1

A Natural History of Plague

Ne pire her biri bir zerk idici div-i hücum
Ne pire bilmez aman, vermez aman cana kıyar
Pire bir heybet ile halka hücum eyledi kim
Div bu yerde eğer bağlasalar ide firar

O'what a flea! Each one is an injecting attack-demon
O'what a flea! Has no mercy, shows no mercy, takes life
Fleas attacked people with such majesty that
Demon would flee this place even tied down[1]

The opportunity to write a natural history of plague in the Ottoman lands is a mixed blessing. On one hand, there is an embarrassment of riches in both the scientific and historical literature on plague, which is generally helpful for understanding plague's emergence, transmission, and effects. On the other, this literature has little bearing on the Ottoman experience of plague in the late medieval and early modern eras. My task in this chapter is to reconstruct a natural history of plague in the areas where the Ottomans came to rule, drawing from the scientific literature and from Ottoman sources. This effort will involve highlighting plague's main protagonists (its host and vector organisms and its causative agent or pathogen) and their interactions in the context of the physical, climatic, and environmental conditions of Ottoman history.

Plague is a zoonosis (animal-to-human disease) that primarily affects rodents; humans are only accidental hosts to it. It has a complex etiology that involves a system of entanglements between rodent hosts, arthropod vectors, the pathogen, human populations, and the environment. These agents interact with one another, while they themselves change in response to their

[1] Evliya Çelebi, *Evliyâ Çelebi Seyahatnâmesi* (Istanbul: Yapı Kredi Yayınları, 1999), 4:214–15. Evliya Çelebi claims to quote these couplets by Baba Abdi-i Horasani, composed during the latter's visit to Balıkesir, complaining of the fleas there.

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interactions with other organisms and the broader environment. To get a holistic sense of this dynamic, it may be useful to consider the actors one by one. However, before introducing plague's protagonists, it may be useful to comment briefly about the phases of epidemiological activity, especially as they manifested in the Ottoman case. When historical sources mention outbreaks of plague, they are referring to the epidemic phase of the disease, that is, when it affects human populations, causing a certain degree of mortality. Even without any attempt to control, contain, or cure the disease, the epidemic phase of plague does not last very long. In the Ottoman areas with moderate climate, plague epidemics typically started slowly, gradually peaked, and then receded. The climatic and environmental conditions of a city could shape plague's *seasonal signature* (the temporal patterns of epidemic activity) – an unambiguous marker of plague. As far as the early modern Ottoman cities are concerned, we know fairly well how this took place, as did the Ottomans themselves. For example, in Istanbul, plague would typically start in April or May, peak in August and September, and recede in November and December. In the warmer temperatures and (higher) humidity of Thessaloniki, the right conditions for sustaining such outbreaks were between April and July; plague would normally break out in March, peak in June and July, and lose intensity by late summer to recede in September or October. In yet warmer cities, the timing could be slightly earlier. For example, in Alexandria, plague would usually break out in January, peak in April, and finish in June or July.[2]

Decreasing mortality signaled that the plague was on the wane and the end of the *plague season*, but not necessarily the end of the epidemic. The disease could return the following spring and follow a similar seasonal pattern. Hence, the epidemics came and went in waves, sometimes lasting several

[2] Panzac, *La peste*, 223–25, 628–29. Panzac noted that plague started in Istanbul when temperatures reached 11–12 degrees Celsius with an average humidity between 67 and 79 percent throughout the year; peaked in August when temperatures reached their maximum average (23.9 degrees); and continued in the fall with milder temperatures (20.2 degrees) and a high level of humidity (70 percent). As for Thessaloniki, plague started when temperatures were around 11 degrees Celsius and humidity between 60 and 70 percent. It peaked in June when temperatures were 23.9 degrees and humidity was around 50–55 percent. Increasing temperatures in July (27 degrees Celsius) and a fall in humidity (57 percent) impeded further activity of plagues. For the seasonality of plague epidemics in Thessaloniki, see Eleni Xoplaki et al., "Variability of Climate in Meridional Balkans during the Periods 1675–1715 and 1780–1830 and Its Impact on Human Life," *Climatic Change* 48, no. 4 (2001): 581–615, esp. 584–87. As for Alexandria, Panzac indicated that it broke out in January when both the temperature (14.4 degrees Celsius) and humidity (66 percent) were high enough, continued into a mild spring, and ended when higher temperatures were reached in June and July. Compare with the discussion "the season of plague" in Egypt in Alan Mikhail, "Plague and Environment in Late Ottoman Egypt," in *Water on Sand: Environmental Histories of the Middle East and North Africa*, ed. Alan Mikhail (Oxford: Oxford University Press, 2012), 120–23. For the most part, these figures for temperature and humidity represent the eighteenth and nineteenth centuries and may need to be compared with those of earlier eras of Ottoman history.

years in a row, at other times skipping a year or two but returning again later. Epidemic waves were separated by interepidemic phases of nonactivity (or low-level activity) among the human populations, depending on a variety of factors, including climate, the availability of a replenished pool of human and rodent hosts that lacked immunity, and flea-to-host ratios.

The epidemic phase of the disease, however, is not its only manifestation. In a plague focus or reservoir, the disease is sustained by ground-burrowing rodents that are resistant to the infection (*enzootic* phase), until it breaks out and starts affecting the rodents that are susceptible to it (*epizootic* phase). Typically, epizootics last for a period of one to two years, sometimes longer. Similar to epidemics affecting human populations, epizootics display a bell-shaped curve of activity with a slow onset, gradual increase, and decrease after peaking. Like epidemics, they come and go in waves, with interepizootic phases that depend on a complex web of factors, such as climate and the fluctuations in the host and vector populations.

The Hosts

Many species of mammals, such as cats, rabbits, goats, deer, and camels, can become infected with plague and thus serve as incidental hosts to the disease.[3] However, rodents are known to be particularly important in maintaining the disease in naturally occurring enzootic cycles, between hosts (some burrow-dwelling wild rodent species, such as marmots, voles, prairie dogs, ground squirrels, and gerbils) and their fleas. Today, several of these rodent species maintain the infection in the plague foci of the tropical and subtropical belt.[4]

Unless the disease becomes epizootic among susceptible rodents, it is normally not a direct threat to human populations. This being the case, the

[3] Handling *Y. pestis*–infected dead or live animals and eating their meat can transmit the disease to humans. Didier Raoult et al., "Plague: History and Contemporary Analysis," *Journal of Infection* 66, no. 1 (2013): 18–26; Abdulaziz A. Bin Saeed et al., "Plague from Eating Raw Camel Liver," *Emerging Infectious Diseases* 11, no. 9 (2005): 1456–57; A. B. Christie et al., "Plague in Camels and Goats: Their Role in Human Epidemics," *Journal of Infectious Diseases* 141, no. 6 (1980): 724–26; V. N. Fedorov, "Plague in Camels and Its Prevention in the USSR," *Bulletin of the World Health Organization* 23, nos. 2–3 (1960): 275–81. For the involvement of mammals, such as pigs, dogs, and cats, in the transmission process during the Black Death in Europe, see Stephen R. Ell, "Some Evidence for Interhuman Transmission of Medieval Plague," *Reviews of Infectious Diseases* 1, no. 3 (1979): 563–66; Ell, "Immunity as a Factor in the Epidemiology of Medieval Plague," *Reviews of Infectious Diseases* 6, no. 6 (1984): 866–79.

[4] Kenneth L. Gage and Michael Y. Kosoy, "Natural History of Plague: Perspectives from More Than a Century of Research," *Annual Review of Entomology* 50, no. 1 (2005): 505–28. More than two hundred species of wild rodents that inhabit all continents except Australia can host plague. See Andrey P. Anisimov, Luther E. Lindler, and Gerald B. Pier, "Intraspecific Diversity of *Yersinia pestis*," *Clinical Microbiology Reviews* 17, no. 2 (2004): 434–64.

presence of plague among ground-burrowing rodents could easily go unnoticed in the historical sources. Even though the sources would record plague epidemics that affected human populations, the epizootic and enzootic forms of the disease remain largely invisible. Hence, it is more difficult to identify the species of wild rodents that hosted plague in the Ottoman fauna of the late medieval and early modern eras. Though scanty, there is evidence that some rodent species that once likely inhabited the area, such as jerboa, marmot, and jird, may have hosted the plague.[5]

In the context of plague's transmission to humans, commensal rodents are more important to consider. *Commensal* species (literally, species that "eat at the same table") consume the food supplies of human populations and hence live in close proximity to them. When they are affected by plague, the infection can be transferred to humans via their ectoparasites. Of all the commensal animals, the rat is the most widely distributed species across the world today.

With respect to historical pandemics of plague, two species of rats in particular come into focus. It is generally accepted that while black rats (*Rattus rattus*) were the main hosts to the infection in the First and the Second Pandemics, brown rats, or Norway rats (*Rattus norvegicus*), starred in the Third. Even though both species belong to the same genus (*Rattus*), there are some important differences between them. The black rat has a tail longer than its body, which makes it a good climber, enabling it to live in the roofs of houses, close to humans. The brown rat, in contrast, has a tail shorter than its body and is not as good a climber as the former. It prefers to live away from humans, in basements and sewers.[6] Overall, the brown rat is larger, stronger, and more ferocious than the black rat, which perhaps has helped it become the dominant rat species everywhere it has colonized since its global dispersion in the early eighteenth century.[7]

As far as the late medieval and early modern eras are concerned, the black rat was the principal species of rodent serving as a host to plague. The origins of the genus *Rattus* are traced to south Asia, with the first members

[5] See my "New Science and Old Sources."

[6] In addition to these differences, it has also been noted that whereas black rats are commonly found on board ships, brown rats leave ships when they sail. See Bruce Skinner, "Plague and the Geographical Distribution of Rats," *British Medical Journal* 1, no. 2314 (1905): 994–95. This author noted in 1905 that Norway (brown) rats might possibly be immune to plague, which he took as one of the reasons how it managed to replace black rats. It is interesting that this view will be taken by some historians several decades later to explain the disappearance of plague from Europe.

[7] *Rattus norvegicus* was possibly originally a native of Palearctic Asia that came to be distributed worldwide, though it is more common in cooler countries of Asia. See John Reeves Ellerman and Terence Charles Stuart Morrison-Scott, *Checklist of Palaearctic and Indian Mammals, 1758–1946* (London: Printed by order of the Trustees of the British Museum, 1951), 588; Frédérique Audoin-Rouzeau, *Les chemins de la peste: le rat, la puce et l'homme* (Rennes: Presses universitaires de Rennes, 2003), 8n1.

of the species having probably evolved around three million years ago.[8] Owing to their highly adaptive nature, black rats followed human movements and migrations to spread around the world. It has been generally accepted that black rats migrated from the Indian subcontinent to Europe, whence they dispersed to different parts of the world by means of shipping.[9] A recent global study that surveyed the mitochondrial DNA (mtDNA) of black rats has identified well-differentiated lineages of the species and confirmed its migrations and patterns of dispersal. This phylogenetic research contributes significantly to our understanding of the species from an evolutionary perspective concerning the geographic patterns of diversification of the black rat and the direction and timing of its (prehistoric, historic, and contemporary) dispersals. Furthermore, it offers new ways of understanding associations of different lineages of black rats to diseases. According to this research, *Lineage I* black rats display the broadest distribution outside of Asia, occurring in a wide range of regions, including Europe, the Americas, Africa, Australia, and the Pacific Islands. This particular lineage moved from southern India to the Middle East and from there spread independently to Madagascar and Europe, and from those points globally as part of the Columbian Exchange, mainly on board ships, thus becoming referred to as "ship rats."[10] Although there seems to be consensus about this particular trajectory of spread, that is, from India to the Middle East–Mediterranean area, the timing of this migration seems unsettled. The scattered zooarcheological evidence for the presence of black rats in Palestine and Egypt seems to date to as early as the eighth to fourth millennium BCE, though this dating has been challenged; a safer assumption is the third millennium BCE.[11] However there is no doubting its widespread occurrence in the Mediterranean basin and islands over the last two thousand years, as zooarcheological evidence demonstrates.[12]

[8] Aplin et al., "Evolutionary Biology of the Genus *Rattus*: Profile of an Archetypal Rodent Pest," in *Rats, Mice, and People: Rodent Biology and Management*, ed. Grant R. Singleton, Lyn A. Hinds, Charles J. Krebs, and Dave M. Spratt, 487–98 (Canberra: Australian Centre for International Agricultural Research, 2003).

[9] Philip L. Armitage, "Unwelcome Companions: Ancient Rats Reviewed," *Antiquity* 68 (June 1994): 231–40. Also, for possible scenarios of the migration patterns of commensal black rats, see Anton Ervynck, "Sedentism or Urbanism? On the Origin of the Commensal Black Rat (*Rattus rattus*)," in *Bones and the Man: Studies in Honour of Don Brothwell*, ed. Keith Dobney and Terry Patrick O'Connor, 95–109 (Oxford: Oxbow, 2002).

[10] Aplin et al., "Multiple Geographic Origins of Commensalism and Complex Dispersal History of Black Rats," *PLoS One* 6, no. 11 (2011): e26357.

[11] Ervynck, "Sedentism or Urbanism?," esp. 100–104.

[12] Lise Ruffino and Eric Vidal, "Early Colonization of Mediterranean Islands by *Rattus rattus*: A Review of Zooarcheological Data," *Biological Invasions* 12 (2010): 2389–94; Robert Sallares, "Ecology, Evolution, and Epidemiology of Plague," in *Plague and the End of Antiquity: The Pandemic of 541–750*, ed. Lester Little (Cambridge: Cambridge University Press, 2007), 268. Also see Hans Zinsser's classic book for a brief account of rats in the Near East during antiquity: *Rats, Lice, and History* (New Brunswick, N.J.: Transaction,

When and to what extent black rats colonized Anatolia and the Balkans, as well as other parts of the Near East, are not well known. After the introduction of the species to the Mediterranean basin, there is no clear evidence about its presence in what later became the core areas of the Ottoman Empire. On the basis of the general tendency of the species to be dispersed through the movements of ships, it seems plausible to assume that it spread into this area following maritime links. Thus we may hypothesize that the black rat first gained a foothold on the coast, then spread farther inland, into the interior of Anatolia and the Balkans. Zooarcheological evidence from Continental Europe cautions us that during the first millennium, black rats mainly inhabited coastal and riverside towns and villages and almost always lead a commensal existence.[13] However, things started to change for black rats in Europe from the eleventh century onward. Zooarcheological evidence strongly suggests a substantially increased rat population in Europe between the eleventh and thirteenth centuries, spread across major trade routes.[14] Evidence from northwestern Russia also seems to support this pattern of spread.[15]

Unfortunately, we do not have comparable data for the core Ottoman areas in Anatolia and the Balkans for the late medieval and early modern eras. Indeed, very little is known about the historical presence of black rats in this region. According to zooarcheologists, this lack of information largely results from the fact that the bones are not collected from historic layers in archeological excavations. Even when they are collected, this is done by hand, whereas small bones, such as those of *R. rattus*, can only be retrieved through sieving. This makes it difficult to have a healthy set of data even for advancing most basic assumptions about the black rat's spread.[16] In the absence of zooarcheological findings, our understanding of past geographical distribution and populations of black rats in areas where the Ottomans came to rule has to draw from their distribution records in more recent times. The few twentieth-century distribution records available identify the black rat as a wide-ranging species but suggest that its presence across Anatolia,

2008), 193–94. For the late antiquity, the presence of black rats has been demonstrated on the basis of textual evidence in Lawrence Conrad, "The Plague in the Early Medieval Near East" (PhD diss., Princeton University, 1981), 402–12.

13 Ruffino and Vidal, "Early Colonization of Mediterranean Islands by *Rattus rattus*," 2392.

14 Audoin-Rouzeau, "Le rat noir (*Rattus rattus*) et la peste dans l'occident antique et médieval," *Bulletin de la Société de Pathologie Exotique* 92 (1999): 424–25.

15 A. B. Savinetsky and O. A. Krylovich, "On the History of the Spread of the Black Rat (*Rattus rattus* L., 1758) in Northwestern Russia," *Biology Bulletin* 38, no. 2 (2011): 203–7. The authors believe that black rats came to northwestern Russia from the west via trade routes.

16 On the basis of personal communication with zooarcheologist Canan Çakırlar of the University of Groningen, the Netherlands, March 28, 2013.

Syria, and the eastern Mediterranean varied greatly.[17] Today, the largest population of black rats can be found in Thrace, the Black Sea region, and western Anatolia, as well as in Anatolia littoral, the eastern Mediterranean, the Nile Valley, and the Balkan Peninsula. Current distribution of black rats varies greatly depending on altitude and climate, showing considerable variation in different physiogeographic settings.[18] Even though this may not necessarily reflect the black rat's past geographic spread, it gives at least some insight into its uneven distribution and diversity.

Similar to human populations, rat populations also change over time. Both commensal and wild rats are subjected to environmental and climatic conditions that have an impact on their survival. Any major changes in their natural habitat or their built environment can increase or decrease their population. For example, earthquakes and floods affect wild rodents by damaging their underground burrows and forcing them to move elsewhere, and commensal rats by changing their built environment. Similarly, epizootics are important for causing changes in rat populations.[19]

It is important to address the question whether commensal rodents, especially *R. rattus* colonies, are capable of sustaining plague over a prolonged period of time. There is greater emphasis in the ecological scholarship on the ground-burrowing wild rodents' role in sustaining the infection, but commensal rodents' ability to function in the same manner has not been sufficiently explored. However, important studies suggest that plague can be maintained over long periods of time in small commensal rat subpopulations without any contact with wild rodents. For example, plague has been

[17] Bathscheba Aharoni, *Die Muriden von Palästina und Syrien* (Lucka (Bez. Leipzig): Druck von Reinhold Berger, 1932), 177–82; Gabriele Neuhäuser, *Die Muriden von Kleinasien* (Lucka (Bez. Leipzig): Druck von Reinhold Berger, 1936), 173–74, 209. In the early 1930s, Neuhäuser caught no rats in Konya (in central Anatolia) and did not catch any black rat within the city of Zonguldak (on the Black Sea coast). He observed that the black rat has not filled in its available habitat and thus suggested that its presence in most Anatolian towns does not go back very long. Similarly, Misonne reports an absolute absence of black rats in southeastern Turkey and northern Syria in 1955. See Xavier Misonne, "Mammifères de la Turquie sud-orientale et du nord de la Syrie," *Mammalia* 21, no. 1 (1957): 53–68; Robert T. Hatt, *The Mammals of Iraq* (Ann Arbor: University of Michigan Press, 1959), 85.

[18] David T. Dennis, Kenneth L. Gage, Norman Gratz, Jack D. Poland, and Evgueni Tikhomirov, "Plague Manual: Epidemiology, Distribution, Surveillance and Control," report WHO/CDS/CSR/EDC/99.2 (Geneva, Switzerland: WHO, 1999); Nuri Yiğit et al., "A Study on the Geographic Distribution along with Habitat Aspects of Rodent Species in Turkey," *Bonner Zoologische Beiträge* 50, no. 4 (2003): 355–68; Nuri Yiğit et al., "Contribution to the Geographic Distribution of Rodent Species and Ecological Analyses of Their Habitats in Asiatic Turkey," *Turkish Journal of Biology* 22, no. 4 (1998): 435–46. Unfortunately, these studies focus on rodents in rural areas and offer limited insight into the historical distributions and populations of commensal black rats in urban areas.

[19] For a treatment of how epizootics correspond spatially, chronologically, and quantitatively to epidemics, see Audoin-Rouzeau, *Les chemins de la peste*, 42–45.

calculated to persist for one hundred years in a commensal rat population of sixty thousand without importations of new infection.[20] Hence, even if a certain rat population were killed, the infection could still be kept alive over a long time. This research has tremendous implications for explaining the historical persistence of plague in urban centers. It suggests that the disease could persist in an urban area, even if quarantine measures were in place, as long as there was a sufficient commensal rat population. Hence those towns would have served as self-perpetuating engines of epidemic activity or plague foci.

Rats are generally described as opportunistic creatures, and their populations grow as long as there is food to support them. In an urban setting, for example, the opportunities to obtain food from garbage and other sources support the growth of rat colonies. Regardless, it is difficult to estimate the density of rat populations in cities, not only in the past but also today. Studies show that the population density of rats in different patches exhibits great variation even within a given city.[21] The primary reason for this is that rats do not move much, unless they are forced to migrate. Normally, when black rats leave their location in search of food, they only move within a very limited radius. This explains why rat colonies in modern cities live in patches, usually around a row of houses or a neighborhood block, separated by man-made obstacles, such as wide roads, that inhibit their movement. Some areas in a premodern city, such as garbage disposal areas, places where waste accumulated, grain and cotton warehouses, and slaughterhouses, were most likely favored by rats. Moreover, the close proximity of houses, narrow and unpaved streets, and easy access to garbage and other food sources in a premodern city could help rat colonies prosper. Early modern Ottoman cities were no exception to this.[22]

We do not lack evidence for the presence of rats in early modern Ottoman cities. The seventeenth-century traveler Evliya Çelebi presents ample evidence that rats were common throughout Ottoman towns and cities. His account seems to suggest a distinction between commensal rats (in this case,

[20] Gage and Kosoy, "Natural History of Plague"; M. J. Keeling and C. A. Gilligan, "Bubonic Plague: A Metapopulation Model of a Zoonosis," *Proceedings of the Royal Society of London, Series B* 267, no. 1458 (2000): 2219–30; Keeling and Gilligan, "Metapopulation Dynamics of Bubonic Plague," *Nature* 407, no. 6806 (2000): 903–6.

[21] Doris Traweger et al., "Habitat Preferences and Distribution of the Brown Rat (*Rattus norvegicus* Berk.) in the City of Salzburg (Austria): Implications for an Urban Rat Management," *Journal of Pest Science* 79, no. 3 (2006): 113–25. Even though this study is on brown rats, it gives a fair idea about patterns of urban distribution.

[22] On the basis of the available evidence, some continuity can be presumed between Byzantine Constantinople and Ottoman Istanbul as regards the density and distribution of rat populations. Arguably, it worsened in the Ottoman era, as a result of fewer restrictive blocks than the Roman model of urban planning used and because of increased density of population and housing. See Michael McCormick, "Rats, Communications, and Plague: Toward an Ecological History," *The Journal of Interdisciplinary History* 34, no. 1 (2003): 1–25.

presumably the black rat) and wild rodents.[23] During his long travels, Evliya Çelebi did not fail to note places that had an excessive number of rats. For example, he mentions the Kurdish village Bekufar (he translates as the "rats-village" in the Kurdish language) as having many rats.[24] In a similar vein, he wrote that the castle of Pontikos/Pondikoz (Pontiko-Kastro in Greece) was full of rats that were sometimes "as big as cats."[25] For other towns, he comments on the surprising rarity or even the total absence of rats. For example, he writes that the town of Muş in eastern Anatolia had no rats because it was protected by a spell.[26] Similarly, he commented on the rarity of rats in the town of Sarajevo (Bosnasaray), also protected by a spell.[27] About the Hungarian castle of Košice (Kaşa), he remarks on the absolute absence of rats and any other pests, which he explains by spells made by one of the apostles of Jesus being still extant.[28] The absence of rats and other pests in a city had to be seen as an anomaly, and perhaps it took a supernatural power to keep pests away from towns. Judging from this account, it may be assumed that rats were accepted as a common pest in the urban texture of early modern cities. That this may be so in the case of Istanbul can be evidenced in his praise of weavers (*esnâf-ı örücüyân*) skilled in repairing textiles gnawed by rats.[29]

[23] Even though the term rat (*sıçan*) was used as a general taxonomic caterogy comprising many rodent species in Ottoman Turkish, there seems to be a distinction between commensal and wild species depending on their denomination with their natural habitat. For an example of how Evliya Çelebi clarified his terminology, see Evliya Çelebi, *Seyahatnâme*, 5:211: "fâre, yaʿnî kesegen, Türkçe sıçan dedikleri muzırr." In other instances, he uses the term *muş*. For wild rodents, he uses terms such as *yer sıçanı*, *Erdebil sıçanı*, and *pündika*. Even though it is difficult to know what species these terms exactly referred to, there is some evidence about their use in the nineteenth century. The Redhouse dictionary lists the following terms and the corresponding species: *orman sıçanı* (the short-tailed field mouse, *Arvicola arvalis*); *çöl sıçanı* (the jerboa, *Dipus aegyptius*); *su sıçanı* (the water vole, *Arvicola amphibius*); *dağ sıçanı* (the marmot, *Arclomys marmotta*); *yaban sıçanı* (the lemming, *Myodes lemmus*); *yer sıçanı* (the bank vole). Alexander Russell also names different species of rodents occurring in eighteenth-century Aleppo, along with their names in Arabic and Latin. See Alexander Russell and Patrick Russell, *The Natural History of Aleppo: Containing a Description of the City, and the Principal Natural Productions in Its Neighbourhood: Together with an Account of the Climate, Inhabitants, and Diseases, Particularly of the Plague* (London: Printed for G. G. and J. Robinson, 1794), 2:180–82. Needless to say, further research is needed to clarify the taxonomy of rodents in the Ottoman landscape.

[24] Evliya Çelebi, *Seyahatnâme*, 4:289.

[25] Ibid., 8:129.

[26] Ibid., 3:132.

[27] Ibid., 5:211.

[28] Ibid., 6:21. The example of the fortress of Košice (Kaşa) is also interesting because Evliya Çelebi mentions the absence of rats as well as the lack of plague in that town, without necessarily establishing a connection between the absence of the two.

[29] Ibid., 1:303: "bir Kişmîrî şâl ve dülbend ve atlas ve hârâ ve ihrâm makûlesi eşyâları sıçan delse veyâhûd bir gûne âfet etse bunlar ol rahnedâr olan yerleri örüp gaʾib ederler, aslâ maʿlûm olmaz, musannaʿ kârdır."

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With the abundance of rats, people tried to control their numbers by killing them, possibly with traps or rat poison. There do not seem to be professional rat catchers in the early modern Ottoman Empire organized in a guild, either because this was a service performed by another group of professionals or more likely because this was something done by individuals.[30] Incidentally, we find anecdotal evidence in one of Evliya Çelebi's stories of the use of rat poison. Even though we do not know exactly what substance was used, it might have been a mixture of arsenic and oxymel (probably to give substance to powder arsenic and attract rats to the poison by its sweet smell).[31] Other sources support this claim and indeed point to the fairly common use of rat poison in Ottoman lands. For example, according to the sixteenth-century probate registers, rat poison was sold in the apothecary shops in Edirne.[32] Sources mention different substances used as rat poison. For example, the seventeenth-century mystic Niyazi-i Mısri believed that he had been poisoned by rat poison (*sıçan otu*, "arsenic") but also noted that there was another kind of poison, known as *sülümen*, which was more effective and could easily be found in apothecary shops.[33] It seems that both *sıçan otu* and *ak sülümen* – the colloquial form of *süleymani* (a white powder of mercuric chloride) – were sold at these shops.[34] Evidence for the use of these substances by physicians and apothecaries points to the circulation of the substance in the Ottoman markets.[35] In eighteenth-century Aleppo, using arsenic to poison rats was common enough to cause accidents, and care was

[30] No guild of rat catchers is listed in the processions of guilds in the sixteenth and seventeenth centuries. See Eunjeong Yi, *Guild Dynamics in Seventeenth-century Istanbul: Fluidity and Leverage* (Leiden: Brill, 2004), appendices. Professional rat catchers were known in Europe. For an illustration of a "fully equipped" rat catcher, see Werner Schreiber, *Infectio: Infectious Diseases in the History of Medicine* (Basel: Roche, 1987), 28.

[31] Evliya Çelebi, *Seyahatnâme*, 6:130. Cf. Kari Konkola, "More Than a Coincidence? The Arrival of Arsenic and the Disappearance of Plague in Early Modern Europe," *JHMAS* 47, no. 2 (1992): 186–209.

[32] Ömer Lütfi Barkan, "Edirne Askerî Kassamı'na Âit *Tereke Defterleri* (1545–1659)," *Türk Tarih Belgeleri Dergisi* 3, nos. 5–6 (1966): 104.

[33] [Niyazî-i Mısrî, *Mecmua-i Kelimat-ı Kudsiyye-i Hazret-i Mısrî*, Bursa Eski Eserler Kütüphanesi, MS Orhan 690, 9a], cited in İsmail Hakkı Altuntaş, *Niyazi-i Mısri Divan-ı İlahiyyat ve Açıklaması* (n.p., 2010), 1:86.

[34] Turhan Baytop, "Aktarlar," in *Dünden Bugüne İstanbul Ansiklopedisi* (Istanbul: Tarih Vakfı, 1993–95), 1:172.

[35] Coşkun Yılmaz and Necdet Yılmaz, eds., *Osmanlılarda Sağlık* [Health in the Ottomans] (Istanbul: Biofarma, 2006), e.g., see the probate record of a fifteenth-century physician (2:21, doc. 2); the inventory of a late-eighteenth-century shop (presumably an apothecary shop) differentiated between *taş zırnıh* (most probably, realgar) and *zırnıh-ı meshuk* (most probably, orpiment) (2:344–46, doc. 773). Even though these documents do not tell us about the uses of the substance, some medicinal uses should account partially for their availability and circulation. Also see note 75 for its use for depilatory purposes.

taken not to use it in households where there were children. Instead, most people simply relied on cats to catch rats.[36]

It should not come as a surprise that most historical accounts on rats occur in contexts other than plague. We, as moderns, expect to see evidence of rat mortality in an account of plague, but the link between rats and plague was not scientifically demonstrated until the end of the nineteenth century. As we shall see later, it was not until the Third Pandemic that the Swiss-born French bacteriologist Alexandre Yersin of the Pasteur Institute successfully isolated the pathogen causing the plague and observed that rats were primary hosts for it. Following this, the French biologist Paul-Louis Simond, also of the Pasteur Institute, demonstrated that fleas were instrumental in communicating the disease from rodent hosts to humans. In the absence of a known link between plague and rats, it was not uncommon for premodern observers to see rats, and even dead rats, during a plague epidemic and not take note of it.[37] Nonetheless, we are fortunate to find such references on a few occasions. For example, one of these references comes from Byzantine Constantinople during the Black Death and may be taken as evidence for the presence of black rats in the city before the Ottoman conquest. As an eyewitness to the plague, the Byzantine historian Nicephorus Gregoras left a brief description of the epidemic in which he noted the dead rats. He wrote: "The calamity did not destroy men only, but many animals living with and domesticated by men. I speak of dogs and horses and all the species of birds, even the rats that happened to live within the walls of the houses."[38] This seems to be a clear reference to an epizootic among the black rats of the city. Even though it may be a literary motif borrowed from the ancient Greek tradition, we may nevertheless assume that it was an acute observation.[39] Another piece of evidence for rat mortality during a plague epidemic in Istanbul comes from a late-sixteenth-century traveler's account. Michael Heberer von Bretten, a southern German traveler who witnessed a plague outbreak in Istanbul in the early months of 1588, noted dead rats, horses, and dogs left lying in the alleys. He described the streets of the city

36 Russell and Russell, *Natural History of Aleppo*, 2:180–81. Also see Maurits van den Boogert, *Aleppo Observed: Ottoman Syria through the Eyes of Two Scottish Doctors, Alexander and Patrick Russell* (Oxford: Oxford University Press, 2010), 164.

37 The scarcity of references to rats in historical sources was taken by some historians as evidence for the rejection of bubonic plague as the cause of the Black Death. For an insightful criticism of these "heretical" views, see Sallares, "Ecology, Evolution, and Epidemiology," 269–70.

38 Christos S. Bartsocas, "Two Fourteenth Century Greek Descriptions of the 'Black Death,'" *JHMAS* 21, no. 4 (1966): 395; also reprinted in John Aberth, ed., *The Black Death: The Great Mortality of 1348–1350: A Brief History with Documents* (Boston: Bedford/St. Martin's, 2005), 15–16.

39 According to Bartsocas, rats were considered to be related to epidemics in Greek mythology. See ibid., 399.

as filthy, which he thought was the cause of the pestilence.[40] This piece of evidence not only indicates rat mortality but also suggests an overpopulation of rats, which may cause rats to leave their safe location indoors and die on the streets.

For the most part, however, the references in the historical sources are too few and too scanty to draw healthy conclusions about epizootics and to establish their links to changes in rat populations. It is generally accepted that there is a threshold, a *critical density*, of the rat population to sustain an epizootic, and that when the number of rats exceeds that threshold, epizootics occur; conversely, when populations fall below the threshold, then epizootics recede.[41] Changes in rat populations also depend on the season, which relates the seasonality of human plague to that of the breeding patterns of rats. For example, in Istanbul, the disease manifested itself in its bubonic form typically from mid-spring to late summer or early fall, owing to the particular climatic conditions that favored the reproduction of rats and their fleas. Yet these conditions suggest only indirectly that rat populations were sometimes above the critical threshold level in Ottoman cities to sustain plague epidemics. Given the limitations of our current state of knowledge on this issue, it is nearly impossible to establish a direct relationship between plague and black rats in Ottoman cities and rural areas. Neither can such a relationship be supported by the present state of the zooarcheological data at hand. Zooarcheologists caution us that because rats live by burrowing, taphonomic analysis and dating would be necessary to check its association with the historic layer under question. Therefore, to talk about the rat-plague relationship with confidence, large, multilayer samples are necessary to study population boom and depletion around the investigated time period, rather than absence-presence surveys for the period of interest.[42] It seems that until more research is done in this area, it will be difficult to relate plague to black rats in the Ottoman landscape with any confidence.

The Vectors

Even though more than eighty species of fleas are known to have the ability to transmit plague at varying degrees of efficiency, the rat flea (especially the Oriental rat flea, *Xenopsylla cheopis*) has received the greatest attention

[40] Johann Michael Heberer, *Aegyptiaca servitus* (Graz: Akademische Druck und Verlagsanstalt, 1967), 303; Metin And, *16. Yüzyılda İstanbul: Kent, Saray, Günlük Yaşam* (Istanbul: Yapı Kredi Yayınları, 2009), 90.

[41] Konkola, "More Than a Coincidence?," 204–5.

[42] Based on personal communication with zooarcheologist Canan Çakırlar of the University of Groningen, the Netherlands (March 28, 2013). I would like to thank Canan Çakırlar and Scott Redford for their invaluable assistance with this issue.

in both the scientific and historical scholarship since the Third Pandemic.[43] During his investigations of the plague in India, Paul-Louis Simond noticed that fleas were the essential vectors for transmitting the disease between rats as well as from rats to humans.[44] Even though his observations and the experiments he conducted to demonstrate the role of fleas as plague vectors were initially received with skepticism in the academic community, later research confirmed his findings.[45] In the early twentieth century, the process by which *X. cheopis* transmitted the plague was clearly identified: when *X. cheopis* fed on the blood of an infected host, plague bacteria multiplied in its gut, causing a blockage in its digestive system, urging it frantically to feed again and again. When the blocked flea bit a new host to feed, it regurgitated the multiplied bacteria, which entered the blood stream of the new host. It was this process of blockage, along with other factors, that made *X. cheopis* recognizable as the champion of plague vectors.[46] Unlike most other flea species, *X. cheopis* is not host-specific, that is, it can live and feed on different species, which makes it an important link between wild and commensal rodents and between rodents and humans, as well as a critical medium of transmission locally and over long distances. Moreover, *X. cheopis* can live in off-host environments, such as in fur, woolen cloth, grain debris, dead hosts, and the soil, even while infected with *Y. pestis*.[47]

[43] Anisimov, Lindler, and Pier, "Intraspecific Diversity of *Yersinia pestis*"; Rebecca J. Eisen, Lars Eisen, and Kenneth L. Gage, "Studies of Vector Competency and Efficiency of North American Fleas for *Yersinia pestis*: State of the Field and Future Research Needs," *Journal of Medical Entomology* 46, no. 4 (2009): 737–44; Carmichael, "Plague Persistence in Western Europe."

[44] Simond's observations and experiments on the mechanisms of plague transmission were published in 1898. See Paul-Louis Simond, "La propagation de la peste," *Annales de l'Institut Pasteur* 12, no. 10 (1898): 625–87. The role of fleas was also simultaneously observed by the Japanese investigator Masanori Ogata of the Hygiene Institute in Tokyo. For a detailed discussion of these discoveries, see Ann G. Carmichael, "Plague, Historical," in *Encyclopedia of Microbiology*, vol. 4, ed. Moselio Schaechter, 58–72 (Oxford: Elsevier, 2009).

[45] See M. Simond, M. L. Godley, and P. D. Mouriquand, "Paul-Louis Simond and His Discovery of Plague Transmission by Rat Fleas: A Centenary," *Journal of the Royal Society of Medicine* 91, no. 2 (1998): 101–4. Later on, Paul-Louis Simond was to serve as the director of the Ottoman Bacteriology Institute in Istanbul between 1911 and 1913. See Şeref Etker, "Paul-Louis Simond ve Bakteriyolojihane-i Osmani'nin Çemberlitaş'ta açılışı (21 Eylül 1911)," *Osmanlı Bilimi Araştırmaları* 10, no. 2 (2009): 13–33.

[46] A. W. Bacot and C. J. Martin, "Observations on the Mechanism of the Transmission of Plague by Fleas," *Journal of Hygiene* 13 (Suppl. III) (1914): 423–39.

[47] Distinguishing between *fur fleas* and *nest fleas*, historian Ole Jørgen Benedictow puts forward that *X. cheopis* is a typical fur flea because of its ability to move along with its hosts. He suggests that the human flea (*P. irritans*) is a typical nest flea, which does not move along with its human hosts, nor does it remain in the clothing; instead, it prefers to nest in or near bedding. See Benedictow, *Black Death*, 19–21.

Hence, infected fleas can carry the disease from one place to another and from one season to the next.[48]

Particular environmental and climatic conditions seem to favor the survival and reproduction of fleas, as their number fluctuates by the season (lower flea density in lower temperatures and cooler season to higher density in mild temperatures or warmer season). Temperatures between 24 and 27 degrees Celsius are ideal for most epidemic activity.[49] Temperature is also important for sustaining a successful blockage in X. *cheopis* to guarantee the transmission of the infection. In their classic 1914 study, Bacot and Martin observed that infected "fleas lived as long as 50 days at from 10°C to 15°C and 23 days at 27°C, and died infected."[50] Research in tropical medicine in the 1960s and 1970s further confirmed the critical importance of temperature. For example, it was demonstrated that the transmission rate fell in infected fleas in temperatures higher than 27.5 degrees Celsius. Another experiment showed that when temperature increased from 23.5 to 29.5 degrees Celsius, clearing of the infection increased more than ten times, and blockage rates fell more than 50 percent.[51] In addition to temperature, humidity is also an important factor. Humidity greater than 40 percent is critical for the survival of fleas and flea larvae.[52] All of this has great significance for our understanding of the *seasonal signature* of plague in a given locality.

Nonetheless, the leap of the infection to humans takes place as the result of bites by fleas. Classical plague studies demonstrated that when an infected rat died, this was detected by its fleas because of the drop in the body temperature of their host, which forced them to seek other hosts. If humans are present in close proximity, then fleas bite and infect them. Most human infections take place when large numbers of rats die from an epizootic, increasing the possibility of encounters with rat carcasses and their fleas in search of new hosts. The fact that X. *cheopis* is a highly efficient vector of transmission means that its capacity to infect new hosts is very high. This also means that the number of fleas per host does not need to be high to maintain epidemic conditions. On the contrary, it has been shown that the ratio of X. *cheopis* per host can be quite low to maintain transmission cycles.[53]

Most of this body of knowledge regarding X. *cheopis* and the mechanisms by which it transmits infection to humans is drawn from the twentieth-century ecological context of South Asia. The particular model of flea-borne

48 Gage and Kosoy, "Natural History of Plague," 517–18.

49 Rebecca J. Eisen and Kenneth L. Gage, "Transmission of Flea-Borne Zoonotic Agents," *Annual Review of Entomology* 57, no. 1 (2012): 64.

50 Bacot and Martin, "Transmission of Plague by Fleas," 437.

51 Gage and Kosoy, "Natural History of Plague," 516.

52 Audoin-Rouzeau, *Les chemins de la peste*, 47.

53 Eisen and Gage, "Transmission of Flea-Borne Zoonotic Agents," 62, 69.

plague transmission as a result of blockage has long been accepted as the dominant paradigm. However, recent research has shown that transmission could take place as a result of other processes, such as mechanical or early-phase transmission. In the meantime, other flea species, such as cat fleas (*Ctenocephalides felis*), Asiatic/northern rat fleas (*Nosopsyllus fasciatus*), and human fleas (*Pulex irritans*) are recognized as vectors.[54] Likewise, the question about the ability of human ectoparasites to transmit the infection between humans has long intrigued the scientific community. On the basis of research in Morocco in the 1940s, Blanc and Baltazard posited the human (head and body) louse as a potential plague vector. Similar observations with respect to the role of human ectoparasites were made in the 1950s based on cases of plague in Iran, Syria, and Turkey, in the absence of domestic rats.[55] Even though the findings of this research remained controversial for many decades, there seems to be a renewed interest in the role of human ectoparasites in plague transmission.[56] Most recently, promising steps have been taken to investigate the role of the human louse (*Pediculus humanus*) in the transmission of plague, both in field observations and in laboratory experiments. According to this, human lice can become infected with *Y. pestis* after one blood meal from an infected host, and as few as ten lice may be sufficient to infect a new host.[57] All this reminds us that different species of fleas and lice could have worked as plague vectors in different ecological settings.

[54] Gage and Kosoy, "Natural History of Plague"; Eisen and Gage, "Transmission of Flea-Borne Zoonotic Agents." For a detailed discussion of the vector capacity of different flea species, see Audoin-Rouzeau, *Les chemins de la peste*, 59–83.

[55] G. Blanc and M. Baltazard, "Rôle des ectoparasites humains dans la transmission de la peste," *Bulletin de l'Académie nationale de médecine* 126 (1942): 446; M. Baltazard, "New Data in the Interhuman Transmission of Plague," *Bulletin de l'Académie nationale de médecine* 143 (1959): 517–22.

[56] Benedictow, for example, is very critical of this research. See Benedictow, *Black Death*, 17–19. Alternatively, also see Michel Drancourt, Linda Houhamdi, and Didier Raoult, "*Yersinia pestis* as a Telluric, Human Ectoparasite-borne Organism," *The Lancet Infectious Diseases* 6, no. 4 (2006): 234–41. On the reception of Blanc and Baltazard's position, see Audoin-Rouzeau, *Les chemins de la peste*, 97–101.

[57] Renaud Piarroux et al., "Plague Epidemics and Lice, Democratic Republic of the Congo," *Emerging Infectious Diseases* 19, no. 3 (2013): 505–6; S. Badiaga and P. Brouqui, "Human Louse–Transmitted Infectious Diseases," *Clinical Microbiology and Infection: The Official Publication of the European Society of Clinical Microbiology and Infectious Diseases* 18, no. 4 (2012): 332–37; Saravanan Ayyadurai et al., "Body Lice, *Yersinia pestis* Orientalis, and Black Death," *Emerging Infectious Diseases* 16, no. 5 (2010): 892–893 (experimentally demonstrated that only *Y. pestis* Orientalis could be transmitted via human body lice); Thi-Nguyen-Ny Tran et al., "Brief Communication: Co-detection of *Bartonella quintana* and *Yersinia pestis* in an 11th–15th Burial Site in Bondy, France," *American Journal of Physical Anthropology* 145, no. 3 (2011): 489–94; Linda Houhamdi et al., "Experimental Model to Evaluate the Human Body Louse as a Vector of Plague," *Journal of Infectious Diseases* 194, no. 11 (2006): 1589–96.

Fleas and lice were abundant in Ottoman society, as they were in other premodern societies, though it is difficult to determine exactly what species they were. Our sources do not make a clear distinction between the types of ectoparatises, even though some species were clearly known to all. As with rats, we encounter the problem of taxonomy, as both lice (head and body lice) and fleas were used as generic categories in premodern Ottoman society. For example, the term used in reference to lice (*bit*, "louse") was also used for other insects, such as bedbugs (*tahta biti*). In the absence of archeoentomological evidence, it is nearly impossible to establish what flea species lived in the Ottoman landscape. It would be tempting to assume the historical presence of *X. cheopis* in this area owing to the occurrence of their black rat hosts. However, even though it is possible to assume that this flea species arrived to the Mediterranean basin along with their rodent hosts (*R. rattus*), some scholars suggest that *X. cheopis* previously lived in the Nile Valley on other hosts and that it adapted to *R. rattus* only after the latter's arrival to the area.[58] This does not rule out the possibility that other flea and lice species were common in the Ottoman landscape, given that humans are only incidental hosts to rat fleas.

The abundance of fleas in premodern societies may be attributed to poor hygienic practices. In the absence of modern standards of hygiene, houses could be a breeding ground for rats and fleas. Especially wooden houses favored the survival of the latter, as small crevices in the wood provided the ideal niche for the survival of flea eggs; however, sources also report their abundance in stone structures. European travelers to the Ottoman Empire often complained about rats, fleas, and other pests. For example, Hans Dernschwam, who traveled with the Habsburg ambassadorial mission to the Ottoman Empire in the mid-sixteenth century, commented on the abundance of pests in the rooms of stone buildings and noted how, in the summer months, the locals ate, relaxed, and slept outdoors on the elevated patio of a caravanserai in Istanbul to avoid pests, such as insects, mice, lizards, and snakes.[59] Similarly, Salomon Schweigger, the preacher in the retinue of the Habsburg ambassador, commented on the abundance of fleas, lice, bedbugs, mice, rats, weasels, and other pests in their ambassadorial residence

[58] Eva Panagiotakopulu, "Pharaonic Egypt and the Origins of Plague," *Journal of Biogeography* 31, no. 2 (2004): 269–75. Paleoentomological research suggests that the historical distribution of ectoparasites does not necessarily coincide with the patterns of migration of their current hosts. For example, it is possible that human fleas (*P. irritans*) were spread via other species before they adjusted to humans. See Paul C. Buckland and Jon P. Sadler, "A Biogeography of the Human Flea, *Pulex irritans* L. (Siphonaptera: Pulicidae)," *Journal of Biogeography* 16, no. 2 (1989): 115–20. About the Egyptian origins of *X. cheopis*, also see Robert Traub, "The Fleas of Egypt: Two New Fleas of the Genus *Nosopsyllus* Jordan, 1933," *Proceedings of the Entomological Society of Washington* 65, no. 2 (1963): 96.

[59] Hans Dernschwam, *İstanbul ve Anadolu'ya Seyahat Günlüğü*, trans. Yaşar Önen (Ankara: Kültür ve Turizm Bakanlığı, 1987), 61–62.

in Istanbul in the late sixteenth century.[60] Dark houses, with no direct sunlight, favored the survival of fleas indoors; rugs and woolen bedding material made the perfect hiding place, and fur and woolen clothes provided a good shelter. Infrequent washing of clothes could preserve the fleas, even if one's body was clean.

Items of clothing were expensive and changed hands rather frequently. Sometimes family members inherited them; other times, they were sold after one's death. In Ottoman cities, secondhand clothing items were sold in the appropriately named flea markets (*bit pazarı*). Istanbul's notorious flea market and its brokers have been described by contemporary sources. For example, historian Mustafa Ali's *Camiʿ ü'l-buhur der mecalis-i sur* (Gatherer of the Seas in the Gatherings of the Festival) mentions these merchants (*bit pazarı halkı*) in the description of the procession of the guilds in Istanbul in 1582. He wrote elsewhere that the brokers of the flea market made at least a hundred gold pieces every year, and most more.[61] Evliya Çelebi also seems to be suspicious of their trade, as he labeled them treacherous (*ehl-i hilekar, bitbazarı*). He reports there being four hundred shops and seven hundred dealers, larger than many other groups in the textile trade.[62] The trade was a profitable one, which resisted the attempts of the Ottoman administration to regulate it in the late-sixteenth century by issuing a series of orders for that purpose. For example, a *mühimme* order of 1581 refers to an earlier prohibition of the trade within the walled area of Istanbul, which clearly was not honored.[63]

Several European observers of the Ottoman Empire believed that selling secondhand clothes in the flea markets was instrumental in spreading plague. For example, the French physician François Pouqueville (d. 1838) observed that the fur clothes of deceased plague victims, harboring *miasma*, were sold in flea markets, and the market was a plague focus.[64] A. Brayer, a French

[60] Salomon Schweigger, *Sultanlar Kentine Yolculuk, 1578–1581*, trans. S. Türkis Noyan (Istanbul: Kitap Yayınevi, 2004), 57. Before him, Stephan Gerlach had also written that there were scorpions, lizards, mice, and insects crawling around in the very same ambassadorial residence. Stephan Gerlach, *Türkiye Günlüğü* (Kitap Yayınevi, 2007), 1:77.

[61] Yi, *Guild Dynamics*, Appendix B, 255; Mustafa Ali, *Câmiʿu'l-Buhûr Der Mecâlis-i Sûr*, ed. Ali Öztekin (Ankara: TTK, 1996), 154–55; Mustafa Ali, *Mustafā Ali's Counsel for Sultans of 1581*, ed. Andreas Tietze (Vienna: Österreichischen Akademie der Wissenschaften, 1979), 57.

[62] Evliya Çelebi, *Seyahatnâme*, 1:316; for the flea market in Edirne, see 3:241; in Tokat, 5:35. It appears that the number of shops in the flea market was much smaller in the late fifteenth century. See Çiğdem Kafesçioğlu, *Constantinopolis/Istanbul: Cultural Encounter, Imperial Vision, and the Construction of the Ottoman Capital* (University Park: Penn State University Press, 2009), 40.

[63] MD 42/276/851, 27 Ca 989/June 29, 1581.

[64] François-Charles-Hugues-Laurent Pouqueville, *Voyage en Morée, à Constantinople, en Albanie, et dans plusieurs autres parties de l'Empire othoman, pendant les années 1798, 1799, 1800 et 1801* (Paris, 1805), 2:109–11.

physician who lived in Istanbul for nine years in the early nineteenth century, remarked that the secondhand clothing trade was a deadly business because the personal belongings of the deceased, including those who died of plague, accumulated in the shops of the flea market and produced "pestilential miasmas." Like Pouqueville, Brayer singled out the Jews as those who bought the items of clothing and bedding from the houses of the deceased, including plague victims. Claiming that the personal belongings of the 150,000 plague victims of the 1812 outbreak ended up in the flea market, he exclaimed, "What a focus of pestilential miasma!"[65] Likewise, Helmuth von Moltke (d. 1891), a German military officer who served as adviser for the Ottoman Empire in 1830s, commented on the role of used items of clothing and linens in spreading the plague. Moltke noted that some of these items were sold in the streets by itinerant Jewish merchants.[66] Panzac also discussed this as one of the social practices that contributed to plague's dissemination in late Ottoman society. He suggested that the circulation of cotton, wool, and fur clothing items was instrumental in spreading plague.[67]

Evliya Çelebi makes clear that having lice or fleas on one's body was accepted in Ottoman society as the norm. Not having any lice or fleas or having too many was considered inappropriate. In his travel account, he mentions peoples who had too many of them (including on their body, hair, beard, and even nose and ear hair) or those who did not have them at all. He notes that the absence of fleas and lice was commonly attributed to foul smells on one's body or to leprosy. He adds, "The louse is of delicate nature, it likes clean places, does not like the leper body."[68] This seems to be a common belief in Ottoman society, so much so that having lice could be taken as evidence that one was not a leper. The famous story of the grand vizier Rüstem Pasha's louse illustrates the case. According to the story, when Süleyman was about to marry his daughter Mihrimah off to Rüstem, he heard rumors that Rüstem suffered from leprosy. So he sent one of the court physicians to Diyarbakır, where Rüstem was a governor, to examine the latter's body and clothes. If the physician were to find a louse, then the rumors of leprosy would be proven wrong and Rüstem

65 A. Brayer, *Neuf années à Constantinople, observations sur la topographie de cette capitale, l'hygiène et les mœurs de ses habitants, l'islamisme et son influence: la peste . . . les quarantaines et les lazarets . . .* (Paris: Bellizard, 1836), 2:354–56; quote on 355.

66 Helmuth Karl Bernhard von Moltke, *Türkiye'deki Durum ve Olaylar Üzerine Mektuplar (1835–1839)* (Ankara: TTK, 1960), 89.

67 Panzac, *La peste*, 176. Even though this seems to have provided some local circulation of the disease, it may be worth considering what species of fleas would move with used clothing. According to Benedictow, human fleas did not move with clothing, but rat fleas did. See note 47.

68 "Kehle nâzük tabîʿatdır, pâk yeri sever ve cüzâm vücûdu sevmez. Ve cüzâm ve miskîn âdemde kehle olmaz. Olmamak ve çok olmak dahi fenâdır." Evliya Çelebi, *Seyahatnâme*, 4:48; 7:303–4; quote on 304.

would be allowed to marry the princess. Upon finding a louse, the doctor confirmed that the latter was not a leper, and Süleyman let him marry his daughter.[69] As someone who speedily rose through the ranks of the Ottoman administration, Rüstem Pasha was perceived by his contemporaries with suspicion and contempt. Hence, this story circulated to emphasize that even his louse brought him good fortune (*kehle-i ikbal*). Whether true or not, the story might be taken as an indication of how common ectoparasites were in early modern Ottoman society, even among the elite.[70] Further testimony for fleas being a commonly occurring nuisance in Aleppo comes from the eighteenth-century Scottish physician Alexander Russell. According to him, it was "impossible to walk about without collecting a colony," and even bathing was "no remedy against fleas."[71]

It was not only the Ottoman urban population who suffered from ectoparasites. Fleas were as much a part of Ottoman sedentary life as they were of the nomadic, so much so that the beginning of warmer seasons, marked by a proliferation of fleas, meant that it was time to move to highland pastures (*yayla*). For Anatolian nomads (*yürük*), the winter quarters becoming flea ridden or full of vermin (*pirelendi*) marked the time to move to cooler temperatures.[72] For example, Dernschwam noted, while traveling to Amasya in the early days of April 1555, that the seminomadic pastoralists of Anatolia were busy leaving their residences in low-lying villages to move to the mountains for the summer months. This move, he claimed, was mainly to avoid fleas and other pests that bred in their houses over the summer months.[73]

When fleas and lice were so abundant, people often had to delouse themselves or each other. This could be done simply by hand. A more radical solution against lice and flea infestation was to shave off hair and beard completely, a practice reserved for slaves in Ottoman society for the most part.[74] Depilating body hair, however, was common both among men and women because it was believed to prevent such infestations.[75] Also, fragrant

[69] Mustafa Ali, *Künhü'l-ahbâr: dördüncü rükn, Osmanlı tarihi* (Ankara: TTK, 2009), 358b. For rumors about Rüstem's indisposition, also see Bernardo Navagero, "Relazione dell'impero ottomano del clarissimo Bernardo Navagero stato Bailo a Costantinopoli fatta in pregadi nel mese di febbraio de 1553," in *Relazioni degli ambasciatori Veneti al Senato*, series III, ed. Eugenio Albèri (Florence, 1840), 1:99. I am grateful to Zahit Atcıl for his help on the stories of Rüstem Pasha's louse.

[70] For an account of how common ectoparasites were among the members of European nobility in the same era, see Audoin-Rouzeau, *Les chemins de la peste*, 242–43.

[71] Russell and Russell, *Natural History of Aleppo*, 2:225–26.

[72] Fernand Braudel, *The Mediterranean and the Mediterranean World in the Age of Philip II* (Berkeley: University of California Press, 1995), 1:97.

[73] Dernschwam, *Seyahat Günlüğü*, 265, 298, 300–302; for the same in the Balkans, 337, 342.

[74] Gerlach, *Türkiye Günlüğü*, 2:794; Schweigger, *Sultanlar Kentine Yolculuk*, 109.

[75] Brayer, *Neuf années à Constantinople*, 1:162. In the mid-sixteenth century, Dernschwam described the use of a powder in the bathhouses for depilatory purposes. According to his

oils (or grease) could be used for repelling fleas and lice. For example, Evliya Çelebi mentions the use of clarified butter (*say yağı*) by locals of the Nile Valley, who rubbed it on their bodies.[76] Some essential oils were much praised in plague treatises because they were believed to clear the miasma and keep plague away, without any mention of their flea-repellent properties. For example, the sixteenth-century Sephardic convert physician İlyas bin İbrahim recommended the use of almond oil and violet oil to preserve health in times of plague, as well as chamomile oil and rose oil in preparing ointments for the treatment of plague buboes.[77]

Similar to the case with rats, the connection between plague and fleas and lice was not noted by premodern observers. In the absence of knowledge of plague vectors, even when an association was observed, the authors did not think of a causal relationship; rather, they attributed the association to larger natural or supernatural factors. This is perhaps most remarkably illustrated in the case of William Quacquelben, the physician in the Habsburg ambassadorial mission to the Ottoman Empire in the mid-sixteenth century. On account of his doctor's death of plague, the Habsburg ambassador Busbecq mentioned the discovery of fleabites on the former's body, which was soon followed by death. He wrote, "He [William Quacquelben] himself noticed on his body, when it was stripped, a purple spot, which they declared was a flea-bite. However, seeing more and larger spots, he exclaimed, 'These are no flea-bites, but a warning that death is at hand.'"[78] Neither the physician himself, nor anyone else suspected that the fleabites had anything to do with his affliction. A similar example comes from Evliya Çelebi, who noted the absence of plague in places where he also noted the absence of rats and fleas, though making nothing of the connection. Rather, he attributed their absence to the power of spells or talismans, as we have seen earlier. The eighteenth-century Scottish physicians Alexander and Patrick Russell also observed fleabites on the bodies of plague patients in Aleppo but failed to make the connection.[79] By the same token, later accounts of Pouqueville and

account, one unit of *zırnık* was to be mixed with two units of quicklime and with water into a thick paste. This paste was to be applied to the skin where unwanted hair grew and washed away with water shortly afterward. For this purpose either the black or the yellow *zırnık* was used, which could be found everywhere in Istanbul. See Dernschwam, *Seyahat Günlüğü*, 80–81, 185–86. Schweigger also mentioned that both men and women in Istanbul used a powder in bathhouses to rid themselves of body hair. See Schweigger, *Sultanlar Kentine Yolculuk*, 131.

76 Evliya Çelebi, *Seyahatnâme*, 10:435.

77 İlyas bin İbrahim, *Majannah al-taʿun wa al-wabaʾ*. Süleymaniye Library, ms. Esad Efendi 2484/3, 28–42ff.; İlyas, *Tevfîkatü'l-hamidiyye fi defʿi'l-emrazi'l-vebaʾiyye*, trans. Ahmedü'ş-Şami Ömeri, Istanbul University Cerrahpaşa History of Medicine Library, ms. 105, 42, 45–47.

78 Ogier Ghiselin de Busbecq, *The Turkish Letters of Ogier Ghiselin de Busbecq* (Baton Rouge: Louisiana State University Press, 2005), 185.

79 van den Boogert, *Aleppo Observed*, 166.

Brayer attributed plague etiology to climatic and environmental factors. In the absence of the knowledge of vectors, Brayer comes close to identifying the problem by talking about the trade of secondhand clothes and other personal items of plague victims being a channel for the distribution of the disease, not only locally but also over long distances.[80] As for Pouqueville, he dismisses all evidence believed by locals to be signs of plague outbreaks. Among them, he mentions epizootics that took place concurrently, insects, and the presence of oil stains on walls (clear reference to traces left behind by rats), but dismisses them all.[81]

As a final point, some animals could have served a secondary role as transitory hosts to vectors, that is, plague-infected fleas. These can be wild or commensal rodents or other mammals that can transmit the disease. In particular, the role of carnivores, such as hyenas and weasels, feeding on infested rodents may be taken into consideration in this process.[82] For example, the Habsburg ambassador Busbecq mentioned hyenas that dug up human bodies from graves in sixteenth-century Anatolia and noted that people placed heavy stones on top of graves to protect them from hyenas.[83] Similarly, the fifteenth-century account of Pero Tafur commented on the abundance of weasels in Damietta both in the streets and in the houses.[84] More or less limited to local transmission, such activities would take place alongside other means of transmission and thus may be difficult to trace. However, one type of such transitory host deserves more careful consideration because of its potential to transmit the disease over long distances and cause metastatic leaps. Predator birds that fed on dead rodents, especially migratory birds, may be significant in the dissemination of infected fleas.[85] Sixteenth-century Ottoman plague treatises loosely observed a connection between the behavior of migratory birds and epidemics. For example, İlyas bin İbrahim mentioned that outbreaks of disease were preceded by certain environmental events, including the flight of certain animals and birds.[86] For some, the arrival of migratory birds, especially the white stork, was seen as a sign of a coming plague. The sixteenth-century theologian and biographer Ahmed Taşköprizade (d. 1561) mentions this in his comprehensive plague treatise. The appearance of certain species of insects and animals, such as

80 Brayer, *Neuf années à Constantinople*, 2:354–56.

81 Pouqueville, *Voyage en Morée*, 1:408.

82 Ruth I. Meserve, "Striped Hyenas and 'Were-Hyenas' in Central Eurasia," in *Archivum Eurasiae Medii Aevi*, ed. T. T. Allsen, P. B. Golden, R. K. Kovalev, and A. P. Martinez (Wiesbaden: Harrassowitz, 2012), 199–220. I am grateful to Ann G. Carmichael for bringing this piece to my attention.

83 Busbecq, *Turkish Letters*, 48–49.

84 Pero Tafur, *Travels and Adventures, 1435–1439*, ed. Malcolm Letts (New York: Harper, 1926), 68.

85 Benedictow, *Black Death*, 47.

86 *Tevfīkāt*, 28.

the white stork, was considered, according to Taşköprizade, as a precursor of plague.[87] This association between the arrival of migratory birds and that of the plague may have been based on coincidental seasonality, in the absence of knowledge about plague vectors. White storks (*Ciconia ciconia*) are predatory birds that feed on insects as well as small rodents, such as voles and possibly rats. Some of the practices of the white stork, such as feeding at garbage dumps and nesting at roofs, poles, and straw stacks, make them a prime candidate for carrying diseases.[88] Research has shown their role in carrying and spreading diseases, such as the West Nile virus.[89] It is suggested that migratory birds can be a factor in disseminating fleas infected by plague.[90] Interestingly, the migratory route followed by the white stork, from Europe to southeast Africa, crisscrossed the Ottoman lands from northwest to southeast and largely corresponded to the pilgrimage route in the eastern Mediterranean before crossing over the Sinai Peninsula to Egypt, Sudan, and farther south into Africa.[91] This trajectory also corresponded, as we shall we, with one of the major trade routes of the empire, and not surprisingly with one of the plague routes as well.[92]

The Pathogen

The pathogen causing plague is *Yersinia pestis*, a gram-negative bacillus that belongs to the group of enteric bacteria – the kind of pathogens that develop in the intestines of a host organism and spread through contaminated food

87 Ahmed Taşköprizade, *Risalah al-shifaʾ li-adwaʾ al-wabaʾ* ([Cairo]: al-Matbaʿah al-Wahbiyah, 1875); Süheyl Ünver, "Türkiye'de veba tarihçesi üzerine," *Tedavi Kliniği ve Laboratuvarı Mecmuası* 5 (1935): 70–71.

88 Willem van den Bossche, Peter Berthold, Michael Kaatz, Eugeniusz Nowak, and Ulrich Querner, *Eastern European White Stork Populations: Migration Studies and Elaboration of Conservation Measures* (Bonn: Bundesamt für Naturschutz (BfN)/German Federal Agency for Nature Conservation, 2002); Zdenek Hubálek, "An Annotated Checklist of Pathogenic Microorganisms Associated with Migratory Birds," *Journal of Wildlife Diseases* 40, no. 4 (2004): 639–59.

89 Mertyn Malkinson et al., "Intercontinental Transmission of West Nile Virus by Migrating White Storks," *Emerging Infectious Diseases* 7, no. 3(suppl.) (2001): 540; Mertyn Malkinson et al., "Introduction of West Nile Virus in the Middle East by Migrating White Storks," *Emerging Infectious Diseases* 8, no. 4 (2002): 392–97.

90 Lise Heier et al., "Emergence, Spread, Persistence and Fade-out of Sylvatic Plague in Kazakhstan," *Proceedings of the Royal Society, Series B* 278, no. 1720 (2011): 2915–23.

91 van den Bossche et al., *Eastern European White Stork Populations*. Incidentally, this migratory route went right over Istanbul, across the Bosphorus. Historical sources sometimes mention the sight of flocks of storks. For example, Dernschwam noted seeing flocks of thousands of storks near Edirne ([August 19, 1553]). See Dernschwam, *Seyahat Günlüğü*, 44.

92 For a discussion of the implications of transitory hosts of vectors, such as hyenas and white storks, to the spread of plague in the Ottoman context, see my "New Science and Old Sources," 213–16.

and water – though *Y. pestis* is quite atypical in its choice of host environment (blood) and the method of transmission (vector borne). Our knowledge of this pathogen is about 120 years old, even though it has been around for at least fifteen hundred years, and quite likely much longer. *Y. pestis* was first isolated in 1894 by Alexandre Yersin in Hong Kong during the Third Pandemic.[93] For most of the twentieth century, the body of scientific knowledge on the pathogen (*Pasteurella pestis*, later recognized as *Y. pestis*) came from scientific observations drawn from South Asia. However, the last decade witnessed revolutionary changes in *Y. pestis* research. The recent work of geneticists, especially from the perspective of evolutionary biology, has improved our understanding of this pathogen considerably. For example, we now know that *Y. pestis* did not exist from time immemorial, as it was once believed, but that it evolved from *Y. pseudotuberculosis*, another enteric bacterium, about fifteen hundred to twenty thousand years ago.[94] Hence, it is considered to be a young bacterium, with surprisingly limited genetic diversity, which makes it a model organism for studying bacterial virulence.

This new understanding of the recent evolution of the bacterium triggered further research efforts in the scientific community, resulting in the sequencing of the genome of *Y. pestis* for the first time. In 2001, a group of English scientists declared the triumphant significance of their effort as follows: "*Y. pestis* is a pathogen that has undergone large-scale genetic flux and provides a unique insight into the ways in which new and highly virulent pathogens evolve."[95] Continued efforts of biological archeologists and geneticists culminated ten years later in the reconstruction of the full genome of *Y. pestis* entirely from aDNA recovered from the remains of fourteenth-century plague victims buried in East Smithfield Cemetery in London.[96] Both molecular archeology and genetics research have contributed massively to our understanding of the evolutionary history of the pathogen and its adaptations to different environments. Every time *Y. pestis* acquires new genes

93 Alexandre Yersin, "La peste bubonique à Hong-Kong," *Annales de l'Institut Pasteur* 8 (1894): 662–67. A near-simultaneous discovery was realized by the Japanese physician and bacteriologist Shibasaburo Kitasato, a former student of Robert Koch.

94 Mark Achtman et al., "*Yersinia pestis*, the Cause of Plague, Is a Recently Emerged Clone of *Yersinia pseudotuberculosis*," *PNAS* 96, no. 24 (1999): 14043–48. More recent estimates project a slightly older date for the evolution of *Y. pestis*, e.g., see Yanjun Li et al., "Genotyping and Phylogenetic Analysis of *Yersinia pestis* by MLVA: Insights into the Worldwide Expansion of Central Asia Plague Foci," *PLoS ONE* 4, no. 6 (2009): e6000; Yujun Cui et al., "Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*," *PNAS* 110, no. 2 (2013): 577–82.

95 J. Parkhill et al., "Genome Sequence of *Yersinia pestis*, the Causative Agent of Plague," *Nature* 413, no. 6855 (2001): 523–27, quote on 523.

96 Kirsten I. Bos et al., "A Draft Genome of *Yersinia pestis* from Victims of the Black Death," *Nature* 478, no. 7370 (2011): 506–10.

and loses others, these changes indicate important genetic events in its environment, host susceptibility, or vector dynamics. It was demonstrated, for example, that at some point during its evolution, *Y. pestis* adapted to the flea environment and began to be transmitted by fleas efficiently.[97] This meant that *Y. pestis* first acquired the critical ability to colonize the flea so as to be transmitted as a flea-borne septicemic disease of limited transmissibility. From the vantage point of the bacterium, this transition would diminish its evolutionary chances of survival. It was only later, as a result of the acquisition of new genes, that *Y. pestis* obtained the bubonic form and hence increased its capacity for epidemic spread.[98] Questions of the bacterium's evolutionary history were accompanied by questions of its origin. Even though where *Y. pestis* first originated exactly is still contentious, most recent research suggests its origins to be "in or near the Qinghai-Tibet Plateau," followed by its distribution to other areas as a result of rodent and human migration and travel.[99] Historically, this spread is believed to have taken place across three pandemics.[100]

In the mid-twentieth century, it was proposed that *Y. pestis* had three biovars or biotypes (Antiqua, Medievalis, and Orientalis), assumed to have caused the First, Second, and Third pandemic, respectively.[101] This typology – which was produced on the basis of the bacillus's nutritional properties, more specifically, its ability to ferment glycerol and reduce nitrate – was widely accepted in the scientific community and remained as the dominant paradigm of its classification until recently. In this scheme, the evolutionary context of these differences between *Y. pestis* biovars went largely unnoticed.[102] New research grew critical with this typology and taxonomy of *Y. pestis* on grounds that the differentiation of the three biotypes could

97 Unlike *Y. pestis*, *Y. pseudotuberculosis* is orally toxic to fleas, which suggests evolutionary changes between the pathogen and the vector. David L. Erickson et al., "Acute Oral Toxicity of *Yersinia pseudotuberculosis* to Fleas: Implications for the Evolution of Vector-borne Transmission of Plague," *Cellular Microbiology* 9, no. 11 (2007): 2658–66; B. J. Hinnebusch, "The Evolution of Flea-Borne Transmission in *Yersinia pestis*," *Current Issues in Molecular Biology* 7 (2005): 197–212.

98 Florent Sebbane et al., "Role of the *Yersinia pestis* Plasminogen Activator in the Incidence of Distinct Septicemic and Bubonic Forms of Flea-borne Plague," *PNAS* 103, no. 14 (2006): 5526–30.

99 Cui et al., "Historical Variations."

100 For the periodization of plague pandemics, see the introduction.

101 R. Devignat, "Variétés de l'espèce *Pasteurella pestis*: nouvelle hypothèse," *Bulletin of the World Health Organization* 4, no. 2 (1951): 247–63.

102 An exception to this was John Norris's suggestion that adaptation to different rodent species may have been responsible for biochemical differences of *Y. pestis* biovars. See Norris, "East or West? The Geographic Origin of the Black Death," *Bulletin of the History of Medicine* 51, no. 1 (1977): 22–24. This point is also highlighted in George D. Sussman, "Was the Black Death in India and China?," *Bulletin of the History of Medicine* 85, no. 3 (2011): 331.

not be translated into genetic changes in the pathogen's history. In a pioneering article that came out in 2004, an international group of researchers rejected the use of biovars for evolutionary or taxonomic purposes. Instead, they proposed that *Y. pestis* be subdivided into populations based on molecular groupings.[103] Even though some early efforts within the scientific community tried to refute the hypothesis of matching the three biovars with the three pandemics,[104] the methods used were not accepted as sound.[105] Because of the limited pool of modern *Y. pestis* strains (most isolated in the second half of the twentieth century) and even a smaller sample of aDNA fragments, the scientific community had to wait for more aDNA evidence from past pandemics and for better means of analysis before an association between modern molecular groupings and premodern pandemics could be confidently advanced.[106] More robust studies, using a much greater pool of *Y. pestis* isolates and more rigorous methods of analysis, only started to come about toward the end of the decade. Hence, in 2009, an international team of researchers firmly rejected the position that the Orientalis biovar could have been responsible for all three pandemics.[107] This was followed, the next year, by another authoritative phylogenetic study based on a broad spectrum of *Y. pestis* aDNA recovered from plague pits throughout Europe (dated to the Black Death and its successive waves). This team declared, "The strains causing mass deaths were unrelated to either Medievalis or Orientalis biovars."[108] The scientific community seemed to have left behind the use of biovars for purposes of taxonomic and genetic classification.

Questions of transmission also seem to have benefited from phylogenetic methods. A nuanced analysis of variations in *Y. pestis*'s evolution may offer invaluable insights for historical pandemics. The evolution of *Y. pestis* in times of epidemics and epizootics seems to be much faster than in enzootic periods of inactivity because of the higher rates of bacterial replication involved. This means that demographic changes can affect the pathogen's speed of evolution, which has tremendous implications for understanding

[103] Mark Achtman et al., "Microevolution and History of the Plague Bacillus, *Yersinia pestis*," *PNAS* 101, no. 51 (2004): 17837–42.

[104] Michel Drancourt et al., "Genotyping, Orientalis-like *Yersinia pestis*, and Plague Pandemics," *Emerging Infectious Diseases* 10, no. 9 (2004): 1585–92.

[105] For a summary account of why this finding was criticized and how it was refuted, see Michaela Harbeck et al., "*Yersinia pestis* DNA from Skeletal Remains from the 6th Century AD Reveals Insights into Justinianic Plague," *PLoS Pathogens* 9, no. 5 (2013): e1003349.

[106] In 2007, some French researchers triumphantly announced that all three historical pandemics were caused by the Orientalis biovar. See Michel Drancourt et al., "*Yersinia pestis* Orientalis in Remains of Ancient Plague Patients," *Emerging Infectious Diseases* 13, no. 2 (2007): 332–33. This was once again followed by a round of stern criticism. See Harbeck et al., "*Yersinia pestis* DNA from Skeletal Remains."

[107] Li et al., "Genotyping and Phylogenetic Analysis of *Yersinia pestis* by MLVA."

[108] Stephanie Haensch et al., "Distinct Clones of *Yersinia pestis* Caused the Black Death," *PLoS Pathogens* 6, no. 10 (2010): e1001134; quote on 2.

historical pandemics. It follows that a greater number of hosts would mean a faster rate of bacterial replication and thus would imply the possibility of faster evolution. In other words, large colonies of ground-burrowing or commensal rodents or, alternatively, crowded urban areas might have been instrumental in the process of the emergence of different *Y. pestis* populations and lineages. In fact, researchers suspect that this is exactly what happened during the Black Death. Nonetheless, they do not rule out the involvement of other factors, such as variations in host density, that may result from climatic and environmental changes.[109]

Biologists caution us that more than one strain of a pathogen might be at work in a given epidemic or pandemic. They argue that "at least two related but distinct genotypes of *Y. pestis* were responsible for the Black Death and suggest that distinct bacterial populations spread throughout Europe in the 14th century."[110] Another team of researchers further supported this conclusion by demonstrating that several *Y. pestis* genotypes circulated in medieval Europe.[111] The fact that distinct bacterial populations were circulating in a given epidemic may suggest that these distinct entities came from different places, along different routes, and/or at different times.

Scientific studies in plague research continue at full pace. What has become clear is that the implications of this new body of scholarship for studying past pandemics can no longer be ignored by historians. Tracing the movements of different populations of *Y. pestis* and correlating them to different historical periods has tremendous implications for the temporal and spatial identification of *Y. pestis* in the historical study of plague pandemics. Thus, genetic changes of the pathogen serve as markers of temporal and spatial spread of historical pandemics. In the light of this body of research and its implications, it should be possible to make new historical suggestions. Nevertheless, despite the vast array of research on *Y. pestis* and the many questions addressed by plague scientists, it is still difficult to nail down some of our immediate historical questions. For example, were the Ottoman areas visited by the same strain of *Y. pestis* as those in other parts of Eurasia? How many different strains of the bacterium circulated in the Ottoman Empire throughout the early modern era? Which plague foci were the source of these strains in this era? Answers to these questions require collaboration between bioarcheologists and geneticists. Currently no archeological dig from the Ottoman period has aimed to find evidence of *Y. pestis* in the aDNA of the remains of suspected plague victims. We can only hope for such evidence to be revealed in the future. For now, we are not in a position to answer most of these questions with clarity. Nevertheless, the evidence drawn from digs in Europe may shed some light

109 Cui et al., "Historical Variations."
110 Haensch et al., "Distinct Clones of *Yersinia pestis*," 4.
111 Tran et al., "Brief Communication."

on our concerns, especially when combined with the available historical evidence.[112]

Alternatively, it is also possible to draw from modern *Y. pestis* isolates that were collected from former Ottoman areas (including Turkey, northern Iraq, and western Iran) and included in recent phylogenetic analyses. These studies shed some light on where these strains originated with respect to the evolutionary subdivisions of *Y. pestis*.[113] An article published in Turkey in 1952 presented four *Y. pestis* isolates that were preserved in Refik Saydam Institute in Ankara, three of which were defined as biotype Orientalis.[114] Of the four isolates, three were not clearly identified as to when or where they were isolated. The authors believe they were isolated in Istanbul and Antalya. One isolate was known to have been isolated in a human case of plague in the Akçakale (Urfa) outbreak of 1947. This was a small outbreak of plague – in February and March, a total of thirteen deaths took place out of a total of eighteen cases affected – in two Turkish villages on the Syrian border where bubonic cases were identified. In the absence of recent plague outbreaks in Turkish port cities prior to it, this outbreak puzzled the authors, who personally observed the epidemic in the field. The presence of an excess of number of fleas was reported in the dwellings, which the authors believed was responsible for transmitting the disease from person to person. Where the epidemic took place, house mice were observed in great numbers, but not a single rat was found, which led the authors to believe that humans were accidentally infected by a plague of sylvatic character.[115] The 1947 outbreak appears to be the last recorded outbreak of plague in Turkey, with no record of further human cases.

Humans

Despite being only accidental hosts to plague, humans have been perhaps the most important of all protagonists in shaping the natural history of this disease. How did human agency make a difference in spreading or containing

[112] For a more detailed discussion of the growing imbalance between the "new science" and the historical sources in the study of plague in the Ottoman Empire, see my "New Science and Old Sources."

[113] 2.MED1, isolated from this region, evolved more than 235 years ago (in 2010), which places it before 1775, i.e., before the Third Pandemic. Similarly, 1.ORI3 is thought to have come from Madagascar during the Third Pandemic, most probably via the pilgrimage route. See Giovanna Morelli et al., "*Yersinia pestis* Genome Sequencing Identifies Patterns of Global Phylogenetic Diversity," *Nature Genetics* 42, no. 12 (2010): 1140–43.

[114] Bilal Golem and Kemal Özsan, "Türk Veba Suşlarında Biyoşimik Karakter Farkları," *Türk İjiyen ve Tecrübi Biyoloji Dergisi* 12, no. 1 (1952): 29–51.

[115] The same observation regarding the absolute absence of rats in the area was further confirmed in 1955 by Xavier Misonne. Xavier Misonne, "Mammifères de la Turquie sud-orientale et du nord de la Syrie," 53–68.

the disease or in changing its course?[116] The human actors come into play in different capacities. For example, as hosts to plague, infected individuals can directly infect other human beings, which is known to happen in the pneumonic form of the disease. Humans can also alter the course of an epidemic by efforts at controlling, containing, and now treating the disease (with antibiotics). However, most important, humans can (inadvertently) facilitate the movement of plague hosts and vectors beyond the natural abilities of these agents and carry the disease over long distances as a result of their own movements. Hence, they provide enhanced means of mobility to plague hosts and vectors that have limited ability to move. In other words, the long distance spread of *Y. pestis* is mostly owed to human agency in moving infected rodents and/or vectors from one place to another. This could happen in different forms and through varying activities involved in human mobility, such as travel, migration, or transportation of goods. As we shall see in more detail, all of these human activities have contributed to circulating the plague within the Ottoman domains and beyond.

Among various forms of mobility, warfare is long known to affect the spread of epidemics, perhaps must notably in the dissemination of the plague out of the Genoese colony of Caffa at Crimea, besieged by a Mongol army in 1346.[117] Warfare certainly contributed to both the local and long-distance spread of the disease. The movement of large numbers of people, close army encampments, and the lack of hygienic conditions have been associated with outbreaks since the ancient period and elaborated in the *miasma* paradigm. According to this paradigm, the stench and putrid vapors rising from rotting corpses of soldiers fallen dead on the battlefield could contaminate the air and produce miasma, considered to be the cause of epidemics.

As we shall see in greater detail in Part II, we do not lack examples in late medieval and early modern Ottoman history to link the spread of plague to warfare. The fourteenth through seventeenth centuries were marked by intense military activity in Ottoman history, in which massive territorial expansion took place, accompanied by the simultaneous expansion of plague. Some military practices used by the Ottomans, such as digging

116 For a comprehensive overview of the human experience with epidemic infectious diseases, including the plague, see Ann G. Carmichael, "Infectious Disease and Human Agency: An Historical Overview," in *Interactions between Global Change and Human Health*, 3–46 (Vatican City: Pontificia Academia Scientiarum, 2006).

117 Friedrich Prinzing, *Epidemics Resulting from Wars* (Oxford: Clarendon Press, 1916). Even though the Italian chroniclers of the Black Death have claimed that the disease was transmitted to the Genoese as a result of the plague corpses being catapulted and thrown at them, modern epidemiological knowledge does not support such a method of transmission. Instead, it has been proposed that infected rodents from the army encampments must have found their way of introducing the infection to the commensal rodents of the town. For a detailed analysis for why the catapulting story does not work, see Benedictow, *Black Death*, 52–53.

underground tunnels during sieges, may have added the additional risk of exposing soldiers to rodents' burrows and possibly to the pathogen kept alive in the soil or in the dead tissues of rodents. The zigzagging underground tunnels the Ottomans used for sieges were fittingly known as *sıçan yolları* (rat tunnels).[118]

Other forms of human mobilities may be worth considering in this context. Among them, pilgrimage involved the movement of large numbers of people across long distances. Even though we do not know the precise number of people traveling to and from the Muslim holy cities of Mecca and Medina every year, the figures were significant enough in premodern standards of long-distance travel. Pilgrims who sometimes traveled and camped in poor hygienic conditions were also prime candidates for local outbreaks and to some extent can be associated with the movement of diseases. This is especially significant for the era of focus here. As we shall see, when the Ottomans took over the pilgrimage routes, they took measures to improve the safety of the journey, which resulted in an even greater number of pilgrims.[119]

Migration constituted another such form of human mobility in Ottoman society. *Sürgün* and *şenlendirme*, policies of resettlement used by the

[118] Even though there is no direct bioarcheological evidence at hand to support this from the Ottoman areas, it may be possible to draw analogies from studies conducted elsewhere. For example, recent research has confirmed cases of coinfection of louse-borne trench fever (*Bartonella quintana*) and plague (*Y. pestis*) in a late medieval mass burial site in France. See Tran et al., "Brief Communication." For associations to epidemic typhus (*Rickettsia prowazekii*) and other louse-borne infections, see Didier Raoult et al., "Evidence for Louse-Transmitted Diseases in Soldiers of Napoleon's Grand Army in Vilnius," *Journal of Infectious Diseases* 193, no. 1 (2006): 112–20; Tung Nguyen-Hieu et al., "Evidence of a Louse-Borne Outbreak Involving Typhus in Douai, 1710–1712 during the War of Spanish Succession," *PLoS ONE* 5, no. 10 (2010): e15405. Considering the louse-borne nature of these infections and evidence for their occurrence, especially in soldiers, it should be possible to seek further links to occurrences of plague. For plague transmission via lice, also see note 57. Given the notorious threat of epidemic typhus in Hungary – known as *morbus hungaricus* – in the early modern era, especially for soldiers, there may be further reason to explore such links. See Gábor Ágoston, "Where Environmental and Frontier Studies Meet: Rivers, Forests, Marshes and Forts along the Ottoman–Hapsburg Frontier in Hungary," in *The Frontiers of the Ottoman World*, ed. A. C. S. Peacock (Oxford: Oxford University Press, 2009), 78.

[119] Ottoman pilgrimage routes are explored in conjunction with plague in Chapter 5. Bruce Masters, "Hajj," in *Encyclopedia of the Ottoman Empire*, ed. Gábor Ágoston and Bruce Alan Masters, 246–48 (New York: Facts on File, 2009); Suraiya Faroqhi, *Pilgrims and Sultans: The Hajj under the Ottomans, 1517–1683* (London: I. B. Tauris, 1994). Also see Richard Blackburn, ed. and trans., *Journey to the Sublime Porte: The Arabic Memoir of a Sharifian Agent's Diplomatic Mission to the Ottoman Imperial Court in the Era of Suleyman the Magnificent* (Beirut: Orient-Institut, 2005). The connection between pilgrimage and epidemic diseases has been better explored for the late Ottoman era. See, e.g., Michael Christopher Low, "Empire and the Hajj: Pilgrims, Plagues, and Pan-Islam under British Surveillance, 1865–1908," *IJMES* 40, no. 2 (2008): 269–90.

Ottoman administration involving forced relocation of entire populations, were most rigorously pursued by Mehmed II (r. 1451–81) as a tool of demographic engineering. These policies were sometimes used to secure underpopulated frontier areas. It was also at times an important concern for Ottoman rule to populate newly conquered areas by Muslim subjects or to relocate the landed aristocracy of a conquered area to limit their power.[120] Even though this was an older practice used by the Byzantines for repopulating imperial domains, and most significantly enforced in the wake of epidemic outbreaks, the Ottomans have pursued such policies thoroughly.[121] As such, population policies intimately linked demographic losses caused by plague in the cities to those of their hinterlands.

The policies of forced migration were also accompanied by voluntary immigration, which constantly increased the Ottoman urban population, most prominently in the sixteenth century. Generally speaking, the fifteenth and sixteenth centuries witnessed the rise and development of many new urban clusters throughout the Ottoman realm. The process of urbanization, however slow in the beginning, took a definitive character in the sixteenth century, when several villages in Anatolia grew into new towns and undistinguished cities developed into thriving metropolises.[122] Such urban clusters with dense populations where people lived in close proximity provided the best environment for the local and regional spread of diseases. As we shall see in greater detail, there was an intimate link between the intensification of urbanization and plague epidemics in early modern Ottoman history.[123]

Moreover, there were mass population movements in this period. For example, an estimated fifteen thousand to twenty thousand Iberian Jews

[120] See e.g., Halil İnalcık, *An Economic and Social History of the Ottoman Empire. Vol. I: 1300–1600* (Cambridge: Cambridge Univerity Press, 1994), 167–71; Cengiz Orhonlu, *Osmanlı İmparatorluğu'nda Aşiretlerin İskanı* (Istanbul: Eren, 1987); İbrabim Solak, "Anadolu'da Nüfus Hareketleri ve Osmanlı Devleti'nin İskan Politikası," *Türk Dünyası Araştırmaları* 127 (2000): 157–92.

[121] Following an epidemic, in 754–55 CE, large numbers of people from the Greek peninsula and islands and the Peloponnese were sent to the capital to repopulate it. See Stathakopoulos, *Famine and Pestilence*, 385. Cf. Kritovolus's description of Mehmed's policy of population management in *History of Mehmed the Conqueror* (Princeton, NJ: Princeton University Press, 1954).

[122] For the rise and development of urban centers in the sixteenth century, see Suraiya Faroqhi, *Towns and Townsmen of Ottoman Anatolia: Trade, Crafts and Food Production in an Urban Setting, 1520–1650* (Cambridge: Cambridge University Press, 1984); Ronald C. Jennings, "Urban Population in Anatolia in the Sixteenth Century: A Study of Kayseri, Karaman, Amasya, Trabzon, and Erzurum," *IJMES* 7, no. 1 (1976): 21–57.

[123] This connection is more thoroughly explored in Chapter 4, in the examples of Bursa, Edirne, and Istanbul in the fifteenth and sixteenth centuries. In Europe, it has been observed that certain professionals, such as bakers, butchers, leather/tannery workers, and artisans handling fabric and paper, were at greater risk of infection at times of plague. See Audoin-Rouzeau, *Les chemins de la peste*, 233–38.

arrived in Ottoman lands toward the end of the fifteenth century.[124] Similarly, seasonal migration of various communities should be taken into account. Pastoralist nomads of Anatolia and the Balkans moved between their summer pastures and winter encampments, between highlands and lowlands. Seasonal workers sought employment in other places. As much as it is difficult to quantify these movements, the seasonality and trajectories of such movements can be established in the sources.[125] In addition to these forms of movement and migration, it should be possible to add the travel of couriers, administrators, officials, and so on. As Ottoman power grew and expanded, and centralization took hold, a growing number of officials were appointed to different locations, where they traveled with their staffs and households. When one takes into account that these officials held appointments for short durations, the number of people who traveled on state duty alone seems to add up to a substantial figure.[126]

Among all forms of human mobility, trade and the transportation of goods are perhaps the most significant. Trade made it possible for people, rats, and fleas to move over considerable distances. Maritime trade in this period was of tremendous importance. Ships were known to transport rats in addition to humans and cargo. A sixteenth-century testimony makes clear that this was known and that precautions were taken against these unwelcome passengers accordingly. Salomon Schweigger wrote that the Ottomans had the habit of carrying weasels or cats on board ships expressly for the purpose of "rat control."[127] Even though all forms of trade could facilitate the metastatic growth of the disease, maritime trade was ideal because of its greater pace and the possibility for rats to travel along in vessels. For example, grain trade almost guaranteed the movement of plague. Grain warehouses attracted rats and provided a suitable habitat in which fleas could live. Shipping grain would almost guarantee shipping rats and fleas along with it.[128] Like grain, other trade items, such as wool, woolen cloths,

124 Benjamin Braude, "The Rise and Fall of Salonica Woollens, 1500–1650: Technology Transfer and Western Competition," *Mediterranean Historical Review* 6, no. 2 (1991): 218.

125 For a detailed discussion of the connections between plague and higher altitudes, as well the implications of this research on the movement of pastoralist nomads in the Ottoman landscape, see Chapter 3.

126 For the growing number of appointees in the Ottoman system of provincial administration, the short term of service, considerable retinues and soldiers, and continuous reshuffling, the classic study is Metin I. Kunt, *The Sultan's Servants: The Transformation of Ottoman Provincial Government, 1550–1650* (New York: Columbia University Press, 1983). More recently, a concise overview of provincial administration was offered in Colin Imber, *The Ottoman Empire, 1300–1650: The Structure of Power* (Houndmills, UK: Palgrave, 2002), 177–215. For a call to a more nuanced vision, with respect to regional variations, see Gábor Ágoston, "A Flexible Empire: Authority and Its Limits on the Ottoman Frontiers," *International Journal of Turkish Studies* 9, nos. 1–2 (2003): 15–31.

127 Schweigger, *Sultanlar Kentine Yolculuk*, 115.

128 McCormick, "Rats, Communications, and Plague."

hides, and fur, could also shelter fleas, if not rats, for several weeks and even months.

It is argued in this book that the process of empire building in the long sixteenth century contributed significantly to increased human mobility. Even though it is difficult to trace and quantify these forms of mobility temporally and spatially, it should be plausible to conceive their contribution to the increased pace and scope of epidemiological activity. Needless to say, more research is needed to explore the various links between empire building and disease ecologies in the early modern Ottoman case.

The Environment

It should be remembered that the etiology of plague involves a complex system of entanglements in which every agent (such as host, vector, and pathogen) is in constant interaction with others as well as with the greater environment around it. As such, the environment is one of the main protagonists of plague etiology because of its tremendous capacity to trigger, sustain, or diminish plague activity; any slight change in the environment can cause a series of changes in the entire complex. Today there is a fairly well established body of knowledge, regarding the behavior of the pathogen, its relationship with its hosts and vectors, and how it adapts to new environments. Nevertheless, we also know that experiences of plague may change from one place to another because of differences in disease ecologies. In other words, the knowledge of plague etiology cannot be applied universally; because plague behaves differently in different environments, its etiology is more like a guideline that should be read in conjunction with specific local conditions.

From the vantage point of plague, there are two different environments. One is the natural environment, the other the built environment of human settlements, towns, and cities. Historically speaking, during the long stretch of plague out of its place of origin in Asia, the disease was spread to numerous regions by different hosts, vectors, or the mediation of humans. Once introduced to a new area, if the pathogen found a favorable ecosystem for its survival, it lived among the wild rodents. In other words, it became enzootic among rodents susceptible to the disease but generally resistant to the infection. These places became reservoirs or plague foci, in which the disease was kept in naturally occurring cycles of activity and nonactivity.[129] As long as

[129] In addition to living in wild rodent hosts, there is also some evidence that *Y. pestis* survives in flea feces, in postmortem rodent hosts, in soil, and in plants. See Gage and Kosoy, "Natural History of Plague"; W. Ryan Easterday et al., "An Additional Step in the Transmission of *Yersinia pestis*?," *ISME Journal* 6, no. 2 (2012): 231–36; Drancourt et al., "*Yersinia pestis* as a Telluric, Human Ectoparasite-Borne Organism"; Saravanan Ayyadurai et al., "Long-Term Persistence of Virulent *Yersinia pestis* in Soil," *Microbiology* 154,

the disease is not transmitted to humans, it is difficult to know much about its enzootic (sylvatic) existence.

According to the World Health Organization (WHO), plague foci fall, for the most part, between the 55 degrees north and 40 degrees south parallels. Some of these foci extend over substantial areas in the western United States, the Russian Federation, China, Mongolia, and southern Africa. For our more immediate area of concern, the plague foci in or around Ottoman areas are known to be located in Libya, Yemen, Iran, the Transcaucasian, and the northwest Caspian regions.[130] These plague foci were active in the Third Pandemic, and perhaps even before. They were identified in the second half of the twentieth century, and there is no precise information as to how old they are. Some of these foci are believed to be older than others. For example, historian William McNeill claimed in the 1970s that while the foci in central Africa and the Himalayan foothills were older, the steppe foci across Eurasia were formed not before the fourteenth century.[131] Chinese epidemiologist Wu Lien-Teh suggested in the 1920s that twelve plague foci antedated the Third Pandemic: two in Africa, ten in Asia (including the Assyr in the western Arabian Peninsula and the highlands of what is today southeast Turkey, northern Iraq, and western Iran).[132] According to Daniel Panzac, some of these foci can be traced as far back as the eighteenth century. Distinguishing between permanent and temporary plague foci in the Ottoman Empire, Panzac claims that the highlands between western Iran, northern Iraq, and southeastern Turkey as well as the mountainous areas of Hijaz and Yemen were permanent foci that supplanted the infection in the eighteenth and nineteenth centuries. Among the temporary foci, he listed the western Balkans focus, Moldavia and Wallachia, Istanbul, the Anatolian peninsula, and Egypt.[133]

Identifying plague foci of the earlier Ottoman eras may be challenging. It may be erroneous to assume that current or recent foci existed long before. It should be remembered that enzootic foci are dynamic complexes. One needs to use caution in making assumptions about the presence and/or function

no. 9 (2008): 2865–71; Rebecca J. Eisen et al., "Persistence of *Yersinia pestis* in Soil Under Natural Conditions," *Emerging Infectious Diseases* 14, no. 6 (2008): 941–43. For a study of *Y. pestis*'s survival in water, see David R. Pawlowski et al., "Entry of *Yersinia pestis* into the Viable but Nonculturable State in a Low-Temperature Tap Water Microcosm," *PLoS ONE* 6, no. 3 (2011): e17585.

130 David T. Dennis, Kenneth L. Gage, Norman Gratz, Jack D. Poland, and Evgueni Tikhomirov, "Plague Manual: Epidemiology, Distribution, Surveillance and Control," WHO/CDS/CSR/EDC/99.2 (Geneva, Switzerland: WHO, 1999); Anisimov, Lindler, and Pier, "Intraspecific Diversity of *Yersinia pestis*."

131 William McNeill, *Plagues and Peoples* (Garden City, NY: Anchor Press, 1976), 137–40.

132 Wu Lien-Teh, "The Original Home of Plague," in *Far Eastern Association of Tropical Medicine, Transactions of the Fifth Biennial Congress Held at Singapore, 1923*, ed. A. L. Hoops and J. W. Scharff, 286–304 (London: John Bale/Danielsson, 1924).

133 Panzac, *La peste*, 105–33.

of a present plague focus in the past. It is difficult to know how old each of these foci is and how long it has remained active. Although under favorable climatic and environmental conditions, plague may seem to remain enzootic indefinitely, myriad changes – ranging from an increase or decrease in the number of predators of wild rodents to rodent migration, from climate to changes in the use of landscape – can make a difference. An old plague focus can shrink or even disappear, and new ones can emerge. Hence, a current plague focus does not guarantee its presence and function in the same manner in the past. While studying the natural history of plague in the Ottoman areas, one needs to take into account where the plague foci were, when they were formed, and how they were connected to the more densely populated human areas to replenish new epidemic outbreaks.

This difficulty in identifying the plague foci of the early modern Ottoman era largely arises from the imprecise and lacunous nature of the sources. Only rarely do early modern accounts specify where plague came from in a manner that would allow tracing the area of known (or suspected) origin. Even then, this reflects rumors or hearsay of the locals about it. By the same token, the importation of the infection to port cities by means of maritime contacts with other infected cities makes it difficult to trace the origins of an outbreak to a particular plague focus. This is further complicated by the possibility of the infection being introduced from multiple foci and/or via multiple channels. For any given past outbreak, it is possible that we are looking at multiple strains of the pathogen circulating through different trajectories. Unfortunately, the available sources do not allow making such micro-scale observations. What can be more confidently ascertained is that some Ottoman cities or areas seem to have been continuously affected by plague in the sixteenth century, first and foremost among them Istanbul, whose emergence as a plague hub is examined in detail here.[134] Similarly, Egypt, Syria, and several cities of coastal Anatolia and the Balkans are documented to have witnessed numerous waves of plague in the early modern era. Despite the unremitting presence of plague in these areas, it is difficult to know whether the infection was introduced each time from outside or was sustained by means of commensal rodents and/or ectoparasites from one plague season to the next, thus acting as independent urban plague foci.

Generally speaking, plague epidemics are related to a variety of environmental conditions, such as changes in climate (temperature, humidity, precipitation, and winds), changes in landscape, vegetation, and the levels of radiation. Drawing from a wealth of sources and scientific analyses, historian Bruce Campbell demonstrates how the emergence of a plague pandemic in the fourteenth century was related to global climatic and environmental conditions.[135] For the most part, though, the effort to understand and study

[134] See Chapter 6.

[135] Bruce M. S. Campbell, "Physical Shocks, Biological Hazards, and Human Impacts: The Crisis of the Fourteenth Century Revisited," in *Le Interazioni Fra Economia E*

the ways in which plague related to environmental changes is frustrated by the very nature of these relations. The environmental changes that can be associated with changes in plague are not easy to identify, as they do not entail direct causal links. They involve the agency of a complex series of factors and thus can be difficult to identify and study. For example, increased precipitation is generally held to bring increased plague activity. The trophic cascade hypothesis can help relate increased precipitation to epizootics in a chain reaction in natural foci (increased precipitation → increased plant size → increased food supply for rodents → increased rodent population → critical threshold exceeded → epizootic).[136] In the Ottoman context, such connections need to be explored especially with respect to the impact of the Little Ice Age on Ottoman plagues in the early modern era. The northern hemispheric cooling starting in the second half of the sixteenth century seems to have adversely affected the plague activity of the region owing to a combination of reasons related to changes in flora and fauna biodiversity, habitat destruction of rodents, and changes in uses of landscape.[137]

In an urban context, increased precipitation may entail a different set of relations between hosts, vectors, and humans. For example, changes in temperature do not seem to affect commensal rats directly in an urban context. Black rats that live indoors have relatively stable living conditions, such as access to food and regulated temperatures of homes.[138] In a similar vein, a study conducted in Egypt in the 1990s found no significant variations of the rat population throughout the year; seasons did not seem to make a major difference.[139] Nevertheless, in rainy seasons, when outdoor humidity is high, rats prefer to stay in indoor human environments, where there is stored food. Humans are also more likely to stay indoors in the rainy season, which may increase the potential physical proximity between commensal rats and humans.[140] Temperature and humidity seem to matter

Ambiente Biologico nell'Europa Preindustriale. Secc. XIII–XVIII (Economic and Biological Interactions in Pre-Industrial Europe from the 13th to the 18th Centuries), ed. Simonetta Cavaciocchi (Florence: Firenze University Press, 2010): 13–32.

136 R. R. Parmenter et al., "Incidence of Plague Associated with Increased Winter-Spring Precipitation in New Mexico," *American Journal of Tropical Medicine and Hygiene* 61, no. 5 (1999): 814–21.

137 Geoffrey Parker, *Global Crisis: War, Climate Change and Catastrophe in the Seventeenth Century* (New Haven, CT: Yale University Press, 2013). For the Ottoman case, see White, *Climate of Rebellion in the Early Modern Ottoman Empire*; White, "The Little Ice Age Crisis of the Ottoman Empire: A Conjuncture in Middle East Environmental History," in *Water on Sand*, ed. Alan Mikhail, 71–90; Faruk Tabak, *The Waning of the Mediterranean, 1550–1870: A Geohistorical Approach* (Baltimore: Johns Hopkins University Press, 2008).

138 J. E. Brooks and F. P. Rowe, *Commensal Rodent Control* (Geneva, Switzerland: WHO, Vector Biology and Control Division, 1987), 13–14.

139 S. Soliman et al., "Seasonal Studies on Commensal Rats and Their Ectoparasites in a Rural Area of Egypt: The Relationship of Ectoparasites to the Species, Locality, and Relative Abundance of the Host," *Journal of Parasitology* 87, no. 3 (2001): 545–53.

140 Jacques M. May, "Map of the World Distribution of Plague," *Geographical Review* 42, no. 4 (1952): 629.

even more for plague vectors because of the nature of the flea's life cycle. Favorable climatic conditions are critical for flea eggs to hatch into larvae and eventually become adult fleas, the only form in which they perform their function as vectors.

Evidently, aside from climatic factors, other changes in the natural or built environment can alter plague etiology, though we do not know as much about the exact mechanisms at work. For example, an earthquake may dislocate ground-burrowing wild rodents from their natural habitat and force them to migrate elsewhere.[141] Similarly, floods can force such dislocations.[142] Such migrations, because they may bring wild rodents into contact with commensal rodents and/or humans, may lead to a plague epidemic. In fact, early modern observers have identified some of these associations that related plague to a larger environmental context. The dominant plague etiology that emphasized miasma had close ties to changes in climate, cosmic, and celestial phenomena that were believed to affect the quality of the air. The sources have often presumed a link between plague and unusual celestial phenomena, such as comets, lunar and solar eclipses, and the like. In that paradigm, the links between epidemic disease and changes in the greater environment have been commonly observed. I shall limit myself to two examples here drawn from late medieval and early modern Ottoman witnesses to plague. First, the aforementioned plague treatise of İlyas bin İbrahim insisted that plagues break out after earthquakes. He claims to draw this view from Aristotle, who posited that during earthquakes, poisonous underground vapors are unleashed to the surface of the earth and, while rising through the air, corrupt the substance of the air and form miasma, leading to epidemics. In fact, İlyas claims to have written his plague treatise following a big earthquake in Istanbul so as to offer means of prevention from the disease and methods of treatment.[143] Second, writing in the second half of the fifteenth century, the Greek historian Kritovoulos of Imbros commented on the unusual celestial phenomena observed before the appearance of plague in 1467. He wrote that a sudden and bright light appeared in the sky, which he did not know whether was a comet or a star. He certainly

141 See Tsiamis et al., "Earthquakes and Plague during Byzantine Times: Can Lessons from the Past Improve Epidemic Preparedness?," *Acta Medico-Historica Adriatica* 11, no. 1 (2013): 55–64.

142 An example for excess rain and flooding leading to plague, possibly as a result of forcing dislocation of rats, can be seen in the outbreak of 1791 in Egypt. For a detailed account of this outbreak, see Alan Mikhail, "The Nature of Plague in Late Eighteenth-Century Egypt," *Bulletin of the History of Medicine* 82, no. 2 (2008): 249–75; Mikhail, "Plague and Environment in Late Ottoman Egypt," in *Water on Sand*, 111–31.

143 İlyas, *Tevfīkāt*. This was a common view in Europe in the seventeenth century. See Daniel Gordon, "Confrontations with the Plague in Eighteenth-Century France," in *Dreadful Visitations: Confronting Natural Catastrophe in the Age of Enlightenment*, ed. Alessa Johns (New York: Routledge, 1999), 6.

interpreted this as a bad omen that would be succeeded by a disaster or calamity, in this case a portent of the devastating plague outbreak in Istanbul.[144] The association between comets and outbreaks of plague was a widely maintained one in early modern Ottoman society, as was most famously illustrated in the closing down of the Ottoman observatory in Istanbul. When plague broke out following the appearance of a comet in Istanbul's skies in 1577, the observatory was closed down on grounds that it was inauspicious.[145]

Conclusion

This chapter has offered an overview of the natural history of plague to better understand the Ottoman experience of this disease in the late medieval and early modern eras. It draws from scientific and historical scholarship, with a view to bringing this body of knowledge in dialogue with the evidence found in Ottoman historical sources. Such an effort requires adopting a multilayered outlook, as it seeks to engage with multiple actors and agencies – especially cumbersome in dealing with nonhuman agencies, a direction that the Ottomanist historiography has only recently begun to pursue more thoroughly. Thus, owing to the complex etiological nature of the disease, the chapter surveys the protagonists of Ottoman plagues in separate sections devoted to hosts (rodents in particular, among various species of mammals), vectors (fleas and lice in particular, among other arthropods), the pathogen (*Y. pestis*), the humans, and the environment. Moreover, each of these protagonists is intimately linked to the others; establishing these connections is essential to fully comprehending the complex of plague.

The chapter has presented scientific and historical evidence about the presence of a number of wild and commensal rodent species in the Ottoman domains that may be associated with plague. In particular, it has emphasized the importance of commensal rodents for sustaining epidemics in urban areas. The analysis of historical sources suggests that the Ottomans did not observe direct links between rodents and plague outbreaks, even though they sometimes made indirect associations. In doing so, the Ottomans were not alone; this association was not identified until the end of the nineteenth century. It appears that the Ottoman urban population saw rats and mice as common pests and used rat poison and other means to exterminate them.

Similarly, vectors of plague (fleas and lice) in the historical and scientific sources are presented here in detail. It appears that such ectoparasites were common in Ottoman society, including among the elite, much like

144 Kritovoulos, *History of Mehmed the Conqueror*, 217.

145 For a discussion of the observatory in historical context, see Avner Ben-Zaken, *Cross-Cultural Scientific Exchanges in the Eastern Mediterranean, 1560–1660* (Baltimore: Johns Hopkins University Press, 2010), 8–47.

other contemporary societies. To a certain extent, such pests were culturally acceptable, even though Ottoman urban populations frequently resorted to hygienic practices to rid themselves of the pests, such as removing body hair, bathing, and using aromatic oils, while nomadic populations moved to higher altitudes to that end. Early modern observers evidently noticed fleabites on the bodies of plague victims but did not link these to the disease. The discovery of fleas as vectors of plague had to wait until the close of the nineteenth century. Drawing from sources of the Ottoman experience of plague, the chapter underlined the transitory role played by some animals in carrying infected vectors locally (predators of rodents, e.g., hyenas or weasels) or over long distances (migratory birds, e.g., white stork).

The discussion of the plague pathogen (*Y. pestis*) almost entirely draws from research from non-Ottoman experiences, owing to a lack of bioarcheological data from Ottoman cases of plague. At present, there is no aDNA evidence of *Y. pestis* recovered from former Ottoman areas. Such studies are much awaited for confirming the presence of the pathogen in this area. The only exception is the availability of modern *Y. pestis* isolates from former Ottoman areas (Turkey, northern Iraq, and western Iran) that have been included in recent phylogenetic analyses of the pathogen. However, these are not very helpful for studying late medieval and early modern plagues.

As incidental hosts to the disease, the agency of the human species has been the most important of all. Humans can spread the disease much more rapidly and widely than any of the other protagonists. At the same time, however, it was the human effort that developed means of containing and treating the plague. The myriad forms of human interaction with natural and built environments had an impact on the spread of the disease. It should not come as a surprise that in an era marked by massive efforts toward empire building, such as the era studied here, human mobility should increase both spatially and temporally. How the Ottoman growth in the fifteenth and sixteenth centuries intensified various forms of human and nonhuman plague agents' mobility (warfare and conquest, urbanization, and trade) and how such mobility stimulated the plague in the Ottoman experience are analyzed in greater detail in later chapters.

Finally, this chapter highlighted the part played by environmental factors in shaping the disease. It discussed how the Ottoman plagues may be linked to the broader environment and offered possible ways of studying these connections, drawing from both scientific literature and Ottoman historical sources. The vision that placed epidemics on a larger spectrum of natural (and supernatural) causes, such as earthquakes, weather events, and cosmic influences, was familiar to the Ottomans in this era.