

Plague in East Suffolk 1906–1918

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Five miles from Ipswich, between the villages of Freston and Holbrook, are two attached cottages, Latimer Cottages. Mr C lived in one of the cottages with his wife and her four children from a previous marriage. On 13 September 1910, his stepchild AG, a girl aged 9 years, became ill with a severe cough, pneumonia, and diarrhoea and vomiting. She died three days later. Six days after her death Mrs C became ill and died after two days' illness. Three days after his wife's death Mr C and Mrs P, a neighbour who had nursed Mrs C, became ill and they too died three days later. All the victims had similar symptoms. Dr Carey, their general practitioner, attended all the patients and called in Dr Brown, a physician from Ipswich, to see Mrs C, but she had died before he arrived. When Mr C and Mrs P became ill the possibility of pneumonic plague was considered: blood was taken from Mr C and some bloodstained fluid was removed by syringe from Mrs P's lung. The specimens were examined by Dr Llewellyn Heath, the bacteriologist at Ipswich, who grew the plague bacillus from both specimens. Dr Heath took his material to Professor Sims Woodhead in Cambridge, who confirmed the diagnosis.

The last two patients were buried on 30 September, the vicar taking the whole service in the open air; all those attending had their clothes disinfected. There were no necropsies or inquests. On 1 October the contacts were removed to isolation accommodation in Tattingstone Workhouse, which had been opened for this purpose. On the same day Dr Sleight, the medical officer of health for the district, notified the local government board of the diagnosis. All four deaths were thought to be due to pneumonic plague, because of the short incubation period, the high mortality and the probability of case to case infection. There was no indication how the first victim, AG, had become infected.

HISTORY AND PATHOGENESIS

Plague in Suffolk has received little attention and the outbreak of 1910 is not mentioned in any of the standard texts on the subject. A few articles appeared in contemporary medical journals^{1–5} and further information emerges from an article, *The Last Epidemic of Plague in England? Suffolk 1906–1918*, by David van Zwannenberg⁶.

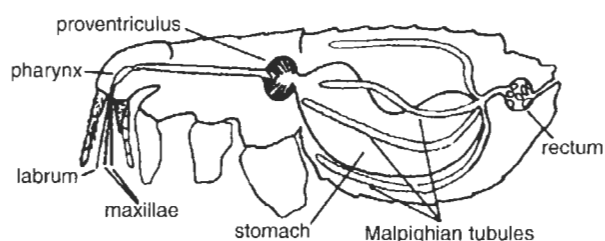


Figure 1 Alimentary canal of adult flea. Reproduced by permission from Ref. 7

The earliest authenticated pandemic of plague was in AD 542–750 (the Justinian plague). The middle of the 14th century saw the beginning of the 'black death', which continued until the middle of the 17th century, killing a quarter of the population of Europe. The great plague of London (1665–1666) was one of the last major epidemics of the black death, with 60 000 deaths in a population of 450 000.

After 1666 there were no more pandemics in Europe—probably because of the replacement of the black rat (*Rattus rattus*), which lives in human habitations, by the brown or sewer rat (*Rattus norvegicus*), which prefers to live apart from man. Outbreaks continued to occur in western Europe in the seventeenth and eighteenth centuries but Britain remained free until a small epidemic occurred in Glasgow in 1900, with 36 cases and 16 deaths. The black rat persists in Africa and Asia, where it continues to be responsible for outbreaks.

The last pandemic started in China in 1894 and continued until the 1930s, spreading east and west and killing over a million people in India, where plague is still endemic.

Plague occurs in man predominantly in two forms, bubonic and pneumonic; septicaemic plague is less common and meningitis may occur in inadequately treated cases. All forms are due to the plague bacillus *Yersinia pestis* which was identified in 1894 independently by Kitasato and by Yersin.

In bubonic plague the victim is infected by the bite of a flea which has sucked blood from a rat with plague. The host's skin is pierced by the mouth parts of the flea and saliva is injected; blood is then sucked up and passes down the oesophagus into a bulbous dilatation called the proventriculus before entering the stomach (Figure 1). The blood is digested and the undigested remains are passed

in an almost dry state as infective faeces. Plague bacilli sucked up with the blood multiply in the flea's stomach, causing the proventriculus to become blocked. When the flea bites another victim some of the infected blood is regurgitated into the new host. Fleas with an obstructed gut become starved and bite repeatedly in order to obtain another meal. In bubonic plague the infection is carried to the regional lymph glands, which enlarge and form buboes in the groin, axilla or neck, depending on the site of the bite. Untreated bubonic plague has a mortality of 70%, reducible to 50% with antibiotic treatment.

Pneumonic plague is thought to arise initially when a victim is infected by a bite; instead of a bubo developing, the disease spreads through the bloodstream to the lungs, causing pneumonia. The sputum of a patient with pneumonic plague contains enormous numbers of plague bacilli and is highly infectious; case to case infection occurs. Pneumonic plague has a mortality of almost 100%; death occurs too quickly for antibiotics to have any effect.

Though the rat is the usual reservoir of infection, over two hundred wild rodents can harbour the plague bacillus⁷; in addition hares, rabbits, ferrets, dogs and cats can become infected though they do not act as reservoirs. Rat plague can continue for many years without infecting humans. The disease is maintained because some rats survive the disease and transmit it to other rats. From time to time, probably when there is a high proportion of non-immune rats or the population rises, an epizootic develops and the disease spreads to animals that are likely to come into contact with man—for example, cats and dogs, or animals that are shot for food, such as hares and rabbits, or used for hunting rabbits or rats, such as ferrets. Man may also be infected by picking up a dead rat. When a rat dies its fleas leave the body and are ready to bite an alternative host; fleas can jump 18 cm vertically and 35 cm horizontally⁷. Some twenty-nine species of flea, including the human flea (*Pulex*

irritans) and dog and cat fleas, can transmit plague. The species of rat flea which can carry plague (*Xenopsylla cheopis* in Africa and Asia, *Nosopsyllus fasciatus* in the Suffolk outbreaks) breed in the rats' nests, which are lined with grass or straw and are usually underground.

ACTION TAKEN AFTER THE FRESTON OUTBREAK

What happened after the deaths in Latimer Cottages? On 3 October 1910 the local government board inspector, Dr Timbrell Bulstrode, telegraphed Dr Sleight that he would visit the area the following day. Later he acknowledged the receipt of a rat caught near Freston. The rat died and was sent to Dr Klein, the board's advisor in bacteriology, at the Lister Institute in London. On 12 October the rat and a hare shot near Freston were both found by Dr Klein to be infected with plague. There were also reports that other infected rats had been found in the district; an infected ferret was found in Woodbridge and a cat died of plague in Stutton.

The Samford Rural District Council (covering an area bounded by the River Orwell to the north, the River Stour to the south, the North Sea coast to the east and the railway line between Ipswich and Manningtree to the west [see Figure 2]), distributed handbills advising the public not to touch dead rats, and farmers were encouraged to kill rats. By the middle of October several letters on rat extermination appeared in the local press. The Rev Marmaduke Washington, Rector of Holbrook, wrote:

'It is said that the Rural District Council of Samford shrink from touching the pockets of the ratepayers [he was presumably referring to the Council's refusal to supply free rat poison] but in a grave emergency of this character I feel assured that the ratepayers of this district would support them in an immediate and effective effort to stamp out a serious plague spot in our midst by removing one potent factor of contagion. I hope that this letter may elicit the opinion of others more competent than myself to deal with this "question". I write, Sir, as the head of a household and one who has deeply at heart the health of this parish and the neighbourhood'.

Dr Bulstrode arranged with the local authority for rat-catchers to be appointed and for free rat poison to be distributed. Four nurses were immunized against plague with Haffkine's vaccine and were available to work anywhere in the area. On 10 November the local government board issued an order making it compulsory for local authorities to report cases of rat plague and to destroy rats.

As a result of these findings a survey was conducted from 8 November to 22 November and from 27 December to 2 January 1911 by Dr C J Martin and Dr Sydney

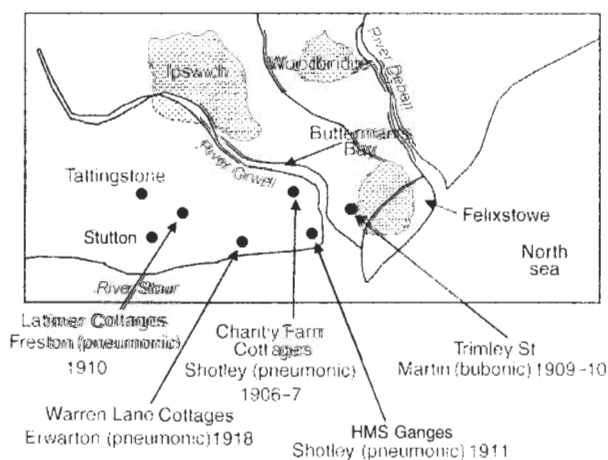


Figure 2 Map of East Suffolk showing the sites where cases of plague occurred

Rowland, both of whom were involved with the Commission for the Investigation of Plague in India. The laboratory equipment was 'conveyed to the site in a small motor car'. The area surveyed was bounded by the coast from the mouth of the Orwell to Orfordness, from Orfordness to Woodbridge, from Woodbridge to Boulge and thence to Claydon, Ipswich and Copdock. The investigators examined 568 captured rats; all were brown rats. Seventeen of these rats were found to be infected. The rats were killed and placed in a jar with chloroform; fleas are very sensitive to chloroform and remain anaesthetized for a long time, greatly helping with species identification. Dr Martin and Dr Rowland paid particular attention to the flea population and obtained 584 fleas, about half of which were of the species *Nosopsyllus fasciatus*, which they demonstrated will readily bite man in the absence of its normal host. The stomachs of three fleas from rats infected with plague were examined; two contained a considerable number of plague bacilli. 40 rabbits were also examined, 2 of which carried the flea described above; 2 rabbits were found to be infected, one either recovering or suffering from chronic plague and one with acute plague.

On 14 January a second more extensive survey was begun by the local government board inspectorate; this involved 22 urban districts, 15 rural districts and 301 parishes. No infected rats were found.

Though the second survey was negative the local government board conducted a third survey between July and October 1911⁵; these months were chosen because rat plague tends to expand into an epizootic in the early autumn. Of 15 332 rats examined by dissection, 35 were found to be infected; diagnosis was mainly on the basis of post-mortem appearance and was confirmed by bacteriological culture in some cases.

The surveys had shown that rats on both sides of the Orwell were infected. During 1912 a quarter of a million rats were killed but no cases of plague were discovered. In 1913 two parishes in the Shotley peninsula and one in the Woodbridge district were found to have infected rats, and 7 infected ferrets were found in the Woodbridge district. In 1914 no infected rats were found and no further action was taken because of the war.

In summary, the rat infection was widespread, and extended in some years at least over a wide area of East Suffolk and into the eastern part of Essex. An important finding was the infection in other animals—hares, rabbits, ferrets and one cat.

OUTBREAKS BEFORE AND AFTER THE DEATHS IN FRESTON

On a third visit Dr Bulstrode made inquiries into the possibility of previous outbreaks of plague in the area. He

was able to identify two probable episodes, one in 1906–1907, in Shotley, and the other in Trimley between December 1909 and January 1910. Dr Bulstrode was informed by a gamekeeper at Woolverstone Park that in 1906–1907 rats were observed to be dying in large numbers on the estate. The gamekeeper at Freston House reported a similar high mortality among rats in the autumn of 1910; in both instances the dead rats were well-nourished, in contrast to rats dying from the Danysz and Liverpool 'viruses' which were in use at that time (these were cultures of enhanced virulence of *Salmonella typhimurium* and *Salmonella enteritidis* [Gärtner's bacillus]).

The Shotley outbreak (1906–1907)

This outbreak involved two families in Shotley living in Charity Farm Cottages and Brickhill Terrace Cottages between Shotley and Chelmondiston, starting on 9 December 1906 and ending on 6 January 1907. In this outbreak eight people were infected with pneumonic plague; one, a young man, recovered. As in the Freston outbreak all the patients were seen by Dr Carey; the deaths were certified as due to pneumonia; there were no necropsies or inquests.

The Trimley outbreak (1909–1910)

This outbreak occurred in the village of Trimley St Martin, to the north of the Orwell and a little to the west of the Ipswich to Felixstowe railway line. The outbreak started on 19 December 1909 and ended in January 1910. The infected family consisted of two adults and their five children, aged from 6 to 18 years. The home circumstances were poor and the house was reported to be infested with fleas. All seven members of the family were affected, of whom three recovered. All the victims developed bubonic plague, at intervals of three to six days between cases.

At the inquest on her mother, a girl aged 18 described the illness of her family. All but one had had a 'knot' (enlarged gland) in the neck, axilla or groin. The inquest on the mother returned a verdict of 'death due to exhaustion, the result of a weak heart'. Dr Bulstrode concluded that the family had suffered from bubonic plague, with case to case infection, probably by the human flea.

Two later episodes

In his article⁶ van Zwannenberg describes two further episodes, in 1911 and 1918.

On 10 October 1911 a sailor, Mr B, based at the Royal Naval Barracks (*HMS Ganges*) in Shotley, complained of headache and pains in his legs; he had a temperature of 104°F. He had cut himself while cleaning a rabbit which he had caught on the Ipswich Road, about a mile from Latimer

Cottages in Freston. He developed severe pneumonia and an examination of his sputum supported the diagnosis of plague. He remained ill for twelve weeks, during which he developed iritis. He recovered but remained almost completely blind and died at the age of 76.

There were no more cases for the next seven years; then two more occurred. A Mrs B, who lived in Warren Lane Cottages in Erwarton, a mile from Shotley, became ill on 8 June 1918; she developed pneumonia and died the next day. Mrs B had been visited by Mrs G, who lived in the same row of cottages, and became ill on 16 June; her breathing became rapid and she began to spit up blood. She was seen by Dr Carey, who had seen the cases in the Shotley and Freston outbreaks. Dr Carey suspected pneumonic plague and asked Captain Cade, RAMC, the bacteriologist to Eastern Command, to visit and to examine Mrs G's sputum. Captain Cade confirmed the diagnosis of plague; the patient died on 19 June. The contacts were removed to Tattingstone Workhouse and all their clothing and bedding was burnt.

There were thus four distinct episodes of plague in the area, three south of the Orwell and one (Trimley) north of the river. Three of the outbreaks were due to pneumonic plague and one to bubonic plague. In the case of the sailor with pneumonic plague, infection was, unusually, acquired through a cut while cleaning an infected rabbit.

HOW DID THE PLAGUE REACH SUFFOLK?

There is no evidence that plague was in existence in Suffolk before 1906, nor were there any reports, apart from isolated cases in ports, of plague in other parts of the British Isles between 1906 and 1918. Dr Bulstrode, in his report to the local government board⁴, considered various ways in which the disease could have been introduced into the area. The most obvious route was by shipping on the rivers Orwell and Stour. Two types of cargo were carried—horse or cattle manure and grain. Manure was brought from London for use on the fields, but there were no reports of plague in the Port of London at that time. Grain was brought up the Orwell from the Black Sea ports and from ports in North and South America; all these ports had cases of plague from time to time. Grain barges also went up the Stour estuary as far as Bures. In the Orwell some of the larger grain ships off-loaded cargo into barges at Butterman's Bay on the north bank of the Orwell, to lighten their draught sufficiently to enable them to dock in Ipswich. It would have been easy for infected rats to swim ashore or for them to be brought on shore in sacks of grain.

A more remote possibility was that the infection was brought ashore by fleas in sacking or grain; a flea can survive without a host under cool and moderately humid conditions for six months or more, but whether infected fleas can survive this long is uncertain.

WHAT CAUSED THE OUTBREAKS OF PLAGUE?

The unusual feature of the Suffolk outbreaks was that they were confined to a few families; evidently infection in man was a rare occurrence. The surveys showed that between 1910 and 1913 there was a widespread rat plague which varied from year to year. A curious aspect of three outbreaks, in 1906, 1910 and 1918, was that the victims had pneumonic plague, transmitted from person to person. In the Trimley outbreak of 1909–1910 all the victims had bubonic plague, with case to case infection.

CONCLUSION

We will never know for certain how or why the rats in the Shotley peninsula and to the north of the Orwell became infected with plague in the early 1900s when nowhere else in the British Isles was affected. This series of outbreaks was to the best of our knowledge unique in western Europe.

It was remarkable that Dr Carey and Dr Brown had the imagination and clinical acumen to diagnose pneumonic plague in Latimer Cottages when, apart from the Glasgow epidemic of 1900, there had been no outbreaks of plague in Britain for nearly 250 years. And, finally, we do not know why there were no more cases after 1918. Perhaps there are still plague-infected rats in the Shotley Peninsula.

Acknowledgment The letter quoted is reproduced by kind permission of the *East Anglian Daily Times*, Ipswich.

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