REPORT OF AN OUTBREAK OF PLAGUE IN QUEENSLAND DURING THE FIRST SIX MONTHS OF 1904.

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Plague was introduced into Queensland from Sydney in 1900, and every subsequent year has been marked by an outbreak in Brisbane. In both towns the authorities are unsparing in their efforts to eradicate the factors contributing to its maintenance, but in both places the infection of the rats and their haunts is so extensive that the disease may now be looked upon as endemic in the two cities. The probabilities are, however, that the pest will be harder to eradicate in Sydney, where the buildings are substantially built, and not as in Brisbane light structures of wood which more easily lend themselves to treatment and are comparatively temporary in their character.

During the period stated twenty-nine cases were reported in the State. Twenty-four cases occurred in Brisbane and the immediate vicinity, one at Ipswich, two at Cairns, and two in Maryborough. (43 cases were reported as suspicious and negatived on clinical and bacteriological examination.)

The routine method of notification and report instituted by the Commissioner of Public Health (Dr Ham) is comprehensive and accurate, and reduces the chances of diagnostic error to a minimum.

Immediately on receipt of notification of a suspect, generally by a medical practitioner, the Government Health Officer visits the case, examines it clinically, and takes specimens from the affected gland, blood, or sputum, as circumstances may indicate. Hypodermic syringes and needles in sterilized test-tubes are carried ready for use. Smear preparations are made and part of the material is forwarded to the Government Bacteriologist, who also examines smears and inoculates a serum tube and guinea-pig. The results of the clinical and immediate bacteriological examination are embodied in the Report, to which the subsequent reports on culture and inoculation are appended.

General features of the outbreak.

The progress of the disease and the incidence in the several months are included in the appended table. The figures for the years 1900, 1901, 1902, and 1903 are submitted for comparison.

Month	1900	1901	1902	1903	1904
January			1		
February		1	14	6	7
March		7	20	4	
April	3	7	28	6	7
May	10	12	18	4	12
June	10	3			3
July	14	3			
August	4	1	1	1	
September	5			1	
October	4	1			
November	3				
December	3	1			
Totals	56	36	82	21	29

The majority of the cases, as shown in Appendix B, were associated with the handling and distribution of produce, and it is instructive to compare Appendix B, relating to the occupations of the patients, with Appendix C dealing with the incidence of plague rats on the premises of the various trades. (See p. 328.)

Most of the patients were men in adult life and there were only two females, one of whom was employed in the produce trade. This disparity in the sexes is accounted for by the fact that women are hardly ever employed in the trades from which the cases are derived.

Causes of recrudescence of plague in Queensland.

The last case of plague which occurred in Brisbane in 1903 was on September 11th; the last infected rat was found during 1903 on September 28th. The first infected rat of 1904 was found on January 8th, and the first case of human plague on February 9th.

The factors contributing to and causal of the present outbreak may be considered under importation, rats, seasonal influence, infection by food, and infection by insects.

Importation.

While there is no evidence to adduce in support of the present outbreak of plague being other than purely sporadic, it is well to direct attention to a possible source of infection in the jute bags which are imported from Bombay and Calcutta. The regulations in force prohibit the importation of used produce sacks, and when it is realised that many of the new bags imported from India are made from material served out by the factories to natives who make it up in their own hovels where plague infection is ripe, the necessity will be apparent for submitting these bags to thorough disinfection before allowing their distribution.

Rats.

During the period January 1st to June 30th, 37,254 rats and mice were destroyed; of these 14,755 were examined. 310 rats out of 11,479 were found to be infected, and 3 mice were infected out of 3,276 examined.

The following table illustrating the number of rodents examined for the last three years shows the work done by the Department of Public Health in destruction and examination.

Until the present year mice were included under rats and no distinction was made.

1902	1903
"Rats" examined 1,315	"Rats" examined 6,500
"Rats" infected 96	"Rats" infected 80
Percentage of "rats" infected 7·3	Percentage of "rats" infected 1·23

1904	Totals
Rats examined 11,479	Rodents examined 14,755
Rats infected 310	Rodents infected 313
Percentage of rats infected 2·7	Percentage of rodents infected 2·1
Mice examined 3,276	
Mice infected 3	
Percentage of mice infected ·90	

As will be seen from Appendix D, 78 of the 313 infected rats were obtained from the "Infected Area," and 88 were found during the cleansing operations in one store in South Brisbane. Appendix D also shows the localities from which the rats were obtained during the several weeks of the period under observation. Appendix C sets forth the

incidence of infected rats on the various business premises. It will be noted that the produce premises are responsible for 111 out of a total of 313.

In view of the heavy incidence of plague-infected rats and plague cases associated with the storing of produce a systematic inspection was made of all the metropolitan stores, and cleansing operations instituted. The results of the investigations are embodied in Appendix E. They may be of use to others who may have to deal with similar infective foci.

Wherever an infected rat is found the immediate and neighbouring premises are thoroughly overhauled and search made for other rats, and all sanitary and other defects made good.

Species of infected rats:

Mus decumanus	249
Mus alexandrinus	31
Mus rattus...	27
Species not given	3
Mice	3
Total	313

It is essential that the operations for rat destruction on any premises be conducted continuously and, if possible, completed in one day. If uncompleted the rats invariably migrate during the night. *Mus alexandrinus* is the most difficult to deal with. It rarely burrows, climbs well, seldom remains long in one place, and decamps at the first sign of operations.

Endemic maintenance and dissemination of the disease by rats are the main causes of the annual outbreak of plague in this State. Many other animals are more or less susceptible to plague. Poultry merit particular attention, as not only are they themselves susceptible but their food is a constant attraction to rats. Here until recently it was a common occurrence to find the ground under the cottage houses barred off as a fowl yard[1].

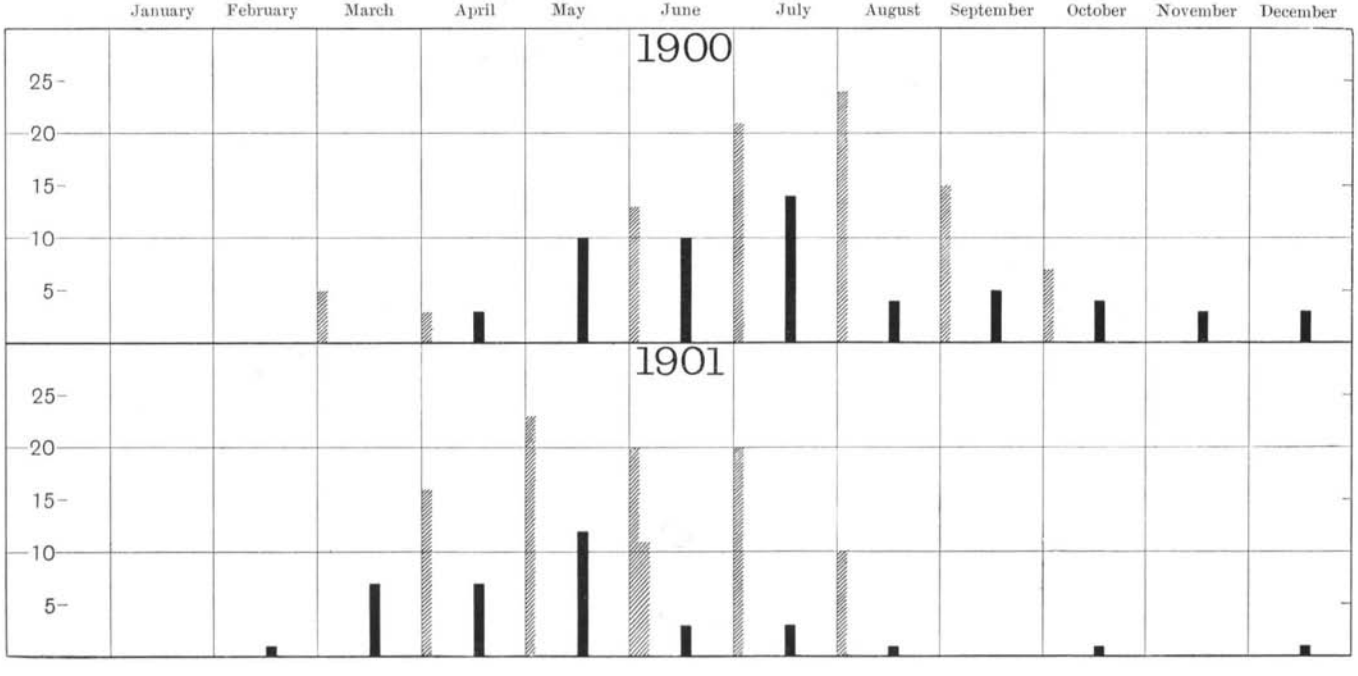
Simpson (Hongkong Report, 1903) puts the case very clearly:—

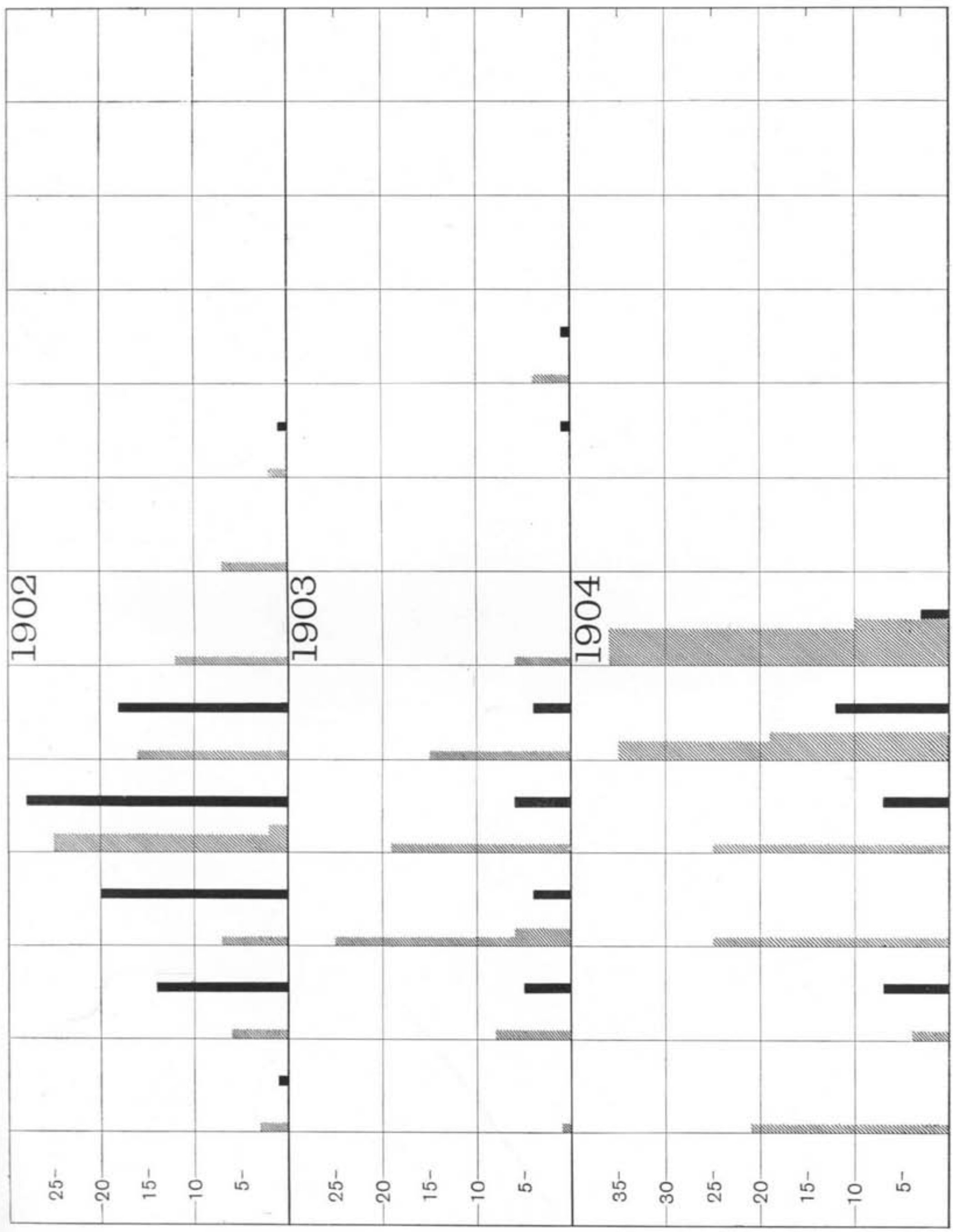
"The mortality of rats preceding and coinciding with the epidemic is significant and important. Were rats and men attacked concurrently at the beginning of an epidemic the spread of the infection would have to be looked for in some common media containing the infectious material and which were likely to affect both simultaneously. The antecedence of rat to human plague in this and every other

[1] Houses are built on piles to protect them from the ravages of white ants.

GRAPHIC CHART OF RAT AND HUMAN PLAGUE IN THE YEARS 1900—1904.

Incidence of Rat Plague shewn shaded. *Incidence of Human Plague shewn in black.*





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epidemic points to a channel of infection which implicates the rats alone primarily. The dissemination of the infection by the rat is necessary to bring it into those channels by which man can become infected."

It is probable that under certain natural circumstances a reduction in the virulence of the organism is effected and a comparative immunity is conferred on the rats. The infection of immigrant rats is, however, severe and their arrival is heralded by a heavy mortality. In the same manner an infected rat imported into a fresh locality produces a similar result. This attenuation of virulence is responsible for the condition known as chronic rat plague. Kolle and Martini[1] first directed attention to its occurrence. Observations and experimental investigations conducted in the Bacteriological Laboratory here by Mr Pound, and discussed by Dr Ham in a State Health Paper, place its existence in Brisbane beyond doubt.

In view of the high probability that infection from rat to rat is mainly by the agency of food it is significant to note that guinea-pigs are easily infected through the digestive tract, in which case there is a special tendency to chronic forms (nodules in various organs including the lungs)[2].

These facts are a clear explanation of the many instances of prolongation of large epidemics and of reappearances of plague after opportunity for infection has apparently disappeared.

It is significant that the rat plague reappeared this year first in the "Infected Area" particularly in connection with the produce stores, and that all the cases with the exception of eight were connected with the produce industry directly or indirectly.

The view that rat to rat infection is effected by insect agency must be greatly discounted in the light of recent investigations into the infectivity of the secretions and excretions of plague-stricken animals. The plague bacillus is found in the mucous secretions, faeces and urine of from 20 to 30 % (see Simpson's Hongkong Report, 1903) of infected rats and there is considerable ground for belief that contamination of food is the chief mode of rat to rat infection. This dissemination of bacilli by the secretions and dejecta explains the retention of the infection in these rat-ridden stores, most of which were at the beginning of the outbreak overcrowded, structurally defective, inadequately ventilated, badly drained, and insufficiently cleansed.

[1] *Deutsche med. Wochenschr.* Jan. 1902.

[2] Bandi and Balistreri, *Zeitschrift für Hygiene*, 1898, XXVIII. p. 261.

Seasonal incidence.

Plague in Queensland as in other affected countries possesses special seasons for its prevalence. It is at its lowest between midwinter and the latter part of summer, and reaches its maximum from February to June. There is a special connection between rainfall and plague. Rainfall during an epidemic is invariably followed by an increase of cases.

This increase is probably due to local conditions. The rain drives the rats from the sewers and secluded underground haunts into buildings and situations frequented by men, who are thus exposed to a more proximate infection.

Infection by food.

Rat to Rat. There is abundant evidence to show that food can and does convey the infection of plague from animal to animal; and considering the high proportion of infected rats which have bacilli in the saliva it is probable that food is the main channel by which the disease is naturally spread among rats.

Rats are continually fighting, and eat their companions that have died from plague. They nibble at food and if infected the bacilli are deposited on the remnant and available for infection of a succeeding feeding rat.

Rat to Man Plague is readily communicated to grey monkeys by food which has been exposed to infected rats, and food is probably the source of infection in man where glandular symptoms are absent or secondary to haemal infection; also probably as will be discussed later in many cases where there is apparently only glandular infection.

Infection by insects.

The experience gained in connection with rat and cleansing gangs points to the conclusion that the relation of fleas to the transmission of the disease has been much over-estimated[1]. The men are being constantly bitten by fleas, especially on the exposed arms. The only cases which have occurred have been due to direct inoculation, one by a splinter, and the other by a rat bite. Isolated examples may

[1] Cf. Kolle and Martini, *Deutsche med. Wochenschr.* Jan. 23rd, 1902, and Indian Plague Commission Reports.

occur where a flea may directly inoculate a victim. Bacilli have been found in fleas as in other insects. The theory that every bubo is indicative of direct inoculation by fleas somewhere in its drainage area is untenable. Their most important rôle is as carriers of infection, the same as bugs, flies, etc. Fleas from their environment of dust and dirt necessarily are more closely associated with plague infection. The significance of this association is evident when it is considered that the plague bacillus, if it does not actually have a saprophytic existence, has not infrequently been isolated from such surroundings[1].

Pound[2] narrates an interesting instance of food infection by cockroaches:

> The facts are as follows: In a room specially set apart for keeping all inoculated animals are two large stands with wide shelves on which are placed long lead-lined trays about two inches deep, containing carbolic solution; standing in these trays and surrounded by the carbolic solution are the various strong glass jars in which are kept the experimental animals. These jars are about sixteen inches high and about nine inches in diameter. Each jar contains only one animal, guinea-pig, rat, or mouse, as the case may be, and is covered with a mosquito-proof fine wire-gauze lid. On the occasion referred to, in November, 1902, a healthy guinea-pig that was being kept as a control for certain experiments suddenly became sick and after three days died. Post-mortem and bacteriological examination proved that this guinea-pig had died from a generalised form of plague, but no lesion was found to indicate that it had been infected through the skin. A careful examination revealed the fact that in the zinc binding of the wire cover there were several very young cockroaches. These were promptly destroyed. On examination of the covers of the other adjoining jars more young cockroaches were discovered.
>
> It is quite evident that these cockroaches had become hidden in the zinc lining of the covers when the jars were not in use, and standing on the shelf alone, unprotected by the tray of carbolic solution.
>
> As soon as a jar was occupied by an experimental animal the cockroaches that had been hidden from view in the zinc lining during the daytime, would, after dark, crawl down the inside of the jar and feed on the animal's food. Apparently, before their presence was discovered, some of these cockroaches had fallen from a jar containing a plague-infected animal into the carbolic solution, and then swam either to the jar containing the healthy guinea-pig or to the side of the tray, and then hid away in the cover of an empty jar. In any case it was more than probable that the food had become contaminated with bacilli.
>
> In order to ascertain whether the cockroaches had anything to do with the transmission of plague, a healthy guinea-pig was placed in a sterilised jar covered with the usual wire lid, but whose zinc lining was free from cockroaches. The jar was placed on the shelf, but not on the tray. In the course of a few days young

[1] Kitasato, Hankin, Leuman.

[2] *Queensland State Health Report*, 1903.

cockroaches made their appearance, and, as usual, lived during the daytime in the zinc lining. Eventually the guinea-pig sickened and died of plague.

There is sufficient convincing evidence in this one observation to show the extreme danger which exists when insects like cockroaches can gain access to places where plague-infected animals are kept.

After this experience, the whole of the building and everything such as shelves, benches, jars, etc., were subjected to thorough and repeated disinfection, and all holes and crevices carefully closed. The result is that no cockroaches have been seen since; and although every day during the past eight months numbers of plague and healthy guinea-pigs and rats have been kept in the same jars and standing in the same trays, no symptoms of the disease have appeared in any animal unless specially infected.

Synopsis of twenty-five cases under direct observation.

Case I. Saddler: Infected rats discovered on premises during the preceding week. Initial temperature 103° F.; pulse extremely weak and dicrotic; serum 120 c.c. injected subcutaneously, 2nd day; temperature normal 4th day; right upper femoral gland affected; suppuration delayed and limited.

Case II. Schoolboy: Plague next door last year; infected rats from store immediately adjoining; initial temperature 103° F.; pulse weak and dicrotic. Serum 200 c.c. subcutaneously. Temperature normal 10th day; had serum rash. Left upper femoral gland affected.

Case III. Produce hand: Ill twelve days before coming under observation. Initial temperature 100° F.; dicrotic pulse. Temperature normal 7th day; gland resolved without suppuration. No serum was given. Left upper femoral gland affected.

Case IV. Produce hand (female): A bag maker. Initial temperature 104° F.; dicrotic weak pulse. Serum 120 c.c. subcutaneously. Temperature normal 18th day; left upper femoral gland affected and sloughed entirely.

Case V. Schoolgirl: from infected area. Initial temperature 105° F.; pulse 180, weak and dicrotic. Serum 240 c.c. subcutaneously, 120 c.c. intravenously. Temperature normal 17th day; upper left femoral gland affected. Very severe case.

Case VI. Produce-bag merchant: Initial temperature 105° F. Serum 240 c.c. subcutaneously. Temperature normal 5th day. Left upper femoral gland affected.

Case VII. Produce hand: Initial temperature 104° F. Serum 240 c.c. subcutaneously. Left upper femoral gland affected; little suppuration. Temperature normal 4th day. 17 days in hospital, the shortest period of detention of the cases.

Case VIII. Produce hand: Initial temperature 104° F.; pulse extremely weak. Serum 200 c.c. subcutaneously. Died on the second day. Complete renal suppression from time of admission. History of previous renal trouble. Autopsy findings: chronic interstitial nephritis and general infection of the organs with the bacilli. Cultures of *B. pestis* were obtained from all the thoracic and abdominal viscera.

Case IX. Stable hand: Initial temperature 104·6° F. Obtained horse feed from infected store. Serum 200 c.c. subcutaneously; temperature normal 5th day. Left upper femoral gland affected; was not removed to Colmslie.

Case X. Produce hand: Initial temperature 99·2° F. Serum injected subcutaneously. Died on the second day. General blood infection, bacilli were mixed in type but mainly coccal.

Case XI. Grocer and Produce hand (Chinaman): Initial temperature 104° F. Died immediately after being seen, had been ill 4 days. Large bubo implicating the whole left femoral group.

Case XII. Produce hand: Initial temperature 103·6° F.; pulse weak and dicrotic. Serum 102 c.c. subcutaneously. Temperature normal 5th day. Left upper femoral gland affected and resolved without suppuration.

Case XIII. Grocer and Produce hand: Initial temperature 104·6° F.; pulse 120, weak and dicrotic. Serum 320 c.c. subcutaneously. Left upper femoral gland affected; serum rash. Temperature normal 20th day. Gland sloughed *en masse.* Very severe case.

Case XIV. Race-horse trainer: Produce obtained from infected store. Rats dying in numbers prior to his seizure. Initial temperature 105° F. Pulse dicrotic. Serum 240 c.c. subcutaneously, right upper femoral gland affected. Temperature normal 12th day.

Case XV. Oyster-saloon cook: Infected rats from the shop this year. Four days ill before seen; initial temperature 105·4° F. Pulse 152, dicrotic. Serum 120 c.c. subcutaneously. Died shortly after being seen. General blood infection.

Case XVI. Produce hand: Initial temperature 120·6° F., pulse weak and dicrotic. Serum 120 c.c. subcutaneously. Temperature normal 6th day. Right upper femoral gland was affected and resolved without suppuration.

Case XVII. Produce hand: Initial temperature 104·2° F., pulse 90, weak and dicrotic. Serum 120 c.c. subcutaneously. Temperature normal 8th day. Lower left femoral gland affected; sloughed *en masse.* Detained in hospital 46 days. The longest case. There was a sore on the inner side of the left calf which yielded plague bacilli on examination. There was marked lymphangitis between the sore and the saphenous gland. The sore (circular, $\frac{3}{4}$-inch in diameter) consisted of a central black eschar round which was a ring of purulent vesicles. Surrounding this again was an area of livid, brawny induration. From the same store as in *Case XVI.*

Case XVIII. Sanitary wharf-man: Ill three days; died before arrival of Health Officer. Left upper femoral gland affected. Autopsy: general blood infection and secondary specific pneumonia.

Case XIX. Estate agent: Initial temperature 104·8° F., pulse 104, very weak and dicrotic; left upper femoral gland affected. Serum 240 c.c. subcutaneously. Temperature normal 7th day. Dead infected rat found in offices.

Case XX. Stableman: Initial temperature 102·6° F., pulse 100, weak and dicrotic. Serum 240 c.c. subcutaneously. Left upper femoral gland affected; temperature normal 5th day. Serum rash. Produce from infected store.

Case XXI. Schoolboy: Initial temperature 105° F.; pulse 136, weak and dicrotic. Serum 240 c.c. subcutaneously. Right upper femoral gland affected; temperature normal 6th day. Infected rats at store close by, which he was in the habit of frequenting. Family cat also brought rats to the house from this store.

Case XXII. Produce Store hand: Initial temperature 103·8° F., pulse 104, weak and dicrotic. Serum 480 c.c. subcutaneously. Left inferior external axillary glands

affected, also left femoral. Developed intense blood infection and secondary specific pneumonia and died on the 10th day. The only case where subcutaneous haemorrhages were present; these appeared on the 6th day.

Case XXIII. Draper: Infected rats obtained in the vicinity of the home. Initial temperature 102·8° F.; pulse 96, weak and dicrotic. Serum 120 c.c. subcutaneously. Temperature normal 9th day. Left upper femoral gland affected.

Case XXIV. Baker: Infected rats from bakehouse. Serum 120 c.c. subcutaneously. Died 5th day. General haemal infection. Developed meteorism the second day, and lapsed into an adynamic condition which continued until death.

Case XXV. Student: A remarkable case; arrived in Brisbane a week previous to his attack. When seen by Drs Bancroft and Love was found to have a condition similar to malignant pustule on the nape of his neck, a central eschar within a zone of confluent pustules and surrounded by an area of hard brawny indurations (cf. *Case XVII*). On bacteriological examination plague bacilli were found in large numbers. The lesion was energetically treated by incision and pure carbolic acid. Serum 240 c.c. was administered subcutaneously. The bacilli were evidently all eradicated in the local lesion as there was no secondary adenitis. Initial temperature 104° F.; pulse 112, dicrotic; rapid constitutional recovery. Temperature normal 3rd day. There were no bacilli obtained in the scar after treatment. The scar healed in a month.

Points of clinical interest.

The average stay of the patients in the hospital was 29 days as compared with 50 days in 1903.

The average time that the patients were ill before serum was administered was 2·6 days as compared with 4·16 days in 1903. The reduction of the period as a result of prompt notification of the cases by the profession is a matter for congratulation, as I think to it is due the very substantial reduction in the death-rate shown by the following tables:—

Gross Case Mortality (Fatality).

1903	1904
53·8 %	31 %

Corrected Case Mortality for White Races.

1903	1904
42·7 %	20 %

Prompt notification is imperative to ensure the early administration of serum.

Serum was administered to almost every one of the 43 suspects. The experience is that if the case proves eventually not to be plague no evil results ensue from the injection.

In addition to the usual concomitant constitutional symptoms special attention was directed to the occurrence of albumen in the urine. Albuminuria was found to be present in 80 % of the cases. Its amount of albumen varied from a mere trace to 5 "Esbach" in a fatal case where the immediate cause of death was suppression. Albuminuria was more marked in fatal cases. In the cases in which it occurred it appeared generally about the third day and persisted usually for about a week. Hyaline, granular, and epithelial casts were frequently observed. In one instance blood casts were present and in two blood was present in small amount in the urine. Difficulty in micturition and associated pain were a marked feature at an early stage of the disease in a large proportion of adult males.

In the State Health Report, 1903, Dr Ham emphasises the early advent of a peculiarly soft and dicrotic pulse in plague. The clinical symptoms in the several cases constituting the present epidemic have varied considerably. Some cases have been associated with high temperature: in others there has been little rise of body heat. In some vomiting has been the salient feature; in many diarrhoea; in others both. The pulse rate varied in the initial stages from 70 to 150. But in all (Case XXV excepted) there have been two constant clinical features, viz. adenitis and marked dicrotism of the pulse. The latter is undoubtedly the most constant clinical symptom in plague. Its occurrence in association with femoral adenitis may be regarded as pathognomic of the disease.

Anderson (*Australasian Medical Gazette*, 1904) states that if the bubo be inguinal or femoral there is always swelling and thickening in the corresponding iliac fossa, coupled with tenderness on firm pressure. He regards the sign as peculiar to bubonic plague. I have directed attention to this condition, and although in a few cases I have detected a slight fulness probably due to propagated adenitis, I am unable to endorse his view of the value of the symptom. The tenderness I have found has been no more than one would expect on firm pressure in the immediate vicinity of any acute inflammatory lesion.

Heart-failure is a grave possibility in the early pyrexial stage of the disease; but as soon as the temperature falls the danger of such a catastrophe disappears. Myocarditis is frequently spoken of as the cause of the feeble heart action in plague, but I think that were myocarditis present in sufficient degree to be alone responsible for the state of the heart the eventual rapid and complete restoration of the heart to a normal condition would be impossible. The circumstances point

rather to a toxaemia affecting the vagal centres and the intrinsic cardiac ganglia.

In bubonic plague sufficient attention has not, I think, been accorded to the significance and influence of kidney complications. They are most important, and there is a fertile field for valuable research in this direction to ascertain how much of the albuminuria is due to specific kidney affection and how much to serum injection; and whether serum injection is contra-indicated where there is evidence of renal complications or indicated irrespective of the state of the kidneys.

Diagnosis—clinical and bacteriological.

All the cases were primarily bubonic. Four succumbed to virulent general haemal infection. In two cases death was due to secondary pulmonary infection. There was no case of primary pulmonic plague. In two cases there were primary plague sores from which plague bacilli were obtained. These cases are reported and commented on in the synopses of case records submitted.

The gland initiating the bubo was, with two exceptions, either the upper external or upper internal femoral, and in 19 out of 25 cases the left side was affected, a proportion of 76 %.

This constant involvement of the upper femoral glands cannot be a mere coincidence. It cannot be explained by their relation to the areas drained by them primarily, which is insignificant. They are really secondary glands interpolated on the collecting trunks from the lower femoral glands which receive the lymphatics draining the major portion of the cutaneous surface of the lower limb. In the case where there was a primary sore on the inner side of the calf in the drainage area of the internal saphenous lymphatics, the gland affected was the lower saphenous femoral gland (cf. Case XV).

In a previous section of the report I have directed attention to researches which tend to minimise the hitherto predominant position occupied by the flea hypothesis of infection. These facts relative to the particular glands and their relation to cutaneous drainage go a long way to further discount the hypothesis.

The extreme range in the degree of constitutional symptoms associated with the local adenitis rendered an accurate diagnosis in many cases extremely difficult. One had to obviate the possibility of a simple adenitis being diagnosed as plague, and the patient being removed and exposed to even the very slight possibility of infection in the plague

hospital. On the other hand was the danger of allowing a mild case of plague to escape recognition. The danger in such an event would be from some of the immediate contacts becoming inoculated by the organisms in the discharge from the gland directly, or through the medium of soiled dressings, etc.

Bacteriological diagnosis is by no means always the infallible test it is supposed to be, especially in mild cases. Interesting points are that the severity of the clinical symptoms is very often in direct proportion to the number of bacilli observed in a smear made from the juice of the affected gland: also that when atypical and coccal forms are present in addition to the ordinary form of the bacillus the prognosis is much graver than in cases where the organisms conform to the regular type.

The triple test—smear, culture, and guinea-pig inoculation—give results in these mild cases which vary as follows:—

	Smear	Culture	Inoculation
A	Positive	Positive	Positive
B	Negative	Positive	Positive
C	Negative	Negative	Positive

(A) If bacteria are recognised in the smear preparations, the culture and inoculation tests invariably yield positive results.

(B) The smear sometimes shows no organisms, yet culture and inoculation are positive.

(C) Smear and culture yield negative results, but the inoculated guinea-pig dies. In these cases the guinea-pig often lives from two to five days longer than the usual time, thus further intensifying the difficulty of establishing an early and accurate diagnosis. Indeed in not a few cases the patient has recovered and is again in sound health before the death of the guinea-pig.

Cases falling under Class C present a difficult problem as regards diagnosis and the propriety of isolating them. An interesting circumstance in connection with these cases is that the gland generally resolves, and in the isolated instances where suppuration occurs its area of implication is limited and its course short. There are authorities who assert the existence of a disease called "pestis minor which is not plague." I have no doubt the majority of cases designated as such belong to the category under discussion, and are simply examples of mild plague resultant on an infection by *B. pestis* of low virulence, whose capacity for multiplication is soon overcome by the bacteriolytic power of the body cells and fluids. The local disturbance is therefore

slight and the clinical symptoms comparatively trivial and evanescent. These details support the dictum that *B. pestis* like many other organisms varies in virulence.

The virulence of the bacillus can be increased or diminished artificially *in vitro* and *in vivo* by methods which are well known. Under natural conditions there are doubtless many circumstances which influence and determine the degree of virulence in particular individuals and epidemics.

This conclusion is supported by experimental infection, and the cutaneous method devised by Weichselbaum, and elaborated by Albrecht and Ghon, is invaluable in determining the degree of virulency of the bacilli in any given case. Guinea-pigs and rabbits though excellent for ordinary diagnostic purposes are very unsuitable for testing the degree of virulence of plague organisms.

The lung tissue (cf. Klein, Local Government Board Reports) has a special power of exalting the virulence of *B. pestis*, since guinea-pigs inoculated with organisms from the lung of an animal dying from plague succumb much sooner than others inoculated from the spleen or other organ. Further, the cultural descendants of bacilli taken from the lung retain exalted virulence for several generations. This emphasises the necessity of faithfully observing the usual special precautions adopted in treating pneumonic plague.

With a view of ascertaining the value of the recent discovery of Bell, of Hongkong, that *B. pestis* is present in the blood of every case of plague, and not as hitherto regarded only in septicaemic cases or as an immediate antecedent of death, I have examined smears from the peripheral blood of the last eleven patients admitted to the hospital; I have found plague organisms present in the blood of eight. Three of these cases subsequently died, two from secondary septicaemic plague and one from secondary pulmonary infection, therefore the results obtained from them are not admissible.

The presence of bacilli in the peripheral blood in the remaining cases, however, lends striking support to the view that there is in every case of plague a dissemination of bacilli through the general circulation. Of the three cases in which I failed to detect the bacillus, two were extremely mild and the third showed a purely cutaneous lesion which was treated in time to prevent even glandular infection. This accords with my previously expressed views that the severity of the disease, given the same degree of virulence, is directly proportionate to the number of organisms present. How soon the organisms appear in the

blood I am unable to say until further investigation has been made. I have been unable to detect their presence after the defervescence of the initial pyrexia.

This demonstration of the presence of bacilli as a common occurrence in the general circulation of individuals suffering from plague introduces a further tangible basis for doubting whether buboes are always indicative of local inoculation[1].

Treatment.

The immediate and remote results attending the administration of Yersin's serum were very gratifying. If a *potent* serum is given early and in sufficient dosage nearly every case of bubonic plague in Europeans ought to get well, provided there are no complications such as kidney mischief, etc.

The published experience of clinicians who have used this serum attribute to it very varying values. Some belaud it, others condemn it as useless. The explanation lies in the varying potency of the serum that has been employed. It deteriorates with age, some specimens more rapidly than others. In other instances it will be found that fresh consignments of serum obtained from the Institutes vary in potency. Why this should be so I am unable to explain; possibly it may be due to the idiosyncrasy of the horses used. In all cases, however, it is imperative whenever a fresh supply is used, and from time to time with the stock in hand, to conduct a series of experiments on guinea-pigs or other animals to determine the degree of potency. By this means one is enabled to discard or reject unsatisfactory serums.

The routine treatment is to give 120 c.c. immediately the case is brought under observation. The dose is repeated in twelve hours if the temperature remains up. Rarely is it necessary to give a third dose.

Cold bathing and sponging are invaluable in combating delirium, pyrexia, and insomnia. Antipyrine and its congeners are useless and dangerous.

It is extremely important that a close watch be kept on the

[1] *Note:* My method of making the smear was (all precautions were taken to eliminate possible contamination) to obtain very thin smears from the patient's blood by Cole's method. In a private communication from Dr Bell in which he furnishes further results he informs me that he works with very thick smears which he decolorises. It is a matter of gratification that we have obtained similar results with diverse methods.

performance of the kidney functions until the initial pyrexia has defervesced. Where possible, the urine ought to be tested for albumen every time passed. Sudden and extensive structural damage or functional disturbances as evidenced by a large amount of albumen in the urine may occur any time and demand prompt and vigorous treatment. Urotropin is useful, whilst subcutaneous and intravenous injections of saline are invaluable, and in many cases will satisfactorily tide over a very ominous and grave crisis.

By far the best hypnotic in plague is morphia given hypodermically. A single sufficient dose even up to half a grain is much more efficacious and safer than repetition of smaller doses. The digestive system stands the strain of plague well, and a generous diet may be exhibited with advantage much earlier than is usual in similar febrile diseases.

The successful management of buboes is an important feature in abbreviating the stay of the patient in the hospital. My experience is that premature incision of buboes ensures protracted, intractable, and defective sinuses. If the buboes are allowed to thoroughly mature the plague bacilli are killed by the pyogenic organisms, and when the abscess is opened the whole of the necrosed gland is speedily discharged and specific organisms have either disappeared or do so in a day or two. A clean, healthy cavity is left, the walls of which speedily granulate up.

Sequelae have been rare and insignificant in this epidemic, and, as after typhus, many patients aver that they enjoy better health than previous to their illness.

In conclusion, I have to acknowledge my extreme indebtedness to the Commissioner of Public Health for his encouragement and kindness in indicating lines of research, and the facilities he has accorded me of access to his investigations and reports.

APPENDIX A.

Brisbane Rodents examined, not examined, infected, and destroyed, for 6 months ending 30th June, 1904.

Date week ending 1904	Rats examined	Rats infected	Mice examined	Mice infected	Rats not examined	Mice not examined	Total rodents destroyed
January 9	518	1	—	—	15	—	534
,, 16	497	1	—	—	24	—	522
,, 23	507	6	—	—	21	—	534
,, 30	677	13	—	—	17	—	707
February 6	506	—	—	—	42	—	548
,, 13	470	1	—	—	34	—	505
,, 20	394	1	—	—	27	—	422
,, 27	529	—	—	—	462	8	999
March 5	458	4	—	—	1251	560	2273
,, 12	492	3	9	—	1477	1593	3574
,, 19	431	5	38	—	1020	1777	3271
,, 26	584	4	195	—	1202	1975	3961
April 2	358	10	186	—	916	1585	3055
,, 9	358	4	247	—	401	482	1492
,, 16	406	7	249	—	241	684	1587
,, 23	443	3	299	—	231	739	1715
,, 30	497	11	345	—	140	417	1410
May 7	428	15	237	—	215	221	1116
,, 14	379	12	222	—	345	331	1289
,, 21	340	15	244	2	263	328	1192
,, 28	300	27	121	—	224	224	896
June 4	364	80	162	1	289	244	1140
,, 11	430	28	129	—	297	250	1134
,, 18	547	36	221	—	277	277	1358
,, 25	371	9	208	—	251	279	1118
,, 30	195	14	163	—	178	352	902
Totals	11479	310	3276	3	9860	12326	37254

Percentage of rats infected 2·7. *Bonus paid £399. 0*s*. 11*d*.
,, ,, mice infected ·09. Wages paid £1183. 15*s*. 2*d*.
£1582. 16*s*. 1*d*.

* Bonus 6*d*. per rat, 3*d*. per mouse.

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APPENDIX B.

Occupations of Patients who suffered from Plague during 1904.

Draper	1
Estate Agent	1
Grocers	2
Produce Store employees	9
Saddler	1
Oyster Saloon Cook	1
Sanitary Wharf labourer	1
School children	3
Storeman	1
Student	1
Stablemen	3
Total	24

APPENDIX C.

Number of Infected Rats, etc., found in the various Business Premises in the Metropolitan Area for six months ending 30*th June,* 1904.

Advertising Agent	1	*Brought forward*	71
Arcade	1	Grocers	20
Bacon Store	1	Hardware and Fancy	1
Bag and Bottle Store	1	Hotels	15
Baker...	1	Municipal Baths	3
Blacksmiths and Ironmonger ...	5	Municipal Markets	12
Boarding Houses	3	Music Shop	1
Bootmakers	2	Printers	2
Butchers	12	Produce Stores	111
Butter Factory	1	Railway Premises	2
Cafés	11	Residences	33
Carpenter	1	Saddler	4
Chinese Dealers	4	School	1
Coachbuilder	4	Secondhand Stores	2
Confectioner	2	Shipping Office	1
Courier Buildings	3	Stationers	2
Dentist	1	Undertaker	1
Draper	1	Unknown	2
Drill Shed	1	Upholsterer	1
Eating, Fruit, and Oyster Saloons	6	Wharves and River-banks ...	20
Farmer	5	Wholesale Warehouses	7
Fish dealer	2	Wood Depot	1
Government Buildings	2	Total infected Rodents ...	313

Of the above number 3 were mice, the remainder rats.

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APPENDIX D.

Brisbane Analysis of Plague-infected Rats, etc., for 6 months ending 30th June, 1904.

1904 Week ending	City	Kangaroo Point	Valley	New Farm	Spring-hill	Petrie Terrace	New-stead	Bowen Hills	Infected Area	South Brisbane	Woolloon-gabba	West End	River & Wharves	Outside Authorities	Totals
Jan. 8	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1
,, 15	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1
,, 22	—	—	—	—	—	—	—	—	4	—	—	—	1	—	5
,, 29	5	—	—	—	—	—	—	—	7	—	—	—	2	—	14
Feb. 5	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1
,, 12	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
,, 19	1	—	—	—	—	—	—	—	—	—	—	—	—	—	1
,, 26	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
March 4	—	—	—	—	—	—	—	—	4	—	—	—	—	—	4
,, 11	—	—	—	—	—	—	—	—	3	—	—	—	—	—	3
,, 18	—	—	—	—	—	—	—	—	3	—	—	—	1	—	4
,, 25	—	—	—	—	—	—	—	—	4	—	—	—	—	—	4
April 1	2	—	1	—	—	—	—	—	7	—	—	—	1	—	11
,, 8	3	—	—	—	—	—	—	—	1	—	—	—	—	—	4
,, 15	2	—	—	—	—	1	—	—	3	—	—	—	1	—	7
,, 22	—	—	—	—	—	—	—	—	2	—	—	—	—	—	2
,, 29	4	—	—	—	—	—	—	—	4	1	—	—	2	—	11
May 6	3	—	2	—	1	—	—	—	5	1	—	—	—	—	12
,, 13	5	—	3	—	2	—	—	—	4	1	—	—	—	—	15
,, 20	3	—	1	—	—	4	—	—	5	4	—	1	—	—	18
,, 27	4	—	—	—	1	—	—	—	2	1	6	—	1	1	16
June 3	4	—	2	—	—	—	—	—	3	4	73	3	1	—	90
,, 10	4	—	—	—	—	1	—	—	1	1	7	1	6	—	21
,, 17	11	—	3	—	1	9	—	—	9	1	1	3	—	5	43
,, 24	1	—	—	—	—	—	—	—	1	3	—	—	1	—	6
,, 30	2	—	1	—	—	8	—	—	3	—	1	—	4	—	19
Total	54	0	13	0	5	23	0	0	78	17	88	8	21	6	313

APPENDIX E.

The condition of the Brisbane produce stores.

The continued incidence of plague-infected rats and plague cases associated with the handling of produce constrained the issuing of instructions by the Commissioner for a systematic inspection and cleansing of the produce stores.

The first conclusion forced upon me was that any building was evidently considered good enough for the storage of grain and fodder. Many of the structures had previously been used for such purposes as omnibus stables, blacksmiths' shops, etc. In most cases where the buildings had been built specially they were of the flimsiest nature, and no attempt had been made to render them rat-proof.

Construction (*Brick buildings*): Of thirty stores cleansed fifteen were constructed of brick, and where cellars occurred the floors were either concrete or asphalt. Eleven were satisfactory, requiring only such minor improvements as wire netting on windows and ventilation openings, and doors made rat-proof.

The remaining four were unsatisfactory and exhibited such faults as bad drainage, insanitary stabling, earthen floors, and general dilapidation.

Wood and iron buildings (15): One was well ventilated; four in fair condition; the remaining eight were unfit for use as produce stores and could not have been made rat-proof without incurring costs out of proportion to their value. They were therefore condemned.

Incidence of plague rats.

In the thirty stores cleansed and disinfected were found 541 dead rats and 957 live rats; 305 dead mice and 607 live mice. Of the 1492 rats 109 (7·30 %) were infected; and of the 912 mice 2 (·22 %) were found to be infected.

Incidence of plague cases.

Of the 24 persons who suffered from plague within the Metropolitan Area nine (37·5 %) were directly employed in the produce business, and three were engaged in stable duties and in frequent contact with produce.

Storage of produce.

The method adopted for storing by merchants in the Area is very objectionable. The produce is dumped on the floor and then piled up from eight to twelve bags high, thus providing ample cover for rats. The fact frequently referred to by merchants that the stock is being constantly changed and turned over does not remove the objection, as a store is never completely emptied and seldom half emptied, and, as long as any stock is left the rats are able to remain in hiding, and as new stock comes in they again spread themselves out.

In support of this contention it may be stated that the total number of rodents caught in produce stores during the $2\frac{1}{2}$ months that the special cleansing operations were being carried on was $6\frac{1}{2}$ % of the total number caught in the whole Metropolitan Area for six months.

If produce is stored on the floor it is impossible to keep the premises clean, and as chaff falls from burst bags, etc., accumulations form between them which cannot be cleared away until the produce is sent out; and it is seldom done then, but new stock is generally dumped down in the position from which the old stock was removed.

Some of the stores are too small for the business done in them: consequently, overcrowding is common and tends to keep premises dirty.

Recommendations.

That all buildings in which it is intended to store produce should be built of brick and cement or concrete, and that all cellar floors should be of concrete on solid foundation to prevent rats burrowing under same; that all cellar walls should be rendered tight inside with cement mortar. That all doors should fit closely to floors when shut; that all air or light openings should be covered with stout wire netting to prevent the ingress of rats, or that such stores should be built of iron frames sheeted with stout corrugated iron with concrete floors as above, and windows and doors similarly fitted.

That all drains be trapped and ventilated and sufficient surface drainage provided to keep yard in a clean and dry state. That stables be of sufficient size, and that stalls be floored with impervious material and the bedding changed daily. That stall floors have a decided fall outward to a surface drainage through trapped gully.

That manure receptacles of impervious material, with floor somewhat above level of ground and of concrete, should be provided in all cases where there are stables, and that the receptacles be provided with proper cover and thorough means of ventilation, and that "weep holes" be provided into a surface drain. All surface drains should be of concrete or hard burned bricks on edge, properly bedded and grouted in cement mortar.

That all earth closets should have concrete floors and should be kept regularly lime-washed.

That all produce should be stacked on sparred stands at least 12″ off the floor, to enable the floor to be periodically cleansed under stock and that, if possible, the produce be not stacked against walls.