



# Pharaonic Egypt and the origins of plague

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## ABSTRACT

**Aim** This paper examines the possibility that bubonic plague was a disease endemic in the wild rodent population of Egypt and East Africa.

**Location** The study focuses on Egypt and the Nile Valley during the Pharaonic period.

**Methods** This paper presents a hypothesis based on archaeoentomological, archaeozoological and biogeographical information on the insect and small mammal species involved in the spread of plague, as well as relevant information from early literary sources.

**Results** The primary host for the rat flea *Xenopsylla cheopis* is the Nile rat, *Arvicanthis niloticus*. Urbanization and the Nile floods brought into contact humans and the Nile rat and its ectoparasite, *Xenopsylla cheopis*, which was able to move to a newly introduced host the synanthropic *Rattus rattus*. The existence of large numbers of human fleas and squalid conditions from the Workmens' Village at Amarna, evidence for Nile rats and black rats from Pharaonic sites and descriptions of an epidemic disease in the Amarna letters, the Hittitic archives and the Ebers papyrus with references to swelling buboes, present a new scenario for the origins of the disease.

**Main conclusions** Most modern researchers have regarded the origins of bubonic plague as a disease of Central Asiatic rodents. Here, I examine the evidence for plague in Egypt, and suggest that the bacillus *Yersinia pestis* was primarily a disease of the Nile rat, *Arvicanthis niloticus*, which only achieved epidemic proportions when its vector, the tropical rat flea, *Xenopsylla cheopis*, was able to make the jump to a new host, the black or ship rat, *Rattus rattus*, introduced from India or indirectly via Mesopotamia during the Pharaonic period. Synanthropy and a high death rate in the new host lead to frequent transfer to human populations and stochastic waves of pandemics.

## Keywords

**Bubonic plague, *Xenopsylla cheopis*, *Rattus rattus*, *Arvicanthis niloticus*, Pharaonic Egypt, Nile floods, urbanization.**

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The origins and dispersal of epidemic diseases have been the subject of much speculation (e.g. Zinsser, 1935; Brothwell & Sandison, 1967; McNeill, 1977; McKeown, 1988; Nikiforuk, 1991). Whilst the archaeological excavation of multiple apparently contemporary burials in pits may provide at the least circumstantial evidence for the occurrence of mass death by disease, most infections leave no specific trace upon the bones, and the causes of death cannot be established. Mummification, however, may lead to the preservation of

evidence for diseases that otherwise leave no trace. Smallpox, tuberculosis, malaria, poliomyelitis and schistosomiasis have been noted from individual Egyptian mummies (Contis & David, 1996; Sandison & Tapp, 1998). Where contemporary written accounts exist, interpretation of the sources is often difficult. Procopius' (1914) description of the 6th century Justinian epidemic in the Eastern Mediterranean is usually regarded as sufficiently detailed to enable the identification of bubonic plague, at least in Constantinople (for popular

accounts, see Ziegler, 1998; Keys, 2000), but earlier plagues are less easily diagnosed. There has been much discussion for example over the exact nature and origins of the plague described by Thucydides in 5th century BC Athens (cf. Longrigg, 1980), and the biblical plague of the Philistines has similarly attracted attention (Shrewsbury, 1964; Goedicke, 1984).

As Busvine (1976) notes, from an evolutionary viewpoint it is not in the 'interest' of any pathogen to generate the large-scale death of its host, and the primary host is likely to have been a species, or group of species, with which the disease has co-evolved in a relatively passive state. The pathogen jump to a new host, usually the result of biogeographic change instituted by humans, leads to the development of new epidemic diseases (Cohen, 1989). Frequently the new population of hosts, lacking the resistance built up by the close evolutionary association between primary host and parasites, suffers high mortality. Once established in a new host, strains of the pathogen may evolve along other pathways, which make it virtually impossible to trace back to either the original host or the primary source areas of the infection. Strains may be rapidly disseminated pneumonically, or their other hosts become cosmopolitan domesticated or synanthropic species, closely associated with people. In addition, new, more virulent strains may emerge in areas remote from the region of primary distribution of the disease and of the host with which the disease initially co-evolved, creating new centres of dispersal. Where an intermediate arthropod vector is involved, an additional line of evidence, albeit also largely circumstantial, becomes available in that fleas, ticks and lice have been increasingly recovered from archaeological deposits (Panagiotakopulu, 2001a).

Buckland & Sadler (1989) have discussed the biogeography of the human flea, *Pulex irritans* L., beginning with material from sites in Greenland and arguing for a New World origin. The recent publication of findings from the New Kingdom Workmen's Village at Amarna, Egypt, the earliest material from the Old World (Panagiotakopulu, 2001b), provides evidence that *P. irritans* had expanded across the Palaearctic by the middle of the second millennium BC. In addition to a very rich coleopterous and dipterous fauna, Amarna has produced large numbers of ectoparasites, mainly fleas and bedbugs, but also a tick. Five specimens of bedbug, *Cimex lectularius* (Panagiotakopulu & Buckland, 1999), 35 human fleas, and one cat flea, *Ctenocephalides felis* (Bouché), were recovered from samples from the Workmen's Village. Continued archaeoentomological research on Amarna and similar sites on the edge of the desert is producing further material (cf. Panagiotakopulu, 2000), and the potential for the study of arthropod-vector diseases is considerable.

Whilst the bed bug has not been implicated in the spread of epidemic disease, fleas have been connected with several, especially plague. *Pulex irritans* in particular may become the vector in the spread of human infection (Gratz, 1999). The role of the black rat flea, *Xenopsylla cheopis*, in the transmission of the plague bacillus, *Yersinia (Pasteurella) pestis*, was recognized

in India towards the end of the 19th century (Hirst, 1953). Whilst several authors have argued that plague epidemics in the past were not the result of this bacillus (e.g. Twigg, 1984; Scott & Duncan, 2001), the metapopulation model of infected flea, rat and human populations recently developed by Keeling & Gilligan (2000), effectively explains the stochastic nature of plague outbreaks. The origins of the medieval plague, the Black Death or bubonic plague, which devastated Europe during the 14th century, have been linked with the spread of black rats from the East (Ziegler, 1998), where the fossil record of both black, *Rattus rattus* L., and brown rat, *R. norvegicus* Berk. has been claimed to extend back into the Pleistocene (Kurtén, 1968, but see Eryvncck, 2002). McKeown (1988, p. 56) suggests that the earliest recorded pandemic, that of the late sixth century AD, had its origins among wild gerbil populations in eastern Asia, although contemporary sources suggest either an Egyptian origin, from the port of Pelusium at the eastern limit of the Nile Delta (Procopius, 1914, *History of the Wars* II, xxii, 6–7), or an Ethiopian, from the kingdom of Axum (Evagrius, 1898, IV. 29). Ethiopia was also thought to have been the source of the Athenian plague according to Thucydides' (1928) account (2.47–2.55). Ziegler (1998) follows Pollitzer (1954), in suggesting that wild rodent populations in the area near Lake Issyk Koul in the district of Semirichinsk in Central Asia provided the cradle for the epidemic, beginning in 1338. From there it is believed to have spread eastwards and southwards to China and India and westwards to the Crimea, from thence to the rest of the Old World (Ziegler, 1998). In 1347, it arrived in Egypt, allegedly with slave traders from the Black Sea. The plague may have killed one quarter of the European population, and was equally destructive from Egypt to Morocco. Smaller epidemics occurred up to 1600 with a pandemic from 1663 to 1668. Between 1347 and 1517 there were 55 recorded plague outbreaks, among them 20 epidemics (Dols, 1983; Rodenbeck, 1998). The subsequent decline of epidemic plague at least in Western Europe has been seen as a result of the expansion of the brown rat, which rarely carries the flea, and improvements in hygiene in the post-medieval period. The pattern of a high initial kill rate and later episodic localized outbreaks does suggest the arrival of a new pathogen, to which the host has little resistance, although Keeling & Gilligan's (2000) model does allow for stochastic local extinction of the disease in the face of declining black rat populations. The tropical rat flea *X. cheopis*, can live away from its host for a month in ideal conditions of temperature and humidity without the need to feed on its host, and it may be transported considerable distances, for example in grain or together with cloth. Starving and aggressive, the flea would bite any possible host (Rothschild & Clay, 1952) at the point of disturbance, and it is therefore an effective vector in disseminating plague to areas where the black rat is not established, although such outbreaks are likely to be limited to port and trade areas, perhaps with a few additional pneumonically spread cases dispersing into the countryside.

The simple model of the dispersal of the medieval Black Death on the back of the primary expansion of a strongly

synanthropic animal, able to exploit both ships and ports, however, has been called into question by archaeological finds of black rat in Roman contexts in northern Europe. Rackham (1979) first drew attention to its presence in securely stratified late Roman deposits in York, and subsequent finds from York, Wroxeter and London show that it was present not only at coastal sites but also inland by the third century AD (Armitage *et al.*, 1984; O'Connor, 1992); finds show its presence in the Anglo-Scandinavian period in York, but O'Connor (1991) has argued that these stem from ninth century reintroductions. The evidence, however, shows that black rats were firmly established in Europe long before the Black Death (Armitage *et al.*, 1984; Armitage, 1994; Ervynck, 2002), and other hypotheses of plague origins should be explored.

In their study of the London material, Armitage *et al.* (1984) note that Tchernov (1984) has recorded black rat remains from Natufian cave deposits in Palestine, and that although most records from Egypt are Ptolemaic, fourth to first centuries BC or later, Boessneck (1976) noted the species amongst the 17th to 16th century BC material from Tell el-Dab'a in the Nile Delta (Table 1). The propensity of rodents for burrowing into sediments means that single records need to be treated circumspectly, and Armitage (1994) expresses some doubts about the material from Palestine. However, the six from the stomach of a mummified cat in Roman deposits from Quseir el-Qadim on the Red Sea coast of Egypt provide a secure group. Armitage *et al.* (*op. cit.*) also query von Niethammer's (1975) chromosomal evidence that the form of the black rat which has become cosmopolitan originates in the Deccan of southern India. The evidence has recently been extensively reviewed by Ervynck (2002), who whilst doubting many records, including the Tell el-Dab'a one, adds material from Mesopotamia from as early as the mid-third millennium BC. Ervynck's discussion is affected by a premise that commensalism in rats reflects an evolutionary change similar to domestication rather than the adaptation of a man-made habitat which mimicks its wild ones. It is to the latter group which most if not virtually all synanthropic animals, both vertebrate and invertebrate (cf. Buckland, 1991), belong. The rat's introduction to the Mediterranean world therefore may have been either a direct one through immediate trading connections across the Indian Ocean to the Red Sea ports of Egypt, or

via the intermediary of trade with Mesopotamia. Connections were considerable (Potts, 1990), and other synanthropic species may have used the same route (Panagiotakopulu, 2003).

The question can now be raised as to whether *Rattus rattus* came with *Xenopsylla cheopis*, or acquired it from another rodent there. Patton & Evans (1931, p. 534) have suggested that the flea was endemic to the Nile Valley, and several other members of the genus are essentially African in their core distribution (Smit, 1973). Whilst there is a tendency to connect the spread of bubonic plague solely with the fleas of the black rat, *X. cheopis* can live on a number of rodents and lagomorphs, and black rat is not the only animal that can carry the flea and disease. Carnivorous animals that eat rats may be infected or be relatively passive carriers. Cats and guinea-pigs, for example are likely to die of the plague, whereas dogs only die in very severe epidemics (Rust *et al.*, 1971). The death of domestic animals as well as humans in the Thucydidean plague of Athens might also suggest that this epidemic was bubonic plague, although his description, perhaps relating to his own experience, suggests septicaemic rather than bubonic form. Whilst the tropical rat flea is the best vector of the disease, in urban situations the human flea, *Pulex irritans*, and the cat flea, *Ctenocephalides felis*, can act as carriers (Smit, 1973; Gratz, 1999). In other words, where there are other suitable rodent hosts, black rats may not be necessary for the spread of the disease.

The fact that plague usually has a high kill rate in its black rat hosts (Keeling & Gilligan, 2000) implies that, like humans, they are not the primary host for either the disease or its vector. In a parallel situation, Buckland & Sadler (1989) have argued that the human flea was acquired from guinea-pig in South America, when the two first came into contact, and *P. irritans* was then able to spread to the Old World initially by a process of gift exchange of furs (Panagiotakopulu, 2001a), and then on its new human hosts. In the case of the black or ship rat, introduction to Egypt from India would have brought it into contact with several other species of rodent from which it could have acquired both fleas and plague. The most obvious of these is the Nile rat, *Arvicanthis niloticus* (Desmarest), which provides a natural reservoir of plague throughout the Nile Valley and into East Africa (Davis *et al.*, 1968; Gage, 1999). The species carries both flea and plague but has a high level of immunity from the disease.

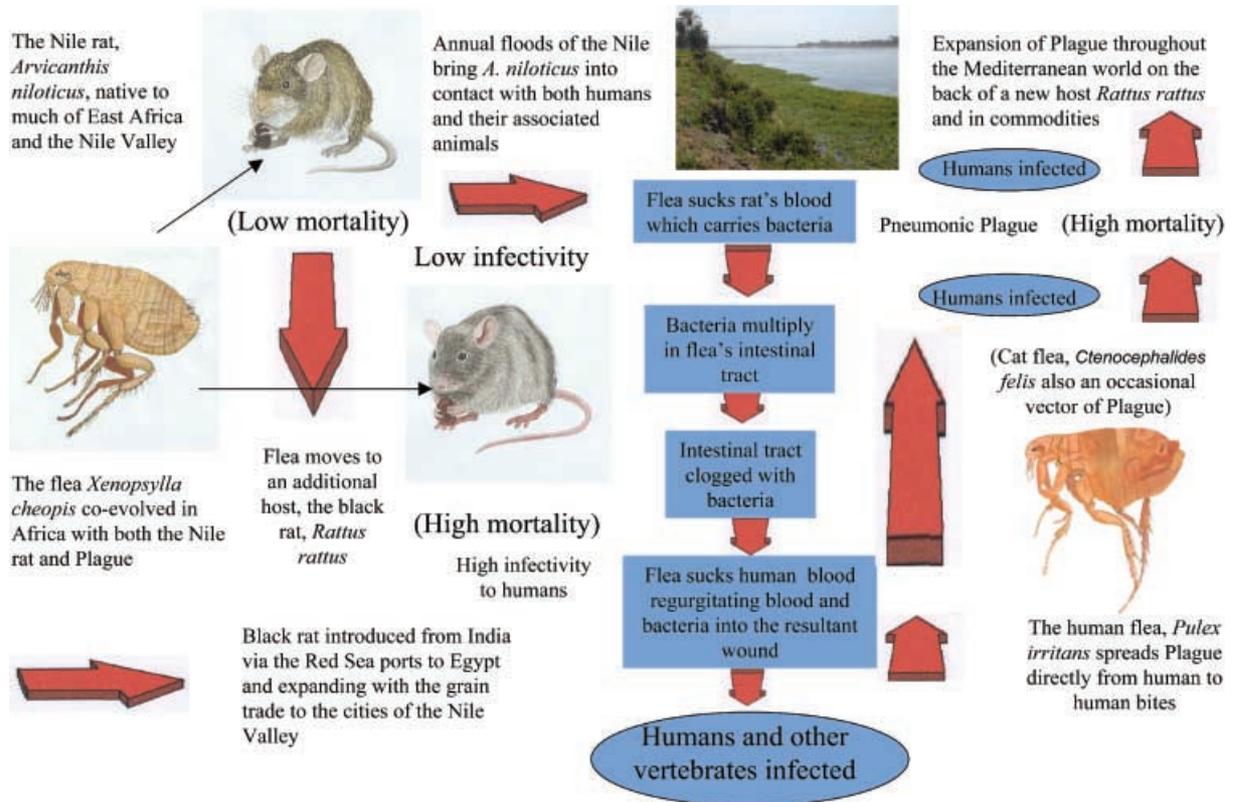
**Table 1** Finds of black rat, Nile rat and a possible plague case from ancient Egypt

Site	Date	Rats	Fleas	Bubonic plague	Reference
Tell el Dab'a	1700–1600 BC	?Black rat? Nile rat			Boessneck, 1976
Debat el Eheima	1600–1000 BC	Nile Rat			Gautier & Van Neer, 1998
Tell el Amarna	1350–1323 BC		Human fleas and cat flea		Panagiotakopulu 2001b
Egypt	Ptolemaic			Mummy?	Ruffer 1910
Alexandria	Ptolemaic	Black rat			von den Driesch & Boessneck, 1983
Quseir el Qadim	Roman	Black rats (in stomach of mummified cat)			von den Driesch & Boessneck, 1983

The Nile rat is recorded archaeologically from Debat el-Eheima in the southern Sudan (1600–1000 BC) (Gautier & Van Neer, 1998) and from Tell el Dab'a (Boessneck, 1976). A rat is depicted in the 11th dynasty rock-cut tomb of the monarch Baket III (no. 15) at Beni Hasan, opposite a cat (Houlihan, 1996, p. 83), and apparently it was familiar to Egyptian households since at least the Middle Kingdom. Despite Houlihan's (1996) identification of the animal as a genet, the painting in the 18th dynasty tomb chapel of Menna (no. 69) at Thebes clearly shows a Nile rat in its natural habitat amongst papyrus (Houlihan, 1996, Pl.XXIV). The annual Nile flood, driving rats from their riparian habitat into fields and settlements, would have provided ample opportunities for the flea to make the species jump to the black rat, and later trade would take the rat and its newly acquired parasite on, across the Mediterranean and into Asia. Urban situations, with their concentrations of people, their detritus and rats, are ideal for the spread of epidemic disease, and trade may be a key activity for turning a local epidemic into a pandemic. There are several foci where plague is endemic in local populations of rodents, not only in Africa, but also for example in Siberia, China, Burma and Vietnam. Serological analysis can be used to track the immunity levels of rodents and to determine whether the disease is endemic or not. The results of the investigation of South African and

East African mammals have indicated that plague is endemic to Africa (Davis *et al.*, 1968; Shepherd & Leman, 1983). The Nile rat appears relatively immune to the plague bacillus (Gratz, 1999), and modern studies have shown that as few as 20 infected animals can trigger plague in a human community. The hypothesis advanced here is that plague began as a disease of the Nile rat in Egypt, was acquired from its flea, *X. cheopis*, by black rat accidentally imported from India, which then became the reservoir in later episodic pandemics. Figure 1 provides a summary of this model.

It remains to consider the evidence for plague in Pharaonic Egypt. The only physical evidence was first noted by Ruffer (1910) in a Ptolemaic mummy as a case of pneumonic plague. Both Rowling (1967) and Sandison & Tapp (1998) regard the case as inconclusive, but if correct, this would indicate the presence of the disease at the time of the earliest widespread black rat records from archaeological assemblages in Egypt, although this may be purely coincidental as the literary evidence, difficult to interpret although it is, suggests earlier cases. Filer (1995, 17) has stressed the difficulty of interpreting the evidence presented in Egyptian sources for diseases and most are questionable. Perhaps the first time plague is mentioned is in the Ebers Papyrus, a medical papyrus dated to around 1500 BC, but probably compiled much earlier:



**Figure 1** Flow diagram illustrating the pathways from a local disease of Nile rat to black rat to humans and the origins of a pandemic. Drawings by Pete Skidmore.

If thou examinest a man who suffers from the said (i.e. from the shivering fit described in 38, 3–10) for hours, like consuming for purulency, and he is weak like a breath that passes away, then thou shalt say that it is (due to) closing (?) of an accumulation, which cannot be raised and does not trust in a weak remedy; it (i.e. the accumulation) has produced a bubo, and the pus has petrified, the disease has hit. Thou shalt prepare him remedies to open it by means of medicines. (Ebers Papyrus 39, translated by Ebbell, 1937)

Another tentative description of a disease that could be the plague is found in the Hearst Medical Papyrus, a text palaeographically related to the Ebers Papyrus, and dated to c. 1520 BC: one of the incantations is against 'the Canaanite illness':

Who is knowledgeable like Re? Who knows the like of this God? – when the body is blackened with black spots – to arrest the God who is above. Just as Seth had banned the Mediterranean Sea, Seth will ban you likewise, O Canaanite illness! You shall not intend to pass through the limbs of X, born of Y. (Hearst Medical Papyrus H XI 12–15, translated by Goedicke, 1984, p. 94)

In the London Medical papyrus, dated to 1350 BC, there are incantations 'in the language of Keftiu' against the 'Canaanite illness' (Goedicke, 1984). A further possible description of the disease could be in the incomplete section of the same papyrus:

When the body is coal black with charcoal (spots) in addition to the water (urine) as red liquid (i.e. bloody)... (London Medical Papyrus 15, 8–10, translated by Goedicke, 1984)

Goedicke (1984) is definitive that this disease is bubonic plague, although the evidence is insubstantial. The disease was probably called the Canaanite or Asiatic (Amu) illness, either because of the place in which it started, or simply because everything deleterious tends to be blamed on somebody else. The case of syphilis, Shakespeare's 'Malady of France' (Henry V, Act V, Scene II), provides a more modern example of the same.

One of the Amarna letters, a collection of incoming diplomatic correspondence sometimes heavy on exaggeration, also refers to a plague. In letter EA 35, the king of Alasia (?Cyprus) explains why the amount of copper he sent to the king of Egypt is small:

Behold the hand of Nergal (pestilence, plague) is now in my country; he has slain all the men of my country, and there is not a (single) copper-worker. (translated by Moran, 1992)

These references have been extended to include Egypt by several authors attempting to explain the eccentricities of the

18th dynasty, from the foundation of the new capital on a pristine site at Amarna c. 1350 BC by Akhenaten to the failure of the pharaoh to intervene on the side of its allies in Syria and Palestine (cf. Goedicke, 1984). Certainly there is evidence of a plague in the contemporary Hittite kingdom in eastern Anatolia. The epidemic killed both Suppiluliuma and his son and successor Arnuwanda II (Kuhrt, 1995, pp. 275–6). According to the plague prayers of Mursili II (c. 1330–1295 BC), it was passed on by Egyptian prisoners taken at the battle of Amka in Syria, and it raged among the Hittites for 20 years (Pritchard, 1955, pp. 394–6):

a.5. When they moved the prisoners to Hatti land these prisoners carried the plague into the Hatti land. From that day on people have been dying in Hatti land... (translated by Goetze)

In more popular works, there is a tendency to accept the circumstantial evidence that plague reached Egypt and had a significant impact (e.g. Aldred, 1988), although there is no concrete evidence, either epigraphic or archaeological.

The total abandonment of Amarna after about 20 years provides an ideal opportunity for palaeoecological research, particularly since the site lies on the edge of the desert with near total preservation. Older excavations were aimed at recovering the plan of the city and artefacts and little note was taken of plant and animal remains. More recent work, however, directed by Barry Kemp of the University of Cambridge on behalf of the Egypt Exploration Society, has begun to add this component to the research (cf. Kemp *et al.*, 1994). In the few small samples, examined from the Workmen's Village on the edge of the city the high frequency of human fleas (Panagiotakopulu, 2001b) and bed bugs (Panagiotakopulu & Buckland, 1999) implies that ectoparasite infestation levels were high, and conditions were ripe for the spread of epidemic disease. The remainder of the insect faunas, dominated by dipterous puparia and coleopterous predators on maggots, also present a picture of squalid living conditions around the houses.

## CONCLUSIONS

Coexistence of the Nile rat, humans and ectoparasites in urban centres in combination with trade with Asia, Africa and the Mediterranean, together with the Nile floods and the introduction of the black rat, circle Egypt as the most probable place of origin of bubonic plague as an epidemic disease. Jumping initially from the Nile rat to the introduced black rat, *Xenopsylla cheopis*, may have carried with it a disease previously rare in humans, but thereby provided with an opportunity to become epidemic. It is unfortunate that archaeologists working in Egypt have rarely sieved their sediments to recover small mammal bones and even more rarely have they been identified. Further, detailed palaeoecological and pathological research will be necessary to substantiate this model.

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## BIOSKETCH

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