Department for 1956*, Dar-es-Salaam, p. 11

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

by CARRIE C. WINTER, M.S., *Venereal Disease Experimental Laboratory, Communicable Disease Center, Chapel Hill, N.C., USA*, WILLIAM B. CHERRY, Ph.D., *Chief, Special Research Unit, Communicable Disease Center, Chamblee, Ga., USA, and* MAX D. MOODY, Ph.D., *Assistant Chief, Special Research Unit, Communicable Disease Center, Chamblee, Ga., USA*

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

reported in California in the summer of 1956.[a] These four strains were pathogenic for mice, produced morphologically discrete " envelopes " both *in vivo* and *in vitro* and were readily agglutinable by several different sera containing " whole cell-envelope " antibody (C. C. Winter and M. D. Moody —unpublished data, 1958). They also were agglutinable by *P. pestis* antisera manufactured by Lederle Laboratories or by E. R. Squibb and Sons. The Bryans strain resembled the other four in respect of the above-mentioned characteristics only in its pathogenicity for mice. It was dissimilar to the others in its failure to produce capsules either in the body of the mouse or in casein-hydrolysate/glucose/mineral medium at 37°C, and it was not stained by a fluorescein-labelled globulin containing " envelope" antibody. The organisms were stained moderately well, however, by fluorescent anti-somatic globulin. Agglutination tests employing " anti-envelope " and anti-somatic sera did not give reliable results, since the antigen suspensions were unstable and spontaneous agglutination occurred in the saline controls. These results were interpreted to mean that the Bryans strain was rough and devoid of or deficient in the " envelope " protein, although it was moderately virulent for CFW[b] mice. Since the presence of Fraction I (F-I) or " envelope " antigen in *P. pestis* may be associated quantitatively with immunogenicity and virulence,[c] it appeared that this strain, pathogenic for both the human and the mouse, was of considerable interest. We therefore urged that it be studied in those laboratories which have had maximum experience with *P. pestis*. Accordingly, a transfer of the Bryans strain was sent to Dr T. W. Burrows at the Microbiological Research Establishment in Porton, Wilts, England, and to Dr Karl F. Meyer at the University of California. The culture also has been studied at the Plague Laboratory of the Communicable Disease Center in San Francisco and at the US Army Biological Warfare Laboratories at Fort Detrick, Md. The detailed findings of the workers in the above laboratories will undoubtedly be reported elsewhere.

Judging from the reports which we have received, all workers agree that this strain presents some peculiar facets, although there is some disagreement about the ways in which these differences affect the behaviour of the strain. Dr Burrows[d] states that he was able to confirm our observations in regard to the instability of suspensions of the Bryans strain, its failure to produce capsules either *in vitro* or *in vivo* (mice), and its failure to be agglutinated by F-I specific antiserum. In respect of these properties, the Bryans strain was similar to his M24 and M30 strains, which were derived from a typical F-I+ strain. On the other hand, by serum agar diffusion methods, he was able to demonstrate in this strain, as in M24 and M30, the presence of material having F-I specificity. Thus, so far as we are aware, the Bryans strain represents the first natural occurrence of a strain possessing the above characteristics.

Dr K. F. Meyer (personal communication, 1958) has reported that he experienced some difficulty with the agglutination tests but was able to prepare suitable antigen suspensions. His tests also established the presence of F-I, but in considerably reduced amounts compared with other virulent strains. Dr Stuart F. Quan's findings at the Communicable Disease Center in San Francisco were similar, except that he apparently had no difficulty with the agglutination tests nor did he find the culture to be rough or unstable (personal communication 1958).

Other particuliarities of the Bryans strain have also been called to our attention—i.e., very low virulence for guinea-pigs (personal communications from Dr Burrows, Dr Meyer, Dr Quan and Dr G. M. Fukui), unusual characteristics of the Fraction II antigen (personal communications from Dr Meyer and Dr Quan), and possible deficiency of a major antigen (personal communication from Dr Burrows).

It is emphasized that the results of fluorescent antibody staining furnished one of the major clues to the bizarre nature of this strain. The low content of F-I antigen in the cells or perhaps absence of it at the cell surface seems to offer a logical explanation of the failure of this strain to be stained by the " whole cell envelope " antibody. Other cultures of *P. pestis* containing normal amounts of " envelope " antigen have consistently stained brilliantly with this reagent. On the basis of the above facts it seems fair to state that the Bryans strain is, indeed, an unusual isolate of *P. pestis*.

[a] United States of America, Department of Health Education, and Welfare (1956) *Communicable disease summary for week ended June 23, 1956*, Washington, D.C.

[b] The initials CFW stand for Carworth Farms Webster, a strain originally derived from Carworth Farms. Inc., New City, N.Y.

[c] Baker, E. E., Sommer, H., Foster, L. E., Meyer, E. & Meyer, K. F. (1952) *J. Immunol.*, **68**, 131.

[d] Burrows, T. W. & Bacon, G. A. (1958) *Brit. J. exp. Path.*, **39**, 278; and Burrows, T. W., personal communication, 1958.