EPIZOOTIC AMONG VELD RODENTS IN DE AAR AND NEIGHBOURING DISTRICTS OF THE NORTHERN CAPE PROVINCE.

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(With Appendices I–III by Dr J. H. Harvey Pirie, Dr W. F. Rhodes and W. Powell respectively.)

On November 5th, 1928, the magistrate, De Aar, telegraphed to this department at Pretoria:

> Reports of rats dying in town, 24 in one garden, 6 in another. Have sent specimens to Medical Research Institute, Johannesburg. Health Inspector cremating dead rats, which are infested with fleas.

De Aar is a town of some 1800 inhabitants—of which about 700 are Europeans—situated in the Northern Cape Province on the main railway line between Cape Town and Johannesburg, and 500 miles distant from each. It is also the junction of the railway lines to Port Elizabeth and East London on the east, and to Prieska and South-west Africa on the west.

A virulent wave of plague infection in veld rodents passed over De Aar and neighbouring districts in the latter part of 1924, the epizootic having spread from infected parts of the Orange Free State. During the course of the epizootic 29 cases of plague, with 18 deaths, occurred amongst the human population of the area. No evidence of active plague, either in man or rodents, has been reported from the area since.

Simultaneously with the magistrate's report, accounts of the epizootic appeared in the public press. It was stated that thousands of sick and dying rodents were swarming into De Aar, and that a great "trek" of rodents—from west to east—was in progress. A local medical practitioner reported that when motoring into the town he had "passed through droves of animals, numbering thousands, over a wide area." The municipality organised rat-killing gangs—equipped with sticks, lanterns and dogs—to operate at night on the outskirts and as far as possible prevent rodents entering the town.

Dr Laing, Assistant Health Officer in the department, was despatched to De Aar to investigate—Rodent-Inspector Chivers arriving a day or two later. Their inspections and enquiries showed that, although some of the press reports were rather exaggerated, a very active and virulent epizootic was in progress, involving a considerable area around the town, most of the invading rodents coming from the western side. They found the town and railway

premises practically free from "domestic" rats (*Rattus rattus*), but with a few house mice (*Mus musculus*) in some premises. Since the plague epizootic of 1924 the Namaqua gerbilles around the town had "bred up," and the commonage—especially on the western side—was extensively honeycombed with their burrows. A sudden and virulent epizootic had occurred amongst these. Gerbilles are strictly nocturnal and are rarely seen by man, but in the evening and at night animals could be seen in some numbers running about excitedly amongst the scrub around the burrows, with occasional dazed-looking rodents wandering aimlessly.

During the first two or three nights of the epizootic sick rodents had entered the town in considerable numbers—10 to 20 carcases having been found in several instances in one garden or premises or within a small area. Considerable numbers were found along the railway line on the side next the burrows —having apparently been too weak to surmount the rail; some which had succeeded in doing so were unable to get over the second rail and died between the metals. A railway ganger during one morning's patrol of his section picked up over 100 gerbille carcases along the line and between the rails. The gangs organised by the municipality killed 103 rodents during the first night, and about the same number during the second night of their operations. Several residents found carcases in their houses, some of these having doubtless been carried in by cats. The carcases were Namaqua gerbilles (*Desmodillus auricularis*) with an occasional long-eared mouse (*Malacothrix typicus*), and most of them were badly flea-infested.

The epizootic was at first suspected to be due to plague, and measures and precautions were taken accordingly. A vigilant watch was kept for sickness or mortality in cats or domestic mice, and for cases of suspicious illness in man, but nothing of this nature came to light.

The carcases sent to the South African Institute for Medical Research, Johannesburg, on November 5th, arrived much decomposed, but the micro scopic results were reported to be "highly suspicious of plague." Test inoculations of animals were made, but were eventually reported negative as to plague; tularaemia was also excluded. Further carcases were sent, with similar results.

A complicating factor was that undoubted plague existed in the neighbourhood of Petrusville, some 70 miles north-east of De Aar, where two fatal cases—one of bubonic type with terminal pneumonia, and one of pneumonic type—occurred in a coloured family during the week ended November 17th. The diagnosis in both these cases was confirmed by post-mortem and laboratory examinations, the District Surgeon, Dr M. Maciver, who performed the post-mortems, also unfortunately contracting the disease and having a typical attack of bubonic plague with axillary bubo, from which he recovered.

Inspections showed that the epizootic extended to a distance of 5 to 10 miles around De Aar, and probably further. The distribution of rodents in the outlying parts of the district was patchy, but in places the mortality had been

very heavy; in one place Rodent-Inspector Chivers picked up 40 carcases in a small area of veld.

The epizootic in De Aar lasted for a week or ten days, but with a further recrudescent wave about a fortnight later. A curious feature of its later stages was the presence of great numbers of white-breasted crows (*Corvus albus*) which had rapidly congregated and were very active in picking up and devouring the rodent carcases on the veld.

On November 13th it was reported that veld rodents were dying in large numbers at Burgerville—a village some 20 miles east of De Aar, numbers of carcases having been picked up, mainly along the water furrows.

During the two or three weeks following, similar reports were received from: Damfontein—15 miles south-west of De Aar; Houtkraal—19 miles north; Britstown—30 miles west; Vosburg—70 miles west; Victoria West—80 miles south; and several farms in the Britstown, Victoria West and Richmond districts. These outbreaks appeared to be similar in all respects to that at De Aar, except that the grouping of carcases along the water furrows (there are no such furrows at De Aar) attracted special attention. It was at first surmised that thirst was a symptom of the disease, inducing the sick animals to make for water, but further experience showed that, as in the case of railway rails, the location of the carcases was determined by the inability of the sick animals to cross the furrows. Very few carcases were found actually in the furrows.

The early reports of the magistrate, Victoria West, were very characteristic of this disease. His first telegram, dated December 8th, stated:

> Reported this morning that rats of unknown type found dead at several places in town, also in some dwelling-houses. Two specimens brought to me; matter very suspicious,

and at 2 p.m. same day he telegraphed:

> Further my telegram. Six rat carcases found gaol yard died since 9 o'clock this morning when yard was cleaned.

In all these outbreaks, carcases were sent for bacteriological examination and proved to be free from plague, but to present the same naked-eye and microscopic appearances as carcases from De Aar.

On November 29th the magistrate, Kenhardt, reported that veld rodents were dying on a farm "Pietersrust"—50 miles north-east of Kenhardt, and about 200 miles north-west of De Aar. On December 11th he further reported that 23 "mice" had been found dead on one acre alongside the water furrow on a farm "Tehaimoepslaagte," some 50 miles west of Kenhardt. (The term "mice" is often applied to various species of veld rodents, including gerbilles.) No carcases were sent for bacteriological examination in either of these outbreaks, but the circumstances suggest that they were due to the same disease as at De Aar.

Meanwhile, laboratory investigations were being prosecuted—by Dr Pirie, Assistant Director of the South African Institute for Medical Research,

Johannesburg, and Dr Rhodes, pathologist in charge of the department's laboratory at Cape Town. It was clearly established that the infection was not plague, but the determination of its precise nature proved a difficult and tedious matter. The first notable step towards the clearing-up of the matter was the discovery by Rodent-Inspector Chivers that the disease could be communicated by scarifying healthy gerbilles with material from infected carcases; incidentally, this discovery rendered it possible to maintain a supply of fresh material to the Johannesburg Institute and the Cape Town Laboratory —by sending batches of live gerbilles so scarified before despatch.

Early in December Dr Pirie spent a few days in De Aar and neighbourhood with the department's field officers, investigating the epizootic on the spot. Preliminary reports of the laboratory investigations made by him at the Institute, Johannesburg, and also by Dr Rhodes at the department's laboratory in Cape Town, are attached (Appendices I and II).

On December 15th the District Surgeon, Brandvlei, Calvinia district, some 200 miles west of De Aar, reported:

> We found mice and hares exceptionally plentiful at Swartkop (a farm some 12 miles south of Brandvlei), but dying in large numbers; they appeared lacking energy as if they were sick.

Carcases were sent for examination and were found free from plague; definite cause of death not established.

On January 28th, 1929, the magistrate, Calvinia, reported that "veld rodents were dying by scores" on farms "Welgemoed" and "Katjeskolk" in his district—some 50 miles west of Calvinia, and about 30 miles south of Swartkop. This epizootic was investigated by Chief Rodent-Inspector Powell, who reached the locality as the wave of infection was subsiding. A report of his observations, and his views on the differentiation of this disease from plague, is attached (Appendix III). Carcases of Namaqua gerbilles were sent to Cape Town and examined, with negative results as to plague. No carcases of hares were found. Identity of the infection with that of the De Aar epizootic was not established, but the collateral facts leave no reasonable doubt on the point. A wave of plague in veld rodents, with a few "secondary" cases amongst the human population, passed over the Calvinia district during 1926 and 1927.

From the information now available this epizootic appears to have involved a tract of country measuring about 130 to 150 miles in a north-and-south direction, and some 250 miles in an east-and-west direction. Apart from a few small towns and villages, the area is very sparsely populated. The country is, for the most part, sand veld with sparse scrub—the veld rodent population being mainly Namaqua gerbilles, but with Karroo rats (*Parotomys*), striped mice (*Rhabdomys pumilio*), and long-eared mice (*Malacothrix typicus*) also plentiful in places. Namaqua gerbilles were the animals almost exclusively affected and it would appear that their numbers have been greatly reduced, so that the risk of a plague epizootic occurring amongst them within the next year

or two is now much less than it was. It was reported that hares (*Lepus capensis*) and Karroo rats have died out in places, but this has not been conclusively established, and no carcases of these animals dead of the disease have been found. Mortality in domestic mice was reported from two places in the area—namely at Paarde Vlei station, 8 miles north of De Aar (where two house mice were found dead in a cottage), and in outbuildings on the farm "Doornfontein," 6 miles west of Petrusville. In the former case the carcases proved free from plague; in the latter the carcases were too decomposed for examination, but there were reasons to suspect plague in the locality.

The accompanying reports throw considerable light on the nature of the disease, but several important points still remain to be cleared up. Its mode of spread in nature has not yet been established; the present probabilities seem to be that fleas are not the agents, and that spread occurs through the healthy getting bitten and their wounds contaminated by the sick. When these matters and the seasonal, climatic and other conditions favouring spread are determined, it will be possible to form some opinion as to the possibility of utilising the infection for the purpose of destroying rodents by starting an epidemic—say, by releasing in suitable places a number of susceptible animals which have recently been scarified with infective material. In any case, the facts already elicited are of great scientific interest, and may prove to have important practical applications.

PRETORIA,
April, 1929.

APPENDIX I.

A VELD RODENT EPIZOOTIC DUE TO A *PASTEURELLA* OTHER THAN *PASTEURELLA* (*BACILLUS*) *PESTIS*.

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INTRODUCTION.

ON November 7th, 1928, five specimens of Namaqua gerbilles (*Desmodillus auricularis*) were received at the Institute from the Medical Officer of Health, De Aar municipality, with the information that these rodents were dying in great numbers in the town and in its vicinity. The following report was sent regarding them:

> Three of these five gerbilles are too decomposed to yield reliable information. Of the other two, microscopic examination of the spleen and heart blood of one shews some organisms suspiciously like *B. pestis*; the other shews similar suspicious organisms in such large numbers that we would advise that the cause of death be regarded as plague pending fuller confirmation by the biological tests which have been made.

The usual routine scarification and inoculation tests were made on guinea-pigs from the heart blood of the two suspicious gerbilles, but the guinea-pigs remained perfectly healthy.

During the remainder of the month and in the early days of December, 1928, five more batches of small rodents were received from De Aar and neighbouring districts. In all, there were 38 Namaqua gerbilles, 2 house mice, and 1 dwarf mouse (*Leggada* sp.). Some of these were too decomposed to be of much value, but the majority were in a reasonably fresh condition. Out of these 41, only 2 Namaqua gerbilles showed scanty *Pasteurella*-like organisms in spleen and heart blood smears, and all biological tests on guinea-pigs were negative. Two other gerbilles showed numerous Gram-positive diplococci in the spleen, heart blood, etc., suggestive of a pneumococcal septicaemia, but further investigation of the organism showed it to be a member of the *Streptococcus faecalis* or *Enterococcus* group which was not pathogenic for mice. Probably therefore this organism may be regarded as a secondary invader.

After the epizootic had lasted a month we were no further ahead in discovering its cause, although it seemed fairly clear that it was not plague. In the meantime the extent of the epizootic caused considerable alarm in the neighbourhood, and the occurrence of two undoubted cases of human bubonic plague in Petrusville did not help to allay the alarm.

On December 10th, 1928, I left Johannesburg for De Aar to make investigations on the spot, and spent the following four days there or in the vicinity. During these four days I had an opportunity of examining a number of Namaqua gerbilles in perfectly fresh condition, just after they had been picked up dead on the veld; also other gerbilles and some large-eared mice (*Malacothrix typicus*) which had been scarified by Rodent-Inspector Chivers with material (spleen and heart blood) from Namaqua gerbilles picked up dead.

During the remainder of the month of December I received in Johannesburg further supplies of Namaqua gerbilles found dead, and others—living and dead—which had been scarified on the spot by Mr Chivers and sent off alive.

I also brought back to Johannesburg with me a stock of live, apparently healthy, Namaqua gerbilles which were used mainly to keep the disease going in the laboratory.

This report is compiled from observations made at De Aar and in the laboratory prior to and subsequent to my visit there.

UNITY OR DUALITY OF THE EPIZOOTIC?

The title of this communication indicates that the epizootic was caused by a *Pasteurella*, and I think it will be conceded from the evidence to be adduced that part of the epizootic, at all events, was so caused, but I consider that it must still be regarded as an open question whether or not the whole epizootic was due to this *Pasteurella*.

The reasons for accepting the *Pasteurella* as the causative organism of, at any rate, a part of the epizootic, are as follows:

(1) The organism was frequently found, and often in great numbers, in the blood and organs of animals found dead.

(2) From such animals found dead, other suitable rodents could be killed by scarification with blood or organs, and the rodents so killed presented appearances identical with those of the animals found dead of the natural disease.

(3) The organism could be grown on laboratory media and inoculation or scarification of suitable rodents with the organism in pure culture reproduced the disease in a manner similar to that seen in the case of animals infected by scarification directly from animals dead of the natural disease.

The reasons for questioning whether or not the *Pasteurella* was responsible solely and entirely for the epizootic are as follows:

(1) Numerous gerbilles were examined in which the organism was not seen in smears of blood or organs, nor did it appear in cultures. This applies not only to animals sent up to Johannesburg, but also to some examined perfectly fresh on the spot and found under conditions which made death from anything other than natural causes very unlikely.

(2) Scarifications from such gerbilles were not fatal to others in the way that scarifications from gerbilles showing the *Pasteurella* were fatal.

A priori, of course, it is improbable that the epizootic had a dual nature, and I put forward the hypothesis that the gerbilles not showing the *Pasteurella* did nevertheless die from its effects, but that they failed to show it because of a partial, although imperfect, immunity.

The chief evidence in favour of such a hypothesis is that when Lobengula gerbilles (*Tatera lobengulae*) from Johannesburg were scarified with animals showing the *Pasteurella* or infected by scarification or subcutaneous inoculation with pure cultures they frequently died, but the *Pasteurella* in most cases was not to be seen nor was it recoverable on culture. However, until this hypothesis is more firmly established, the possible dual nature of the epizootic should, I think, be borne in mind, or else some other mode of action of the *Pasteurella* must be postulated.

Pathological anatomy of the disease.

The naked-eye morbid appearances are not as a rule very striking. The most constant feature is a moderate degree of general subcutaneous congestion reminiscent of that commonly seen in plague, but not generally so well marked as in that disease. The subcutaneous gelatinous oedema so common in plague has only been seen once, and then only to a slight extent, in a white mouse experimentally infected. Subcutaneous haemorrhages are not uncommon. In the experimental disease these, when present, are mostly small and in the neighbourhood of the site of infection or of the corresponding lymphatic gland. In the animals found dead in nature some fairly extensive haemorrhages were

seen, mostly on the sides of the chest, and in one gerbille there was also a large blood clot in one pleural cavity. Some of the cases showing haemorrhages were animals in which the *Pasteurella* was not found. Bubos were not observed in any of the cases of the natural disease, but in the experimental disease a slight soft swelling of the nearest gland to the site of infection was the rule, usually with some haemorrhage in or around the gland.

Internally some degree of congestion of the lungs is usually to be observed, and the spleen is also darker in colour than normal and commonly slightly enlarged.

Bacterioscopically no organisms may be visible, or, on the other hand, a Gram-negative, bipolar-staining *Pasteurella* type of organism may be present scantily or in enormous numbers in smears of the heart blood, spleen, liver and lungs.

Other organisms may also be present, either in scanty numbers or in abundance. The two types most commonly noted were an enterococcus and a coliform bacillus whose characters were very close indeed to those of *B. coli*. These are very probably only secondary invaders, but experience of the experimental disease suggests that invasion of the blood by them commonly takes place before death.

THE CAUSATIVE ORGANISM.

The organism believed to be responsible definitely for part of the epizootic and possibly, although not quite certainly, for all of it, is one whose characters—morphological, biological and pathogenic—place it amongst the *Pasteurellae* or haemorrhagic septicaemia group of bacteria.

As seen in blood or tissue smears from infected animals, it is a round-ended, Gram-negative, bipolar-staining bacillus or cocco-bacillus. Like *B. pestis* it varies considerably in size and shape, but it can be said that the average size is less than the average size of *B. pestis*. Many individuals, however, would easily pass for plague bacilli, and the initial mistake of regarding the first Namaqua gerbille examined as probably plague-infected was, I think, excusable. Now that one is familiar with the organism, the whole picture would probably raise one's suspicions that it was not plague, but even yet a certain diagnosis could not be made on bacterioscopic appearances alone but would still have to rest on cultural and biological tests.

The bipolar-staining of the organism is usually quite distinct with Gram counter-stains such as fuchsin or neutral red, but is beautifully brought out by such stains as thionin blue or Leishman's stain.

In addition to the cocco-bacillary forms, some longer, thinner forms are usually present, and many of these do not show bipolar-staining but stain uniformly throughout.

In cultures on ordinary agar, pleomorphism is a very striking feature. Even in a 24-hour-old culture of the first generation from an animal there is a greater variation in size and shape of individuals, and, as is the case in

plague cultures, bipolar staining is less distinct than in the tissue organisms. In older cultures and in further sub-cultures "involution forms," similar to those seen readily in plague cultures grown on salt agar, become numerous. There is a general tendency to enlargement, and big globular or pyriform bodies are common.

The organism is non-motile.

Cultural characters.

Agar slope. Grows readily but never forms a thick growth. 24-hour cultures show small, clear, dewdrop-like colonies, but these are less shiny and moist than in the case of a plague culture of corresponding age. In older cultures there is very little increase in size of the colonies, but they become drier, duller and more opaque.

Gelatine stab. Slight growth on the surface around the stab and along the upper part of the stab. No liquefaction.

Egg media and serum media. Growth similar to that on agar but rather more luxuriant, and colonies tend to be rather larger. No liquefaction. Growth on these media also emulsifies more readily in normal saline than the growth on agar. The growth on the latter, although not so sticky that threads can be drawn up as in the case of plague cultures, is somewhat adhesive and does not emulsify uniformly with ease.

Broth. Rather granular growth with heavy deposit. Slight pellicle formation and only slight general turbidity.

Potato. No growth.

Litmus milk. No change.

Indol formed.

Fermentation tests. Acid but no gas in dextrose, saccharose, mannite, and inulin. No change in lactose, maltose, laevulose, galactose, raffinose, salicin or dextrin.

Facultative anaerobe.

Pathogenicity.

The matter of the pathogenicity of the *Pasteurella* at first presented considerable difficulty to me in the way of accepting it as the cause of the epizootic, but in the end I have come to the conclusion that the difficulties could be quite simply accounted for and that the organism could be regarded as the true cause of the epizootic even if all its modes of action are not completely cleared up.

Namaqua gerbilles. The main incidence of the disease fell on this species, so naturally its reaction to the *Pasteurella* is of great interest.

Experimental scarification of these gerbilles with blood or organs of animals dead of the natural disease and showing *Pasteurellae* in the blood was frequently, but not always, successful in transmitting the disease. Such scarified animals often died within 24 hours, usually within 48 hours, but occasionally one would live as long as 96 hours.

Scarification with material from animals found dead in nature, but not showing *Pasteurellae* in the blood, was never fatal.

One gerbille which had been scarified directly from a dead animal and had also partially eaten another scarified companion, presented an interesting appearance. In addition to subcutaneous haemorrhages on the chest wall, congestion of the lungs and some splenic enlargement and congestion, there was a very striking congestion of the duodenum and jejunum, and these portions of the intestine were distended with a dark, bloody mucus. Cultures from the intestine yielded a mixed growth of a coliform bacillus and an enterococcus. Smears of heart blood, spleen and liver showed a mixture of enterococci and *Pasteurellae*, and both types were found in cultures of the heart blood. These findings are mentioned as suggesting a probable source for the intestinal organisms—streptococci and coliform bacilli—which have been noted not uncommonly along with the *Pasteurellae* in the blood and organs. They also suggest the possibility of the spread of the disease by biting or cannibalism.

Only a very few Namaqua gerbilles were scarified or inoculated subcutaneously with *Pasteurella* cultures, because most of the stock of these was employed for keeping the disease going by direct scarification from animal to animal—this method being the only one by which at first I was able to keep the disease going. Some of these tests with cultures were successful in transmitting the disease, others were not; but the numbers tested were too small to permit of any far-reaching conclusions being drawn. Dr Rhodes has informed me, however, that his experience with Namaqua gerbilles was similar, and that he found he could only kill about 40 per cent. of those tested.

It should be pointed out that all my animals came from the locality of the epizootic, and an explanation which seems to fit in with the facts of the case would be that the Namaqua gerbilles had a degree of immunity acquired as a result of the present epizootic. Alternatively, the immunity might have been a result of the presence of this disease amongst them in the past, or of a natural partial insusceptibility. This supposition would put them on a par with the Lobengula gerbilles of Johannesburg district (*vide infra*), and would also provide a working hypothesis for bringing the animals dying without obvious *Pasteurella* infection into the same category as those with obvious infection, and so obviate the necessity of postulating a dual character for the epizootic.

Lobengula gerbilles. Most of my experimental work has been done with these gerbilles, which were kindly collected for me by members of the City Health Department, through the courtesy of Dr Milne, M.O.H., Johannesburg.

Scarification of these gerbilles directly with blood or organs of a Namaqua gerbille or other rodent dead of the natural or the experimental disease and showing *Pasteurellae* in the blood, was usually, but not always, successful in killing the gerbille in from 36 to 72 hours. In most cases, however, the gerbille, even when killed, showed no *Pasteurellae* in smears, and the organism was

not recoverable in cultures. Moreover, the disease could not be kept going by serial passage through these gerbilles. Scarification of a second gerbille with the blood of the first one killed was only twice successful out of many trials, and even with these the attempt at a third transmission was a failure.

Attempts at infecting Lobengulas by scarification or subcutaneous inoculation of *Pasteurella* cultures yielded very similar results.

Most attempts to infect by scarification, using as much of a culture as could be picked up on a platinum loop, were unsuccessful, although several efforts did succeed when using a first generation culture from another species such as a rabbit.

Subcutaneous inoculations, using small doses of the order of about an eighth of an agar slope, were usually also unsuccessful, and even in such animals as did die the organism was generally not recoverable. Only by using large doses, of the order of half an agar slope or more, could one kill with any certainty, and even then the organisms were not always to be seen in smears or recoverable in cultures.

These results present a rather striking contrast with those which Dr Rhodes informs me he has obtained in Cape Town with gerbilles of the same species caught in that locality. His gerbilles were uniformly infected both by scarification and subcutaneous inoculation with a characteristic *Pasteurella* septicaemia and recoverable organisms.

This difference seems only explicable on the hypothesis that the Johannesburg gerbilles have a considerable degree of immunity, and passage of the *Pasteurella* through these gerbilles results either in a further loss of its virulence for that species or in its disappearance altogether.

This local difficulty of keeping the disease going in the Lobengula gerbille made me at first very chary of accepting the *Pasteurella* as the cause of the epizootic, but it had the advantage of forcing one to explore other possibilities.

One possibility seemed to be that the *Pasteurella* might also be a secondary invader just as the enterococci and coliform bacilli probably were, and that behind all was some other factor such as spirochaetes or a filtrable virus.

Examinations for spirochaetes of gerbilles dead of the natural disease and of others dead of the experimental disease were completely negative, and injections of filtered extracts of organs of gerbilles dead of the natural disease also failed to kill, so spirochaetes and filtrable viruses were ruled out as improbabilities.

The enterococci and the coliform bacilli were also considered as possible prime causative agents. The former failed to produce anything like the natural disease. First experiments with the latter seemed promising, as gerbilles were killed both by scarification and subcutaneous inoculation. When the animals were examined post-mortem, however, the *Pasteurella* dominated the picture in smears, and in cultures the *Pasteurella* might be recovered in pure culture or mixed with coliform bacilli.

Possible explanations of this were either (1) that the *Pasteurella* developed

from within the gerbille infected by the coliform bacillus, or (2) that the culture of the coliform bacillus was not a pure culture but a mixture of coliform bacilli and *Pasteurellae*.

Plating showed that the second explanation was the true one, and when a pure culture of the coliform bacillus was obtained, it was not lethal either by scarification or subcutaneous inoculation.

This finding that a symbiotic growth of the *Pasteurella* and a coliform bacillus was more lethal for the local gerbilles than the *Pasteurella* alone may have a bearing, however, upon the common finding of coliform bacilli along with *Pasteurellae* in heart blood and organs.

Rats. The *Pasteurella* has not been found pathogenic for domestic rats (*Rattus rattus*), either by scarification or subcutaneous inoculation, even with doses as large as a whole 24-hour agar slope culture.

Guinea-pigs. Scarification tests on guinea-pigs have been uniformly negative in their results, whether performed with blood or organs of animals dead of the natural or the experimental disease or with cultures of the *Pasteurella*. Guinea-pigs can be killed by subcutaneous inoculations of *Pasteurella* cultures if fairly large doses are employed. The features of the disease are similar to those seen in gerbilles with the natural disease.

Rabbits. Rabbits are highly susceptible to infection with the *Pasteurella*, both by scarification and subcutaneous inoculation. In my first experiments I did not test rabbits, having concentrated rather on gerbilles, but when Dr Rhodes informed me of his success with gerbilles he also told me that the rabbit was susceptible, and I can now confirm this finding. Both young and adult rabbits can readily be infected by either method. They may die within 24 hours with an intense septicaemia, or they may survive for about 3 days. In those surviving the longer periods, there may be a soft bubo with some pus formation, and subcutaneous, subpleural, and subperitoneal haemorrhages have been noted, with excess of fluid in the serous sacs. The picture is, in short, one characteristic of a haemorrhagic septicaemia.

Mice. Both white mice and brown mice have been tested. They appear to occupy a position between that of the rabbit and that of the guinea-pig as regards sensitivity, usually succumbing both to scarifications and subcutaneous inoculations but not quite invariably.

Fowls. Not susceptible to subcutaneous inoculations even with large doses.

NATURE OF THE DE AAR DISEASE.

From a pathological standpoint this disease is a haemorrhagic septicaemia. From a bacteriological standpoint the causative organism fits in with the characters of the genus *Pasteurella* as defined by Bergey in his *Manual of Determinative Bacteriology*. The various members of this genus are commonly associated with haemorrhagic septicaemias.

The *Pasteurella* described in this communication does not correspond exactly with any of the species of *Pasteurella* recognised by Bergey. The mor-

phological and cultural characters of the De Aar organism are very similar to those of *Pasteurella avicida* (*Bacillus cholerae-gallinarum*, *Bacillus avisepticus*, etc.) the causative organism of fowl cholera and septicaemia in other domestic and wild birds. There are slight differences in the fermentation reactions, but these are probably of too variable a character to base a specific differentiation upon. I have been informed by my colleagues of the Veterinary Research Institute at Onderstepoort that fowl cholera is not known in South Africa, and further, the absence of any pathogenic action of the De Aar organism for fowls seems clearly to rule it out of this category.

From *Pasteurella cuniculicida* (*Bacterium lepisepticum*), the causative organism of "snuffles" in rabbits, it appears to be distinguishable by several cultural characters and fermentation reactions, and also by its pathogenicity. This organism although productive of a septicaemia in rabbits, on inoculation, is also said to be pathogenic for fowls, which the De Aar organism is not.

Before final decision that it is different from these *Pasteurellae*, the organism will have to be compared with actual strains of them. This comparison has not yet been made, because strains of these organisms were not obtainable in South Africa.

Should the De Aar organism prove to be a new species, as I expect it will, I would suggest the name *Pasteurella desmodilli* for it, as indicative of its origin from the Namaqua gerbille (*Desmodillus auricularis*), the species which has mainly been infected by it in nature.

Practically, the disease is of importance locally mainly because of the difficulty which it interposes in both field and laboratory diagnosis of plague amongst veld rodents. So far there appears to be no evidence suggesting that the disease affects anything other than veld rodents.

Summary.

1. During the summer months 1928–9 an epizootic occurred in De Aar and neighbouring districts among small veld rodents, mainly affecting Namaqua gerbilles (*Desmodillus auricularis*).

2. Many of the animals dying showed a haemorrhagic septicaemia, the causative organism of which appeared to be a *Pasteurella* rather smaller in size than *Pasteurella* (*Bacillus*) *pestis*.

3. In a considerable proportion of the animals found dead there was no obvious septicaemia.

4. A description is given of the *Pasteurella*, which is believed to be a hitherto undescribed species, and for which the name *Pasteurella desmodilli* is suggested.

5. This organism was found to be highly pathogenic for rabbits, less so for guinea-pigs and mice, and non-pathogenic for rats and fowls.

6. It was found by Rhodes to be highly pathogenic for Lobengula gerbilles (*Tatera lobengulae*) from Cape Town, but for animals of this species from

Johannesburg it proved to be much less virulent, and even when it killed them there was frequently no septicaemia.

7. The above observation seems to indicate the presence of a considerable degree of immunity among the Johannesburg lobengulas, as compared with those from Cape Town.

8. It is suggested that a similar condition of partial immunity among the Namaqua gerbilles (for which there is other evidence adduced) might account for the finding of many of them dead without obvious septicaemia.

9. The existence of this disease introduces a complication in the diagnosis of plague among veld rodents.

March, 1929.

APPENDIX II.

DE AAR DISEASE IN VELD RODENTS: PRELIMINARY REPORT OF LABORATORY INVESTIGATIONS.

BY DR W. F. RHODES.

(*Senior Pathologist, Government Health Laboratory, Cape Town.*)

THE following carcases were received from De Aar district at the Cape Town Laboratory:

3 Namaqua gerbilles,	Paarde Vlei	6. xii. 28	Batch 1
5 „	Brak River	6. xii. 28	
7 „	Vicinity of De Aar	7. xii. 28	„ 2
3 „	Vicinity of De Aar	8. xii. 28	„ 3
4 „	De Aar (Mr Chivers)	10. xii. 28	„ 4
3 „	Victoria West	10. xii. 28	„ 5

POST-MORTEM APPEARANCES.

Batch 1. On opening up all animals smelt strongly of cyanide and showed the usual appearances of cyanide poisoning, *i.e.* lividity and bright red discoloration of the tissues. No other post-mortem changes were noticed and the animals showed practically no putrefaction.

Batches 2, 3, 4. Moderate putrefaction present. General hyperaemia of abdominal organs; spleen slightly enlarged in two only; liver shows slight mottling; stomachs full of food; lungs pale and collapsed. No enlarged glands were found in any of the animals.

Batch 5. Marked putrefaction present and organs disintegrated.

Note. The average time taken for specimens to reach the laboratory was 3 days.

SMEARS FROM ORGANS AND HEART'S BLOOD.

Two animals from batch 1 (heart's blood only), and all the animals in batches 2 and 4 (from all organs and heart's blood) showed a very characteristic picture as follows:

Large numbers of Gram-negative cocco-bacilli with rounded ends. Many

of them show very marked bipolar staining with carbol thionin whilst others appeared as evenly staining rods. It was at first thought that two different organisms were present in these smears, but inoculation experiments have proved that it is likely that only one organism was present. Both varieties showed marked difference in size, the bulk of the bipolar staining variety being rather smaller than *B. pestis* whilst a few were a good deal larger than that organism. Very active phagocytosis could be seen in the large mononuclear cells and this was always at its best in lung smears.

Batches 3 and 5 showed only putrefactive organisms.

On culture.

Batch 1. All the culture tubes, 30 in number, inoculated with heart's blood and tissue extracts remained sterile. This is of interest as it was afterwards ascertained that cyanide powder was used to powder over the carcases to kill fleas before despatch to the laboratory. There would also probably be a similar germicidal action on *B. pestis*.

Batches 2 and 4. A mixed growth of two different organisms was isolated: (i) to be described later as Type A, and (ii) a coliform bacillus.

Batches 3 and 5 yielded only putrefactive organisms.

Animal inoculation.

Batch 1. Four guinea-pigs and four gerbilles were inoculated subcutaneously with pooled lung, liver, spleen and heart's blood and none of them showed any signs of illness. This could also be due to the germicidal action of cyanide.

Batch 2. Four guinea-pigs were inoculated subcutaneously, two with pooled heart's blood and two with pooled spleens with the following results:

No. 1 died 24 hours after inoculation.
No. 2 ,, 48 ,, ,,
No. 3 ,, 48 ,, ,,
No. 4 ,, 60 ,, ,,

Post-mortem examination showed the following in all of them:

1. Acute purulent general peritonitis and consequent gut congestion and paralysis. The peritoneal cavity contained purulent fluid which in three was blood stained.
2. Spleen and liver slightly congested, enlarged and friable.
3. Lung collapsed, but no other lesions.

Smears from all four showed a similar picture to that described in the post-mortem findings of batches 2 and 4.

On culture Type A bacillus was obtained pure from guinea-pig no. 1 and mixed with a coliform bacillus in guinea-pigs nos. 2, 3 and 4.

Batch 4. Similar results were obtained.

Batches 3 and 5. No results were obtained but scarification was used instead of subcutaneous inoculation owing to the putrefactive organisms present.

DESCRIPTION OF TYPE A BACILLUS.

A rod-shaped non-motile bacillus with rounded ends, varying in size from a short slender cocco-bacillus to a rod-shaped bacillus 6μ long. It is very pleomorphic, Gram-negative and shows only slight bipolar staining on culture. No capsule could be demonstrated.

Growth. It will grow at room temperature, but the growth is much more luxuriant at 37° C. On agar it grows fairly well, the colonies being clear at first and later becoming opaque. After some days the growth looks like a mixed culture. ? Rough and smooth types.

Gelatin stab—slight growth over surface and upper quarter of stab.

Broth shows granular growth, definite pellicle formation and heavy deposit.

Potato—no growth.

Indol formed.

MacConkey's neutral red bile salt agar—no growth.

Nitrates—not reduced.

Acid in dextrose, levulose, sorbite and mannitol.

Slight acidity raffinose, mannose and inulin.

No fermentation lactose, dulcitol, arabinose, amygdalin, maltose, rhannose, adonite, dextrin, salicin and erythrite.

Litmus milk—no change.

From the above cultural characteristics the organism should be placed in the *Pasteurella* group, and as it does not conform to any organism previously described it is suggested that a suitable name would be "Pasteurella TATERICIDA."

PATHOGENICITY.

Guinea-pigs are killed by subcutaneous inoculation but not by scarification.

Rabbits, tame mice, Lobengula and Namaqua gerbilles, and Karroo rats are killed both by scarification and subcutaneous inoculation, but not by feeding.

Fowls and pigeons were not killed by feeding, scarification, subcutaneous inoculation or intravenous injection.

Black and brown rats were not killed by scarification.

One cat was experimented on and failed to die by scarification.

The numbers of animals scarified and results.

Animal	No. scarified	Result
Rabbits	10	All died and organism isolated
Mice	4	,, ,, ,,
Lobengula gerbilles	8	,, ,, ,,
Namaqua gerbilles	22	15 ,, ,, ,,
		7 remained well
Karroo rat	1	Died and organism recovered

The probable explanation of the partial immunity of the Namaqua gerbilles is that the animals used in the earlier work were obtained from the

epizootic area at De Aar. On rescarification two months later two of four previously immune animals died and the organism was recovered.

Remarks on pathogenicity.

Where animals of the same species were scarified and others inoculated subcutaneously the former invariably died first. Rabbits die from scarification usually in 24 hours whilst those subcutaneously inoculated take from 48 to 72 hours to die.

Gerbilles usually have a period of from 3 to 6 hours of drowsiness before death, but during this period also show irritability and very much resent interference. Rabbits, on the other hand, appear well to within an hour or two of death.

It has been possible to kill a Namaqua gerbille with the culture, recover the same from the heart's blood pure, scarify a second gerbille with the culture from the first and on death again recover the organism pure, and continue up to the fourth generation, when the experiment was stopped.

It is a very important fact that gerbilles are killed by scarification as it had been previously thought that if a gerbille died after scarification in conjunction with a bipolar organism being recovered from the organs that organism was probably *B. pestis.*

Fortunately guinea-pigs though not so susceptible to plague as gerbilles do not die by scarification with this organism.

The post-mortem findings in animals experimentally inoculated are more reliable than in the animals received after a lapse of time from the infected area. They are:

(i) Marked haemorrhagic exudation into the tissues at the site of scarification.

(ii) A generally congested appearance of the abdominal organs, a little clear fluid in peritoneal cavity, with occasionally slight enlargement of the spleen, mottling of liver but no areas of necrosis and the stomach full of food.

(iii) A small amount of fluid in pleural and peritoneal cavities; lungs pale and collapsed.

The whole picture is consistent with death from septicaemia. In animals subcutaneously injected a small local abscess has formed from which the bacillus has been obtained in pure culture.

Transmission.

As the disease caused by this bacillus is a septicaemia it was thought possible that fleas would be responsible for its transmission. A number of experiments have been made with different species of fleas, all with negative result. The animals used were Lobengula gerbilles. Work has been begun to see if it is possible to transmit from a sick to a healthy animal by biting, but no definite result has as yet been obtained. Another possible suggestion is that healthy animals get bitten and their wounds get infected either from the

blood or secretions of an infected animal. The experimental work in this direction and field work on the same lines is to be continued. The organism has maintained its pathogenicity unimpaired for $4\frac{1}{2}$ months.

CAPE TOWN,
March, 1929.

APPENDIX III.

DIFFERENCES BETWEEN PLAGUE AND DE AAR DISEASE IN VELD RODENTS.

BY W. POWELL.

(*Chief Rodent-Inspector, Department of Public Health, Union of South Africa.*)

REGARDING the disease recently prevalent amongst veld rodents in De Aar, Victoria West and neighbouring districts, and the probably identical outbreak on farms in the Calvinia district, it is interesting to note—from the point of view of field diagnosis—the differences between this disease and plague—especially as regards symptoms in the sick animals and the localisation of carcases on the veld.

PLAGUE.

Rodents dead of plague are seldom found far from their burrows—usually within a few yards and more often within a few feet, sometimes in the burrow entrances, and occasionally actually in the burrows. The locality of the carcases is exactly the same as in rodents dying from eating strychnine-poisoned grain.

In the case of plague there are, with very few exceptions, hardly any symptoms of illness or pain before the animal dies. I have often kept plague-infected gerbilles and other mice in cages, and watched them closely, and cannot remember one instance where a rodent showed symptoms of illness or pain before death. I have often examined such rodents and found them looking quite healthy and unconcerned, and half-an-hour later found several dead. On one occasion I fed a Zulu hare which had been inoculated with plague material some 36 hours before. It seemed quite well and began to eat, but a few minutes later and while still feeding it suddenly lay down, gave a few kicks, and died. I have never known a veld rodent recover from plague except on one occasion, when I found a sick striped mouse in an ant-bear hole with a number of plague-infected multimammate mice; the striped mouse recovered, while the multimammate mice all died suddenly without showing any symptoms of illness before death.

It may be concluded that plague-infected gerbilles and multimammate mice, and probably other veld rodents, die suddenly—from heart failure—without pain or other symptoms, and without having time to run any distance

from their burrows, or to create disturbance or arouse suspicion amongst others of their kind so as to cause migration.

With strychnine poisoning, the poison takes effect so rapidly that death usually occurs a few minutes after the poison is taken—and the results, as regards the location of the carcases, are very similar to those of plague.

Plague is very deadly to Lobengula gerbilles and multimammate mice; in these there appears to be no recovery or immunity. In Namaqua gerbilles, on the other hand, there is probably a very small percentage which either recover or are naturally immune and so escape attack. Plague never entirely "wipes out" a Namaqua gerbille population, whereas in my experience Lobengula gerbille colonies are always wiped out—those which escape and live to carry on the infection (and later to start a new epizootic when they have multiplied sufficiently) being remote and isolated colonies which entirely escape infection in the first instance.

The mortality from plague is, as a rule, not very rapid—at least, the rodents do not usually die so quickly and in such numbers that many carcases can be found lying about; under ordinary circumstances the natural scavengers of the veld—hawks, eagles, crows, mongoose, wild cats, etc. quickly pick up the dead rodents, so it is often difficult to find carcases when searching for them. The only occasion in my experience when the rodents died so rapidly of plague that carcases could be found in and around most of their warrens, was in the Kroonstad district of the Orange Free State during 1924. It is, however, unlikely that such a sweeping wave of virulent plague infection would pass over a district without cases occurring in the human population. Plague in wild rodents usually remains active in any locality for from six to twelve months, and human cases may be expected up to two years after the wave has apparently subsided.

The foregoing does not apply to town or "domestic" rats (black and brown rats). When any serious mortality occurs amongst these, whether from disease or poison, the healthy will bunch and migrate—often in hordes. I have seen this bunching and migration of town rats during the carrying out of poisoning schemes. Frequently before the migrations rats are seen running about in an excited state, and then they disappear, this disappearance being quickly followed by complaints of invasions of rats from places some distance away, or of dead rats being found in buildings or localities where poison was not laid. In Kroonstad in 1924 I carried out experiments in gassing rats with carbon bisulphide under the boarded floor of a large store; the floor was not sufficiently air-tight, so only a few rats were killed. The gassing took place in the morning and late in the same afternoon a bunch of rats, about 200 in number, was seen migrating across the street from the gassed store. These rats migrated to a rubbish tip some 400 yards from the store, passing a number of stores and other buildings on their way.

"Domestic" rats are much more suspicious than veld rats and mice; they will often migrate as a result of very little disturbance or with small cause for

suspicion—even as a result of removing or disturbing some of their harbourage, or repairing part of a building which they infest. Because of this habit they may spread plague very quickly throughout a town. This is also a drawback to the use of poison for town rats, unless the poisoning is carried out so as to ensure that the bulk of the rats take the poison before they become suspicious and migrate.

According to recent experiments by Dr Rhodes, town rats appear to be insusceptible to the De Aar disease.

DE AAR DISEASE.

In the De Aar disease veld rodents behave quite differently. In addition to my observations in the Calvinia district recently, I have closely watched gerbilles infected with this disease while assisting Dr Rhodes in his experiments during the past week.

The interval between the appearance of first symptoms of illness and death is from 1 to 6 hours. During this time the animal is restless, as if in great pain, and very drowsy; often it seems dead but upon lifting it up it will wake and become quite active, and will usually try to bite. This biting inclination seems to be general, even with the tamest of these rodents. In some cases they feed —or at least try to bite or gnaw—even when so weak that they can hardly stand. I watched one trying to eat a cabbage leaf; he put his teeth into the leaf and then lay as if asleep for a short while—then woke up, gave a few weak bites, and again lay quiet. One sick Namaqua gerbille which I found in the Calvinia district was very weak and drowsy, and was moving round in a circle; the nearest burrows were about 200 yards away, so that it must have wandered some distance. I saw a number of gerbilles in a drowsy state in the entrances of their burrows. One, in particular, I pulled out by its tail thinking it was dead, but it woke up. In my search for fresh carcases, during which I examined hundreds of burrows, I only found two carcases near the burrows, but saw several hundreds of dried remains scattered, or in groups, over the veld—in many cases as far as 500 yards from the nearest warren. The wave of infection had subsided when I arrived there, and in my search I found only one fresh carcase and one sick gerbille.

The disease appears to cause alarm and disturbance amongst the whole rodent population and the healthy animals run about excitedly.

Judging from the duration of mortality in the Calvinia district and at De Aar, the infection seems to remain active for only a few weeks. So far, all the known outbreaks have occurred during the summer season.

Not more than 50 per cent. of the rodents died in the affected area of the Calvinia district, and in parts of this area rodents are still fairly numerous, especially in the valley of the Klein Fish River, to which many rodents from the surrounding country have migrated, owing to the drought—there being more vegetation and food to be found along the river.

Laboratory experience goes to show that most of the rodents which survive

in an area over which a wave of infection has passed, are more or less immune to the disease—at least temporarily.

The sick animals become very weak, and in their wanderings they may be stopped by small obstacles such as the metals of a railway line. At both De Aar and Calvinia numbers of carcases were found lying against or near the rail on the side nearest the burrows; some animals succeeded in getting over one rail but failed to get over the second, so died between the two.

This disturbance and alarm of the rodent colonies and the aimless wandering about of the sick rodents appear to be characteristic of De Aar disease; in no instance have similar phenomena been observed in the case of plague.

Fleas were extremely plentiful in the burrows and on the rodents in the Calvinia epizootic. They are also reported to have been numerous on the rodents at De Aar. These facts may have important bearings on the mode of spread of the disease amongst them.

Summary of differences.

In *Plague* in veld rodents:

(1) The animals rarely show symptoms of illness before death.

(2) There is no alarm or disturbance of the rodent colonies and the sick animals do not wander about.

(3) Extremely few of the rodents attacked recover or become immune.

(4) It is usually difficult to find carcases.

(5) The dead are seldom found far from their burrows.

(6) The mortality is, as a rule, not very rapid.

(7) Infection usually remains active for from six to twelve months.

In the *De Aar disease* in veld rodents:

(1) The animals show definite symptoms and appear to suffer from pains in the stomach, with fever, restlessness and periods of drowsiness—death taking place after some hours of illness.

(2) The rodent colonies are disturbed and alarmed. The healthy rodents run about excitedly and the sick animals wander away aimlessly and may travel some distance before dying.

(3) Only about 50 per cent. of the rodents die; those that recover appear to be immune to the infection—at least for a time.

(4) Numerous carcases are found.

(5) Carcases are usually found some distance from the burrows.

(6) The mortality is, as a rule, very rapid.

(7) Infection usually remains active for a few weeks only.

Cape Town,
March 5th, 1929.

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