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History of the Plague

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The Plague—An Introduction

The infectious disease known as the Plague is caused by the bacterium *Yersinia pestis*, a gram-negative, facultative anaerobic, rod-shaped bacterium belonging to the *Enterobacteriaceae* family. It is a zoonosis found chiefly in feral rodents, spread in the main by infected fleas. Plague epidemics in human populations can, however, occur when the *Yersinia pestis* pathogen invades rat populations that live in close proximity to humans. Once the rats have been largely killed off by the disease, the infected fleas move to human hosts.

In human infections, we differentiate between four possible forms of the plague: bubonic plague, septicemic plague, pneumonic plague, and abortive plague. Bubonic and pneumonic plague are the two most common variants. Bubonic plague is usually triggered by a bite from an infected flea. Following an incubation period of two to seven days, the first symptoms appear: severe malaise, fever, headaches and joint pain, and dizziness. Lymph nodes and lymphatic vessels in the area of the flea bite swell to form sores with a diameter of up to ten centimeters. These sores change color in the course of the sickness, often appearing blue or black due to internal bleeding in the lymph nodes. Without treatment, bubonic plague often leads to septicemic plague, in which the pathogens (*Yersinia pestis*) enter the bloodstream and thus attack the whole body. Septicemic plague causes large-scale bleeding in the skin and internal organs, and is almost always fatal without treatment. If pathogens spread to the lungs, pneumonic plague can develop (secondary pneumonic plague). In this form, pathogens can also be transmitted from person to person by droplet infection (primary pneumonic plague). The incubation period until the first symptoms appear can be from only a few hours up to a full day. Without treatment, pneumonic plague leads inevitably to death in between two and five days. By comparison, the abortive plague is rather less drastic; its symptoms are usually a mild fever and some swelling in the lymph nodes, and it provides long-term immunity to the plague bacterium.

The Plague, and thus its originator *Yersinia pestis*, have been linked to many epidemics in the history of humankind. The only completely proven incidence of the Plague, however, is the most recent worldwide outbreak, which occurred at the end of the

nineteenth century (the Third Pandemic); the *Yersinia pestis* pathogen was first identified during this pandemic (Yersin 1894).

Whether the Plague of Justinian in late antiquity (the First Pandemic) was caused by the same bacterium remains to be seen: molecular geneticists have so far not been able to find conclusive proof. The outbreaks known as the Antonine Plague (165–180 AD) and the Plague of Athens (430–426 BC) are generally assumed to have been expressions of other infectious diseases rather than the plague in its strictly medical sense. However, in recent years, the fields of molecular genetics and immunology have been able to provide ever more convincing evidence that *Yersinia pestis* was the pathogen that caused the Black Death (the Second Pandemic) in the Middle Ages (Raoult et al. 2000; Garrelt and Wiechmann 2003; Drancourt et al. 2007; Wiechmann, Harbeck and Grupe 2010; Haensch et al. 2010; Tran et al. 2011; Kacki et al. 2011; Schuenemann et al. 2011).

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Ole Jørgen Benedictow

The Origin and Early Spread of *Yersinia pestis* and of Epidemic Plague: Palaeobiological and Historical Viewpoints

A recent paper by a team of microbiologists headed by G. Morelli, Y. Song, and C. J. Mazzoni demonstrates the great opportunities offered by the new science of palaeobiology for advancing knowledge of the evolutionary development of infectious diseases—in this case, the bubonic plague—and the indispensable importance of interdisciplinary cooperation with historians (Morelli et al. 2010). They conclude convincingly that “plague originated in East Asia, perhaps in China Proper, more likely in Mongolia or Eastern Russia (Eastern Siberia) “[over] 2,600 years ago” and “spread through multiple radiations to Europe, South America, Africa, and Southeast Asia.” However, they also refer to the development of biovars and various strains in claiming that “the geographical sources and evolutionary branch order of [the biovar of] 2.MED[ievalis] subpopulations, which arose [over] 545 years ago” and that other data “supports the westward spread of 2.MED from China through trade articles that were carried along the Silk Road,” the extensive trade route from China to Western Asia “between 200 [BCE]¹ and 1400.” They also claim that “the estimated age of [the biovar of] 1.ANT[iqua]1 (628–6,914 years ago) slightly predates the extensive voyages from China led by Zheng He between 1409 and

1433. They conclude that, “It seems highly likely that these ships were infested by rats, which could have transmitted *Yersinia pestis* from China to Africa.”

A historian of epidemic diseases will note that, firstly, the indicated routes of spread are not supported by concrete, tenable evidence; in fact, they are at variance with the evidence, and out of chronological/historical sync with other data and events. Secondly, the references to bubonic plague in Chinese sources are both late and sparse when compared with the much earlier, quite numerous references in classical Greek, Hellenistic Greek, and ancient Roman sources. There are also substantially earlier references to bubonic plague, especially the Biblical account of epidemic events associated with the war between the Philistines and the Israelites in approximately 1100 BCE.

Morelli et al.’s biological determination of an East Asian origin of *Yersinia pestis* seems convincing and undoubtedly represents a major scholarly achievement. However, the fact that the earliest references by far to the bubonic plague concern regions on the Mediterranean littoral needs explanation, in order to make these seemingly disparate pieces of information compatible.

Morelli et al.’s attempts at explaining the historical spread of *Yersinia pestis* across the Eurasian continent by transportation along the Silk Roads, or transportation to Eastern Africa by Zheng He’s maritime expeditions in the period 1409–1433, must be deemed untenable. There are also serious problems or at least weaknesses associated with their dating of the time of its origin, and the spatio-temporal pattern of its spread, using biomolecular clocking techniques.

This calls for a possible alternative explanation(s) of how *Yersinia pestis* could have spread out of its original homeland in East Asia, which should be compatible with the spatio-temporal perspectives provided by historical sources. Such an explanation could be based on a notion of the natural pace of the spread of plague disease, caused by *Yersinia pestis*, in sylvatic (wild) rodent populations.

A similar event can serve as a useful basis for clarification of this question. In June 1900, plague entered a new continent, namely North America, in San Francisco. Seventy-five years later, plague had penetrated into 12 states and had crossed the middle of the United States at 100° longitude, a distance of around 2000 km (1250 miles) from

San Francisco, and had also invaded Canada in the north and Mexico in the south. The average spread rate had thus been of the order of 25 km (16 miles) a year. McNeill (1979, 152–55, 272) concludes his discussion by saying that “the geographic spread of plague infection in North America occurred naturally.” It was basically passed on between colonies of ground-burrowing rodents.

At a similar pace of spread, plague would have covered the roughly 8000 km from a central location in East Asia to the Middle East in about 320 years; or about 400 years at 20 km per year. This estimate can now be juxtaposed with Morelli et al.’s conclusions that “*Yersinia pestis* evolved in or near China” over 2,600 years ago, but also that 1.ANT1 developed 628–6,914 years ago; they seem to envisage an age of *Yersinia pestis* in East Asia 6,914 years ago, and that the real age would include the time of its spread to East Africa and the development of the biovar. The natural spread rate of plague is compatible with the historical data, and within the enormous margins of uncertainty inherent in biomolecular dating techniques.

In the Middle East, *Yersinia pestis* would become connected to the black rat and *Xenopsylla cheopis*, “the rat flea *par excellence*,” as it has often been called. Black rats and their fleas have been central in all plague epidemics studied by modern physicians and epidemiologists and the specific defining features of rat-borne plague can be observed with respect to historical plague epidemics. Maps of the global distribution of plague foci of various species of wild rodents show that they stretch almost continuously from Manchuria to the Middle East. After arrival in the Middle East, *Yersinia pestis* could easily have been spread by human agency to western Arabia and further to Central East Africa, triggering the formation of plague foci there.

Clearly, the bubonic plague pathogen originated in East Asia and spread westwards from there. The epidemic powers of plague (as distinct from the epizootic powers) may have evolved in the Middle East, where the black rat and *Xenopsylla cheopis* were present, producing the basic requirements for the adaptation and spread of the plague in human populations. In order to become an epidemic disease spreading in human habitats the disease would have to be introduced among rodents in human habitats with fleas that would also attack and transmit the disease to human beings. Conditions in the Middle East were well suited to allow a transformation of the patterns of the spread of plague. If so, this could have triggered a new phase of plague that spread back

eastwards, with profound epidemic implications that may not be easily identifiable with palaeobiological tools.

Clearly, *wherever* plague's ancestral homeland was, the temporal perspectives provide superabundant time for plague to spread westwards all the way across the Eurasian continent, cross into Africa, and establish the plague foci of the Middle East, the western coastal areas of Iran and Arabia, and Central Eastern Africa. The establishment of these foci would constitute the basis for the biblical account of pre-classical plague, and the classical plague epidemics and clinical descriptions of plague in classical medical works.

This transcontinental spread of *Yersinia pestis* could quite likely have occurred long before antiquity and have caused spread all the way back to China, in time to establish the epidemic combination of the black rat and *Xenopsylla cheopis* and the necessary conditions for plague epidemics much earlier—perhaps even thousands of years earlier—than the “2,600 years ago” suggested by Morelli et al. It raises essential and interesting questions about the possible adaptations between the black rat and *Xenopsylla cheopis* and the biovars and various strains of *Yersinia pestis* encountered on such a journey. Would significant adaptation by selection be necessary at all? Or would the existing adaptations selected by *Yersinia pestis* in relation to various combinations of rodent fleas and their sylvatic rodent hosts function readily and well with *Xenopsylla cheopis* and the black rat?

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Raffaella Bianucci and Sacha Kacki

The Archaeology of the Second Plague Pandemic: An Overview of French Funerary Contexts

Over the last 20 years, several plague mass graves have been unearthed in France, thus enhancing our knowledge of historical plague pandemics. Moreover, recent archaeo-anthropological and palaeoimmunological investigations have shown that abrupt mortality crises caused by plague have been handled differently in urban and rural communities (Kacki et al. 2011). In this paper, we report on the different funerary contexts and the related practices adopted by some French urban and rural communities during the Second Pandemic (1348–1722 CE).

Several eighteenth-century urban plague burial sites have been uncovered in France. Most of them date to the “Great Plague,” which occurred in Provence and Languedoc during 1720–1722. It has been estimated that the Marseilles “Great Plague,” which started on 20 June 1720, affected 242 localities and resulted in 119,811 deaths out of a population of 394,369 individuals (Signoli et al. 2002). In urban centers, such as Marseilles (“The Observance Pit” and “The Major Pit”) and Martigues (“Le Délos” and “Le Couvent des Capucins”), plague epidemics caused the death of dozens or even hundreds of people daily. This led to the digging of large pits or trenches, where the deceased were buried. We can pinpoint a number of differences in the funerary treatments of these eighteenth-century victims, mainly linked to the patients’ admission through the plague infirmaries. To sum up, two different kinds of plague victims can be identified within the mass burials:

1. Those skeletons found together with personal belongings correspond to the plague victims who died outside the infirmaries and who were found dead several hours, or even days, later. They were buried fully dressed, along with their belongings, because gravediggers wanted to minimize contact with the infected bodies.
2. Skeletons found without any personal belongings correspond to the victims who died in the plague infirmaries and were buried according to the sanitary restrictions adopted during the plague epidemics, i.e., undressed and wrapped in shrouds. The transit through the plague infirmaries resulted in a slower rhythm of inhumations,

which corresponded, in general, to the period of time between the declaration of the patients' contagion and their deaths. This, in turn, resulted in a more rational handling of the infected corpses.

Working backwards from the eighteenth to the seventeenth century, and moving away from urban contexts to rural ones, we have a single example of a plague cemetery at our disposal. This site, located close to the Puy-Saint-Pierre village (Hautes-Alpes), dates back to the plague epidemic that devastated the upper Durance River valley between 1629 and 1631 (Signoli et al. 2007). In this plague cemetery, there was an attempt to maintain the customary funerary practices, with interments in individual graves. Whenever higher rates of mortality were reached, double graves were dug; and, finally, multiple graves were dug at the highest peak of the epidemic. It is likely that this cemetery was connected to a nearby infirmary. The construction of temporary wooden burial structures was common in France, starting from the plague epidemics at the end of the fourteenth century. These structures were built in haste at the very beginning of the contagion and were burned at the end of the epidemic.

For the period between the fourteenth to the sixteenth centuries, very few data concerning the impact of plague on the demographic structure of rural communities are available, although 90 percent of the French population was living in the countryside at that time (Benedictow 2004). Therefore, only archaeological data can provide information both about the spread of plague in rural areas and about changes to funerary practices during the epidemics. However, few examples of French rural plague cemeteries are known so far, which might be influenced by the demographic structure of rural populations. In fact, while plague epidemics caused the deaths of several dozens or hundreds of people daily in urban centers and often led to the digging of huge plague pits or trenches, in small rural communities the death toll was much lower. The number of victims to be buried was thus far smaller. It would therefore be managed much more easily by the gravedigger(s), and this made it easier to uphold the tradition of interment in single graves.

One example of the ordered and rational handling of plague victims achieved by the digging of a majority of single graves has been reported at "Les Fédons" rural plague cemetery (Bizot et al. 2005). This graveyard was associated with a plague infirmary and dates to the outbreak that occurred in Lambesc, southeastern France, between April and September 1590. Almost all corpses were laid in dorsal decubitus position (i.e., lying

stretched out on their backs) with their heads oriented to the east. Once again, it appears that the funerary practices observed here are similar to those adopted in a context of normal mortality.

Finally, concerning the “Black Death” rural plague cemeteries, there is evidence so far of only two cemeteries, which are located close to the villages of Vilarnau (Passarius et al. 2008) and Saint-Laurent-de-la-Cabrerisse (Haensch et al. 2010; Kacki et al. 2011). In both cemeteries, most of the plague victims may have been buried in single graves, and mass graves were dug only when the mortality rate peaked. Even in these cases, most of the individuals were deposited in dorsal decubitus position, showing the same orientation as the other deceased (fig. 1). This shows that, although the increased mortality may have led to the simultaneous inhumations of several individuals in the same pit, customary funerary practices were not substantially modified.



Figure 2: Positive identification of *Yersinia pestis* F1 antigen and *caf1* gene in sample SLC1006 from Saint-Laurent-de-la-Cabrerisse burial site.

Where plague victims are buried in single graves and the usual funerary practices maintained, no archaeological evidence allows us to distinguish between plague burials and ordinary graves. On these grounds, the application of biological techniques such as RDT and PCR may permit us better to characterize the demographic impact of plague on rural communities (fig. 2).

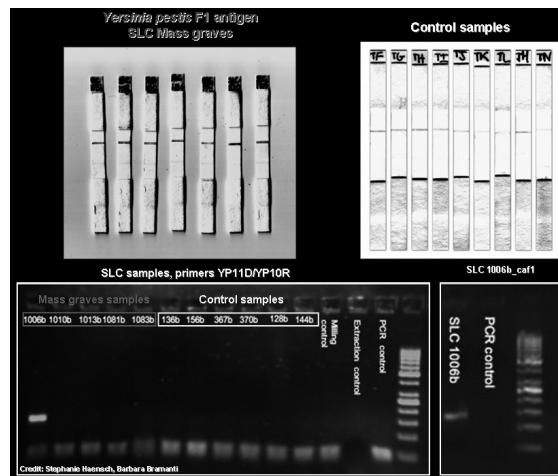


Figure 1: Double grave SLC141 from Saint-Laurent-de-la-Cabrerisse burial site. The excavation of double graves might correspond to punctual peaks of mortality. © A. Gaillard (Acter archéologie) / S. Kacki (Inrap).

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