

THE BLACK DEATH: A PROBLEM OF POPULATION-WIDE INFECTION

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Introduction

The Black Death of 1347–1350, a pandemic generally believed to be bubonic plague, spread rapidly and resulted in a death rate across Europe that far exceeded that produced in the region by any known disease organism in a single episode, yet nineteenth and twentieth century bubonic plague in more favourable environments failed to raise death rates above those of locally endemic organisms.

This anomaly, though already known has received little attention;¹ neither Ziegler nor Gottfried, writing in 1969 and 1983 respectively, questioned the bubonic plague assumption.² Shrewsbury's detailed history of bubonic plague in the British Isles in 1971 did little to explore the problems of plague in a cold temperate climate.³ Indeed, so fixed was he upon the rat-flea plague model that he discarded some material which could not be fitted to it. His arguments were further weakened by his views of rodent ecology. Morris was critical of Shrewsbury, especially his refusal to give greater weight to the part played by pneumonic plague, preferring typhus instead, but neither author gave adequate attention to the factors that limit the diffusion of that form of plague.⁴

It was clear that a comparison of the medieval and modern pandemics was needed, which was undertaken by the present writer and published in 1984.⁵ The radical conclusion of this examination, that the former outbreak could not have been due to plague, has stimulated research, a recent publication agreeing with this conclusion and also presenting evidence for an infection mechanism quite different from that of bubonic plague.⁶ Research based on Italian records, and fresh analyses of the Bishops Registers and other English material, has also cast doubt on the plague thesis.⁷ A major difficulty in accepting bubonic plague in northern Europe lies in its inability to contact a high proportion of a sparsely distributed population in a cold climate. It is that matter which this paper seeks to address.

Pre-modern (1300–1670) European mortality crises

Medieval populations were accustomed to periods of severe mortality resulting from the twin scourges of epidemic disease and famine. Thus it is

reasonable to enquire whether the high level of mortality in the Black Death was unique or still within the bounds of the variation experienced by such populations.

(a) The Black Death in England

i) Evidence from manorial sources

In the absence of direct data on population numbers and the sort of information that was to be provided later by parish registers, estimates of the number dying in the Black Death have to be sought from other sources. Manorial records can theoretically provide data on deaths from the payment of heriots, the tributes paid to a lord on the death of a tenant. However, at the height of the epidemic the keeping of records suffered and to enable accurate estimates to be made there needed to be a list of landholdings made just before the disease arrived. Such calculations as have been made indicate high death rates: two-thirds of the tenantry of some manors in Hampshire, Wiltshire and Witney and Cuxham in Oxfordshire died.⁸ On three Cambridgeshire manors, two Essex manors and two east-Cornish manors between 50 and 60 per cent died.⁹ On the Berkshire manor of Brightwell one-third died.¹⁰

Information on death rates at the manorial level is to be found in the head-tax lists of the manors owned by Glastonbury Abbey.¹¹ The 1348 list records the subsequent deaths of a high proportion of those named, from 36 per cent in the manor of Ham to 76 per cent in Badbury.

ii) Evidence from ecclesiastical sources

Data on the replacement of priests in the episcopal registers has been used to calculate their mortality. There were 17 dioceses, four of which, Carlisle, Durham, London and Chichester provided no statistics. Those of York and Lincoln were studied by Thompson and the remaining 11 formed the body of research by Lunn, summarised by Shrewsbury.¹² Although there was considerable variation the archdeaconry of Cornwall may be cited, with a maximum possible mortality in eight deaneries varying between 41 and 62 per cent.¹³ In the diocese of Lichfield the deanery of High Peak in Derbyshire had 50 per cent mortality and that of adjacent Castleton 56 per cent.¹⁴ There are many difficulties with this material: priests were instituted to benefices for reasons other than the death of the previous incumbent and the varying time intervals between the death of one priest and the institution of his successor, together with pluralities and absenteeism, present problems. One method of correction has been to assume a period of a month between death and replacement but this crude technique has recently been challenged and refined.¹⁵ Even when we are satisfied with the basic data it cannot be assumed that this reflected the mortality of the general population.

(iii) Mortality estimates

In three years, 1347–1350, the population of Britain was reduced by an amount that has been variously estimated at between 20 and 50 per cent although

Shrewsbury argued that outside East Anglia and the larger towns probably fewer than 5 per cent died of the disease.¹⁶ Hatcher reviewed the various estimates and was of the opinion that a national deathrate of below 25 per cent and above 55 per cent was most unlikely and considered that 30–45 per cent was the best estimate of the deathrate in 1348–1349.¹⁷ Whilst local episodes in later centuries may have equalled this figure, it was a population fall of this magnitude over such a large area that made the Black Death unique.

(b) The Great Famine

Between the year 1296 and the Black Death there was only one other comparable period of high mortality, the years 1316–1320. Heavy rain ruined the harvests in 1315 and 1316 and a severe winter followed in 1317–1318. In some parts the harvest of 1318 was also ruined by rain.

During the resultant Great Famine heriot payments on some Winchester manors in 1316–1317 indicate a death rate that was nearly three times the norm.¹⁸ Other records suggest that in several villages of the same manors and on some Essex manors there may have been a population reduction of 10–15 per cent in the years 1316–1318. An increase in mortality was indicated on the East Anglian manor of Coltishall where in the years 1315–1322 there was an increase in the proportion of non-filial heirs from a normal 12–20 per cent to 33 per cent. East Anglia, however, was the most densely settled rural area in England yet it suffered only modestly in demographic terms in the Great Famine for there was no dramatic rise in the number of heriots in the region.

(c) Fourteenth century Italy

Data from Italy at this time provide some information on mortality in years other than the Black Death. It is not easy to compare it with the English material for it is not known whether children were included. In England the mortality rates were for 'adult' males (that is, aged 12 or more). In Siena, burials in 1340 were about half the 1348 level, at 500, but much higher than the normal figure of 50–60. In 1348 about 1,100 died. In 1363 the burial level was about 800 and thereafter there were peaks at about ten year intervals until the mid-1420s. In Florence the Black Death peak was unremarkable at 200 whereas burials in 1340 had been near 700 and the 1400 figure was almost double that. The normal annual number was about 70. Between 1330 and 1400 there were, as in Siena, peaks of burials approximately every ten years.¹⁹ This places the Black Death in a different light in this region whereby it is seen not as a unique event but as one period of high mortality, and not necessarily the most severe one, in a series. It is, of course, possible that in extreme situations not all deaths were recorded as burials.

(d) Plague in early modern England

After the Black Death there was never again a disease outbreak that covered such a large area so quickly. There were many periods of what was termed 'plague' but these were confined to smaller areas or to individual communities. Nevertheless, some of these were severe although the many

references to plague deaths in the literature do not always present them as a proportion of the total population, making comparison difficult.

In Penrith a major plague in 1597–1598 eliminated some 40 per cent of the population.²⁰ In ‘London’s Dreaded Visitation’ of 1665 between 15 and 20 per cent of the population is estimated to have died, although this may in fact be a lower proportion than was experienced in London in the outbreaks of 1563, 1603 and possibly also 1625.²¹ Some provincial towns occasionally experienced even higher levels of mortality: in York in 1604 about 30 per cent of the population was lost, and in Colchester in 1665–1666 the mortality rate rose to 50 per cent, rendering this probably the most devastating outbreak experienced by any large English town in the early modern period.²² In the Derbyshire hill village of Eyam where earlier estimates, based on an unreliable figure for the total population, had suggested a mortality of 76 per cent, a more recent study has suggested that no more than half the original population was lost, a figure later refined to between 33 and 38 cent who died during an epidemic in 1665 and, mainly, 1666.²³

The plague disease

(a) Bubonic plague

Plague is caused by the bacterium *Yersinia Pestis*. It produces an acute disease in rats and man, fatal in both in a high proportion of cases, and is transmitted to man by the rat flea. In man the bacteria become localised in lymph nodes in the neck, armpit and groin forming large swellings, the buboes, from which the disease gets its name. Before antibiotics were available between 60 and 90 per cent of such cases died, usually within three to five days. An important aspect of any epidemic disease is its ability to infect further victims and so ensure its survival. People ill with bubonic plague pose no threat to others since in this form of plague, even in fatal cases, there are few bacteria in the blood and the human flea would not be effective in inter-human transmission because there would be too few bacteria to form colonies in the gut of the flea.

(b) Septicaemic plague

This form of plague is relatively rare. It occurs when the bacteria, instead of entering the lymph nodes, spread throughout the blood system to form an overwhelming septicaemia, death usually occurring within 24 hours. Bacteria are only present in the blood for a very short time. During this period the human flea can take in bacteria and transfer them to a new host without the need of rats. However, the human flea is not a good transmitter and the fact that bacteria are only available for a short time militates against this route as an effective means of infecting many victims.

(c) Pneumonic plague

When plague bacteria enter lung tissue the third form, pneumonic plague, results. This form of the disease is very dangerous, with a 100 per cent fatality rate. It may occur in a small number of cases during a bubonic epidemic and is

then termed secondary pneumonic plague, as distinct from primary pneumonic plague, which is usually acquired by aerosol infection from wild rodents during skinning when fur trapping.

This is the only form of plague with a theoretical capacity for the infection of many people by person-to-person transmission and there is an assumption that in a temperate climate the bubonic and pneumonic forms would alternate with the seasons, bubonic in summer and pneumonic in winter. An example of this is the statement that from January to March, 1349 there was 'a strain of pulmonary plague' but in late spring and summer 'pure bubonic plague came into its own'.²⁴

Shrewsbury was criticised by Morris for ignoring the evidence for what was termed the high percentage of pneumonic cases in the Great Pestilence of 1348–1350, who suggested that Shrewsbury's myopia concerning pneumonic plague arose from the latter's statement that 'pneumonic plague cannot occur in the absence of the bubonic form and it cannot persist as an independent form of plague', pointing to the Manchurian epidemics as evidence that this was not the case.²⁵ However, the leader of the team that had struggled with those epidemics, Dr Wu Lien-Teh, in his account of the outbreaks said, 'pneumonic plague epidemics arise as a secondary manifestation of bubonic plague', the important point being that there must be a rodent population to produce the bubonic epidemic from which pneumonic cases can develop.²⁶

It is important to note that pneumonic plague is not a common outcome of infection by *Y. Pestis*, usually making up less than 3 per cent of cases, and that it is not highly communicable, contrary to what is often thought. The latter point is especially noteworthy for *Y. Pestis* is poorly adapted to transmission by respiratory aerosols, probably because there are loss-of-function mutations in two genes that limit its ability to cross lung epithelium. Furthermore, although bacteria can be spread in the ejected sputum, most patients are dead within 48 hours (1.8 days in Manchuria in 1910–1911) and that already short potentially infective period is reduced still further by the fact that the patient is not coughing out bacteria for the first 24 hours and for the remainder of the illness is prostrate.

Clusters of pneumonic cases start with an outbreak of bubonic plague and the pneumonic form is almost never transmitted more than two or three steps from the initial pneumonic case. As various studies have shown in Manchuria, Mongolia and India, the pattern of pneumonic plague is one of small, scattered, highly localised self-limiting outbreaks. Whether a pneumonic epidemic ends quickly or not depends on the close proximity of infective cases to the as yet uninfected and a rapid dissemination of cases during the few hours that they are infective.

Only two really successful outbreaks have been known: these were in Manchuria and in both cases these two requisites were met. Infected fur trappers, leaving an area which was the first focus of infection, distributed the disease on the railways and at inns where they rested overnight in crowded

conditions. Between September 1910 and April 1911 plague covered 1,700 miles and 60,000 died. In 1920–1921 better control restricted the second outbreak to 9,300 deaths.

In the medieval context, the distribution of the rat and flea vectors must be central to how bubonic plague could have made contact with numerous people, across a region that was almost entirely agricultural and whose population was mainly dispersed in villages and hamlets.

Rats and plague

All forms of plague depend upon a rodent plague reservoir for the initiation of an epidemic and the rodent species that has been responsible for the majority of human plague deaths since 1894 has been *Rattus Rattus*, variously termed the Black, Ship or House rat and the only rat species in north-west Europe in the fourteenth century. This rat probably spread from India, its region of origin, to Egypt and thence in grain shipments from the Mediterranean.²⁷

Black rat remains were found in a late Roman well in York, and there is evidence of an established population in London by the third to fourth century A.D.²⁸ Unlike the Brown rat, a cold-adapted animal from northern Asia, the Black rat is unable to withstand the cold and is almost entirely restricted to the shelter of buildings, especially those that are heated. Even in warm climates it still prefers to live in buildings, hence its alternative names of House rat and Roof rat, rarely leaving shelter and crossing open ground. This localisation runs counter to the idea that medieval rats moved freely across the towns and countryside, their fleas infecting people wherever they went.

It is generally assumed that rats were numerous and widespread but far from the Black rat being continuously distributed across all parts of the British Isles it is now becoming clear that it was probably absent from the countryside and there are various lines of evidence in support of this view. Firstly, avian predators such as owls eat small mammals up to and including those of rat size. Because they cannot digest the fur and bones these are regurgitated in the form of pellets from which the skeletal remains can be used to identify their diet. The Barn owl hunts over open country, farmland and around farm buildings and if the Black rat had been widespread in the fourteenth century its remains should appear in owl pellets from archaeological sites. There does not appear to be any evidence of these, however, and if there was no extensive rural rat population then there would be no widespread rat epizootics linking settlements.²⁹ In any case, if the rat had been present in rural areas its temperature and habitat preference needs would still have confined it to buildings and there would have been no epizootics in the areas between buildings and outside villages.

Further evidence comes from vernacular architecture in the form of dovecotes.³⁰ During the first half of the eighteenth century the Brown rat,

Rattus Norvegicus, arrived in British ports and quickly spread inland. By 1748 it had penetrated far up a tributary of the Thames to the Chilterns and very soon the owners of dovecotes were forced to re-design those structures in order to keep out the newcomer, which climbed inside and ate both doves and eggs. This problem was a new one, not because the Black rat could not have behaved likewise which, being a better climber, it could, but because it was not a farm pest, as agricultural journals of the time make clear.³¹ Another pointer towards the absence of rats in rural areas is that whilst some manorial accounts record the loss of grain during the winter period, these losses were never attributed to rats. If they had been present they would doubtless have been blamed, particularly if pilfering was rife. Up to the arrival of the Brown rat corn ricks were built on the ground, again because the Ship rat was absent on farms. With the advent of the Brown rat these had to be placed on mushroom-shaped stone structures called staddles and the spread of the rat can be plotted by the dates on these stones.³²

A final, but important, point concerning rats is that nowhere in English accounts is there any mention of the rat epizootic at any time between 1348 and the late seventeenth century, when plague is said to have died out in the British Isles. This is a telling point for medieval writers looked anxiously for anything that would indicate a future epidemic: if there had been rat epizootics it is most unlikely that they would have been missed or escaped comment and their presence was well known as a plague precursor in the East.

In modern times Black rat populations in the British Isles have been confined to ports and some inland towns that were connected to ports by canals, whereby rats in cargo could be transported inland to warehouses.³³ With the cessation of the canal cargo trade in the last 40 years these inland rat populations have died out, as also have those in the ports, largely due to the transport of goods in containers. All the evidence points to the fact that this rat was never secure in the British Isles but was at the limit of its biological range with populations always dependent for their survival upon regular 'topping up' by fresh supplies of rats in cargo. Today the Black rat, whose total population number in the British Isles is estimated at only 1,300 animals, is facing extinction.³⁴ Only a few small groups exist on some western islands in the Gulf Stream where it is warmer and there is a lack of ground predators.

The rat flea, *Xenopsylla Cheopis*, is probably even more limited by temperature because egg hatching and the subsequent passage into an adult flea, a process lasting three weeks, can only take place at a temperature that is between 21C and 29C. In order to sustain an epidemic there must be a succession of flea generations and so the warm weather must last for some months: short periods above 21C are of no use for this purpose and, if alternating with cool spells, could be detrimental to development.

Temperatures over Europe in the fourteenth century were at a low point and the autumn of 1348 was very wet: it is said that from 24 June until Christmas it rained either by day or by night almost without exception.³⁵ Such conditions would have been unfavourable to bubonic plague.

Modern outbreaks of plague

The high mortality caused by the Black Death across Europe indicates a high contact rate by the organism concerned whereas bubonic plague at the beginning of the twentieth century showed a generally low degree of contact, especially in India, where conditions for plague were optimal. Nowhere in the records of modern plague is there any evidence of mortality as high as in 1348–1350: Belgaum, south of Bombay, in the years 1897 to 1909 inclusive, can be regarded as typical.³⁶ In 1897, 1.7 per cent of the population died and in 1898, 7.2 per cent. After this the annual mortality due to plague as a percentage of the total population was 5.8, 4.2, 2.8, 4.0, 3.0, 2.3, 2.3, 0.019, 0.44 and 1.12.

Although plague reached most parts of the northern hemisphere in the late nineteenth and early twentieth centuries it was only successful in colonising those parts of North America where the combined factors of a continental climate and a wide variety of colonial-dwelling rodent species provided suitable conditions.³⁷ The fact that plague failed to become established in the British Isles was not due to lack of opportunity for on 54 ships coming to England there were 82 known cases, of which 17 proved fatal.³⁸ Despite these introductions at several places and evidence of plague in port rats throughout the next 20 years, only about 60 people died of the disease in the British Isles.

For various reasons it is unlikely that the low number of plague deaths owed anything to rat control measures. Furthermore, despite multiple introductions of plague the disease failed to become established in field rodent populations in either the British Isles or western Europe, in marked contrast to those parts of the world where the disease became permanent in a variety of rodent species widely distributed across the continents.

Did *Yersinia* plague cause the Black Death?

Simply considering the climate and its effect upon the distribution of rat and flea together with its restriction on flea breeding, the distribution of the human population and the overall mortality would be enough to produce the answer that *Yersinia* could not have caused the Black Death. To these may be added the rapid diffusion rate which was even more at odds with modern plague. Experience of the disease since 1894 has been that it pursued a faltering progress because of its dependence upon the rat epizootic, which was as discontinuous as the rat populations were, even in urban conditions, because of the availability of food and shelter. In India, the rat epizootic could take six weeks to cover 100 metres in urban conditions: in Europe the Black Death progressed at between one and a half and five miles each day.

The very short transmission time of the pneumonic form is an important argument against the suggestion that primary pneumonic plague was responsible for diffusion across Europe during the Black Death. Even allowing for the intermingling at markets the very close contact necessary for the

transmission of this form of plague was probably lacking. Furthermore, as we have seen, the rodent distribution to provide the initial bubonic epidemic leading to the pneumonic form was absent over rural areas of Britain.

However, in Iceland, where the population was even more thinly distributed than in Britain, with a high proportion of isolated farms and fewer small settlements, there were two severe epidemics, in 1402–1404 and 1494–1495, which by popular tradition have been identified as the same disease that caused the Black Death. These, according to Steffensen, were due to primary pneumonic plague, the victims experiencing severe chest pains and being ill for only a day or two before death. He explained the spread and retention of plague in Iceland by assuming that people acquired the infection from clothing, a means of transmission that for pneumonic plague is virtually impossible because of the inability of the organisms to survive outside the body. Karlsson has shown fairly conclusively that there were no rats in Iceland before the seventeenth century and concludes, ‘the case of Iceland seems to prove that in the Middle Ages an epidemic could rage through a large and sparsely populated country, persist for about nineteen months, and be extremely lethal, without the help of rats or any kind of rodent fleas’.³⁹ Despite this he is wedded to the pneumonic plague hypothesis and proposes as a solution to the problem the *deus ex machina* of mutation of the plague bacillus, leaving the matter there without further discussion.

What caused the Black Death?

If both bubonic and pneumonic plague lacked the capacity to contact so many millions of people in such a short space of time across Europe then an alternative hypothesis must suggest an organism that could be easily transmitted from person to person during short contact periods as people went about their everyday business, produce a long infective period before the onset of illness so that as many people as possible could be infected, be less temperature-dependent than bubonic plague, less reliant upon other species as carriers and have clinical features that match at least some of those described by contemporary sources.

Recent work, examining the time-sequence of cases during the early phase of supposed bubonic plague epidemics in Penrith in 1597–1598, Eyam in 1665–1666 as well as a variety of other English outbreaks, some of them in London during the great plague years of 1603, 1625 and 1665, has provided such an alternative.⁴⁰ In both Eyam and Penrith the epidemics began with the arrival of a stranger to a specific house in the community. This newcomer became sick and died and from the timing of the next cases in those families and others an incubation period of approximately 37 days is indicated. This would not only rule out bubonic plague but provide a mechanism whereby an infected and infective person could move considerable distances, infecting those whom he contacted at fairs, markets and other public gatherings as well as within dwellings. This period of infectivity accords well with practice first adopted in the fourteenth century: the quarantine period in 1377 in the

Venetian colony of Ragusa was at first 30 days and later extended to 40 days, a period that was adhered to across Europe for 300 years. It is unlikely that this measure would have arisen in response to bubonic plague for the Italian medical authorities were among the foremost in Europe at the time and it is clear that they were referring to a different disease and to one that was infectious person to person.

Scott and Duncan have analysed many of the major epidemics in Britain and conclude that all exhibited the dynamics of an infectious disease with a long incubation period that contained latent and infectious periods of around 10–12 and 25–27 days, respectively.⁴¹ They concluded that the same agent was probably responsible for all the plagues in England between 1348 and 1666 and that it was a haemorrhagic virus, perhaps of the same type as the modern Ebola virus. They called this 'haemorrhagic plague'.

The haemorrhagic plague hypothesis, whilst denying the major role of bubonic plague in the Black Death, can nevertheless accommodate that disease within Europe although on a marginal basis. Bubonic plague was undoubtedly present in central and eastern Asia and sporadically extended along the Mediterranean coastline where the climate was warm enough to support extensive Black rat populations and a prolonged period for flea breeding. A French team has recovered the DNA of *Yersinia Pestis* from the teeth of nine skeletons from thirteenth to eighteenth century graves in the south of France, but their conclusion that this proves that the whole of the Black Death was bubonic plague is too sweeping.⁴² So far, all attempts to recover plague DNA from Black Death and early modern plague sites in London, Copenhagen and at two sites in France have been unsuccessful.⁴³

There is little doubt that an organism of some considerable virulence was involved but we should not ignore the fact that wider mechanisms are suggested during the fourteenth century. Europe was passing through a difficult period: the climatic change linked with a rapid cooling at high latitudes after 1300 and the growth of sea ice near Iceland was probably the main factor in the harvest failure of 1315 and the famine of 1316–1318. This probably began the slow population decline and by 1347 there were many settlements with uncultivated land and reduced populations. Between 1347 and 1350 settlements high in the Alps and in northern latitudes were deserted and it is unlikely that any of these had ever been inhabited by the Black rat. It is not impossible that the worsening climatic conditions favoured the presence of opportunistic organisms, both those that killed only people and others which were equally dangerous to domestic animals as well.

In addition to these various possibilities Jordan has suggested that the very high mortality of the Black Death in northern Europe might be in part a reflection of the fact that many people, in their 30s and 40s during the pandemic, had been young children in the years 1315–1322 and that the starvation they had experienced had rendered them more susceptible than those who were adults during the famine or who had been born after it.⁴⁴ Conversely, he argues, those regions such as Flanders that had suffered

epidemics between 1315 and 1322 in which large numbers of children died, might have far lower relative rates of mortality during the Black Death.

Conclusion

In the absence of direct mortality data indirect sources suggest that in some parts of the British Isles mortality during the Black Death was probably higher than in the Great Famine, the only other early fourteenth century period of population stress for which data are available. The consensus is that between 30 and 45 per cent of the population died. If this was so the mortality greatly exceeded that produced by any modern outbreaks of known plague, whether bubonic, pneumonic or septicaemic and in order to achieve this effect plague would need to have made contact with 75 per cent of the population. The absence of the Black rat over rural Britain makes this unlikely.

The characteristics of diffusion speed and epidemic length in the Black Death suggest an organism that was not only transmitted by personal contact but had a long infective period thereby enabling infected persons to pass the disease to many others. Plague caused by *Yersinia Pestis* is inappropriate to such a model.

Acknowledgements

I am grateful to many people for their helpful comments during the preparation of this paper, in particular P. Armitage, C.J. Duncan, P. Hordern, J. Rackham, J.D. Twigg and M. Vasold. My special thanks are due to Martin Ecclestone for his patient editing of the text.

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