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The End of Plague in Europe

▼ **SPOTLIGHT ARTICLE** in *How Epidemics End*, ed. by Erica Charters

▼ **ABSTRACT** At the Centre for Ecological and Evolutionary Synthesis (CEES, University of Oslo), a group of biologists has been working for decades to disentangle the complex mechanisms of plague epizootics and epidemics in places where extant wild rodent reservoirs are present. These questions have been approached through ecological and climatic studies, mathematic modeling, as well as genomics and epidemiology. In 2013–2018, the Centre hosted the ERC-project MedPlag, which explored past pandemics through the lenses of additional disciplines, like archaeogenomics (ancient DNA), anthropology, archaeology, and evolution, always against the background of historical information. Here, we reflect on the end of plague in Europe based on the most recent studies on plague carried out in these different disciplines.

▼ **KEYWORDS** *Yersinia pestis*, Wildlife Reservoirs, Pandemics

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Plague is an epidemic-prone disease that has caused millions of deaths throughout history.[1] However, plague is primarily a wildlife disease that only occasionally spills over from hosts such as wild or domestic rodents to humans, through vectors like

1 Stenseth et al. (2008).

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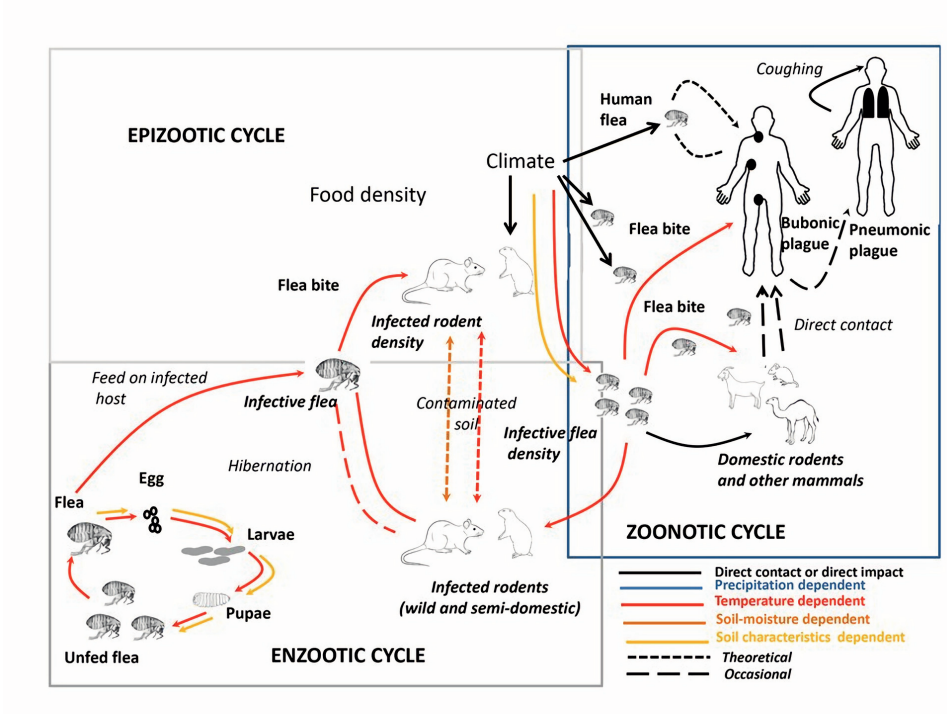

Figure 1. Main mechanisms of plague transmission and how climate variation affects such transitions. Modified from "Plague and Climate: Scales Matter" by M. T. Ben Ari et al., 2011, *PLoS Pathog*, *7*(9), p. 2, CC0; and from "Human Plague: An Old Scourge that Needs New Answers" by X. Vallès et al., 2020, *PLoS Neglected Tropical Diseases*, *14*(8), p. 3, CC-BY-4.0.

fleas or by direct contact (Fig. 1). The ecological conditions for spill-over events are well understood, as is the distribution of plague reservoirs (that is, places where resistant rodent hosts, their associated flea vectors, and the pathogen are present at the same time and constitute a potential source of infection) on all continents besides Europe and Australia.[2] Thus, to understand plague introductions and dynamics in the human environment, we need to understand the plague dynamics in wild rodents. Yet, if we question the reasons for the end of plague epidemics and pandemics, the disappearance of local natural reservoirs might not be the only cause. Although scholars have proposed that historical pandemics in Europe were maintained in a Western European reservoir that caused local outbreaks until it disappeared, we suggest here some alternative reasons for the end of the historical plague pandemics.[3]

The plague bacterium, *Yersinia pestis*, was described (originally under the name *Pasturella pestis*) by Alexandre Yersin in 1894 in Hong Kong, at the onset of the Third

2 Schmid et al. (2015).
3 Carmichael (2014); Spyrou et al. (2016).

Plague Pandemic.[4] Yersin hypothesized that the earlier Second Plague Pandemic (1346–19th century), as well as the First Plague Pandemic (541–7th century), were caused by the same bacterium, a matter that had been a subject of great debate.[5] Today, advances in ancient DNA-sequencing techniques have confirmed that *Y. pestis* was indeed the causative agent of all three pandemics.[6] Further, we now know that plague ravaged Eurasia at least as far back as 5,000 years ago, although it is not yet completely clear how it spread or if it caused large epidemics in prehistoric times.[7]

Genomic studies have shown that the historical pandemics were caused by genetically distinct strains of the plague bacterium.[8] However, in comparison to other highly diverse microbial species like *Escherichia coli*,the *Y. pestis* genome has been relatively stable during the past millennia.[9] Nevertheless, small variants called SNPs (Single Nucleotide Polymorphisms) allow the construction of phylogenetic trees with well-defined branches, corresponding to the three historical pandemics. From the phylogeny, we now know that, towards the end of the first two pandemics, there were two convergent changes in the *Y. pestis* genome (that is, mutations that were concurrently present in the same strains).[10] These convergent mutations involved key genes, and there is a possibility that they attenuated the virulence of the bacteria over the course of the First and Second Pandemics, but not during the prehistoric period or in the most recent strains.[11] Moreover, this attenuation has been interpreted as the cause of the end of the Second Pandemic, although in evolutionary terms, these changes might better indicate a long-lasting adaptation of the pathogen to a new host, which might have been humans, new vectors, or new rodents.[12] With this in mind, we could interpret the two convergent mutations coinciding with two pandemics as bacterial adaptation to the human environment, although this scenario remains inconclusive.[13] Despite valuable attempts to explore the possibility of genomic selection due to plague, it is also unclear if humans developed any form of genetic or humoral protection against this attenuated form of *Y. pestis*, a mechanism that also might explain the end of the first two pandemics.[14] These alternative explanations for the end of the historical plague pandemics in Europe certainly warrant further exploration.

Historians have also offered other explanations for the end of the Second Pandemic in Western Europe. For example, Panzac proposed that the end of plague in the

4 Yersin (1894).
5 Yersin (1894). For example, Cohn (2002); Scott & Duncan (2001); Twigg (1984).
6 Haensch et al. (2010); Bos et al. (2011); Harbeck et al. (2013); Wagner et al. (2014).
7 For example, Rasmussen et al. (2015); Susat et al. (2021); Valtueña et al. (2017). For debate on epidemics in prehistoric times, see Rascovan et al. (2019); Spyrou et al. (2018); Susat et al. (2021).
8 Bos et al. (2016); Feldman et al. (2016); Guellil et al. (2020); Keller et al. (2019); Morozova et al. (2020); Namouchi et al. (2018); Seguin-Orlando et al. (2021); Spyrou et al. (2016; 2019); Susat et al. (2020).
9 Achtman (2012).
10 Guellil et al. (2020); Keller et al. (2019); Spyrou et al. (2019); Susat et al. (2020).
11 Bramanti, Wu, Yang, Cui, & Stenseth (2021).
12 Susat et al. (2020) interpreted the attenuation as the cause of the end of the Second Pandemic, whereas Bramanti et al. (2021) proposed the evolutionary explanation.
13 Bramanti et al. (2021).
14 Immel et al. (2021).

Balkans in the early 1840s coincided with the introduction of a network of maritime and terrestrial quarantine stations, which reduced the spread of diseases between the European provinces of the Ottoman Empire.[15] By the 18th century, quarantine was common in many European port cities, which appears to have reduced the occurrence of major epidemics in Europe, along with increased disease surveillance.[16] Another possible clarification offered by historians for the end of the Second Pandemic is the improvement of housing through the introduction of "rat-resistant brick and stone for urban construction," in addition to the replacement, albeit partial, of the black rat (*Rattus rattus*) by the brown rat (*Rattus norvegicus*) in Europe.[17] The black rat and its fleas were indeed demonstrated to have been responsible for mediating the contact between wild rodents and humans in places where plague reservoirs are present.[18] Yet its involvement in sustaining outbreaks in Europe during the three plague pandemics has been challenged, because, in contrast to what was observed in Asia during the Third Pandemic, no eyewitnesses to the plague in Europe have ever reported epizootic phenomena of the black rat prior to or concomitant with an epidemic, even at the time of the Third Pandemic, although rats were reported to have been present on infected ships, and although they were scrupulously tracked and scrutinized.[19]

Clearly, the first two plague pandemics greatly affected Europe, leaving millions of victims in their wake. By contrast, the impact of the Third Plague Pandemic was much more limited in Europe, yet not absent, allowing for an exploration of its dynamics and end in periods for which we have more detailed information.

At the beginning of the Third Pandemic, plague, like other infectious diseases, became internationally notifiable, a status that it still has today.[20] This action was significant for preventing and monitoring outbreaks of plague worldwide, and the resulting records of notifiable diseases have furnished valuable data for the retrospective study of plague.[21] In Europe, there were 1,692 cases and 457 deaths attributed to plague between 1899 and 1950 recorded in the U.S. Government's *Public Health Reports*.[22] More extensive research into narratives and medical literature has revealed that not all the cases were officially notified. For instance, the last outbreak occurred in Europe—in Taranto, Italy at the end of World War II; it caused 30 cases and 15 deaths, and was only uncovered by dedicated local historians.[23]

In addition to this published dataset, plague cases and outbreaks were reported in the major archipelagos connected to Europe, the Azores and the Canary Islands.[24]

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15 Panzac (1985).
16 Eckert (2000); Guellil et al. (2020); Konstantinidou, Mantadakis, Falagas, Sardi, & Samonis (2009).
17 Appleby (1980, p. 173).
18 See Bramanti, Dean, Walløe, & Stenseth (2019, Section 3a) for relevant literature on this matter.
19 Bramanti et al. (2019).
20 "On the question of notification, instead of being mandatory for the existence of an outbreak, the information to be given to other countries should be for any case of plague." Proust (1897, p. 294).
21 Xu et al. (2019).
22 The dataset is published in Bramanti et al. (2019).
23 Carducci (2001).
24 Bramanti et al. (2019).

Pollitzer accounts for 2,159 cases recorded from 1905 to the 1950s on the Atlantic islands of Madeira and the Azores.[25] The largest epidemic, with 744 cases, occurred on Sao Miguel island in 1922–1923, followed by sporadic cases averaging eight per year in the period 1942–1948.[26] The last two cases were recorded in 1949. Moll and O'Leary recorded an additional 215 cases of plague in the Canary Islands from 1906 to 1935, although the official notification for the first years was only issued in 1924.[27] Reconstructions of the plague's spread showed that the Canaries were involved in the spread of plague to San Juan, Puerto Rico, Habana, Cuba, New Orleans, the USA, and Liverpool, UK in 1912.[28] In the period of the Third Pandemic until the 1950s, almost all the cases notified in Europe and in the major archipelagos around Europe were recorded in ports, fluvial harbors, and islands, suggesting that plague was imported into Europe from outside, mostly by maritime shipping, via infected persons, cloths, goods, or rats.[29] This is not unexpected, since there are no present-day plague reservoirs in Europe, meaning *Y. pestis* can only be imported from outside. Nonetheless, the more limited impact of plague in Europe during the Third Pandemic, in comparison to the previous two, was not necessarily due to the absence of a reservoir.

At the beginning of the Third Plague Pandemic, Europe and the rest of the world were more prepared than before to face the threat of disease introductions. From the first decades of the 19th century, increased globalization spurred by the invention of steamships and steam trains had already spread cholera, smallpox, yellow fever, and flu, requiring the intervention of the international public health system and several international conferences to propose and coordinate preventive measures.[30] The intensive and concerted engagement of international governments, health authorities, and scientists, as accounted by Proust in his extensive report on the International Sanitary Conference of Venice 1897, resulted in the effective control of the pandemic's spread in Europe.[31] Further, the almost complete disappearance

25 Pollitzer (1954; 1951).
26 Pollitzer (1954).
27 Moll & O'Leary (1942).
28 Moll & O'Leary (1942).
29 Bramanti et al. (2019).
30 Bramanti et al. (2019).
31 The public health measures suggested at the beginning of the Third Plague Pandemic by the International Sanitary Conference of Venice on February 16, 1897, which was composed by a technical Commission (of expert physicians) and a Commissions of diplomats, were comprehensively reported by Proust (1897). Here, we include a summary of the proposed rules. Plague foci were to be monitored. The governments of each country should notify the others about cases of plague, including by telegraph, and keep them updated on the measures taken to avoid the propagation of plague. Strict rules were proposed for maritime travel. People departing from a contaminated port should be submitted to rigorous, compulsory, individual medical examinations at the time of boarding. With any suspicion of plague, departure should be restricted. Compulsory and rigorous disinfection of any contaminated or suspect object should be carried out on land, under the supervision of the doctor delegated by the public authority. Dangerous merchandise included clothes, rags, used bags, carpets, used embroidery, untanned hides, fresh hides, fresh animal scraps, hooves, manes, silks and raw wools, and hair. The country of arrival was free to accept or refuse merchandise, or to admit them only after disinfection (which was recommended by the Conference). Disinfection prophylaxis included destruction by fire for valueless objects; steaming for fabric and cloth; washing with disinfectant solutions for equipment, surfaces, and walls of inhabited

of plague from Europe in the 1950s is concomitant with the better hygienic and living conditions in the Western world after World War II, with the introduction of domestic baths, domestic appliances, and insecticides likely reducing the presence of parasites.[32] Still, it remains to be clarified what kind of vectors might have been responsible for plague transmission, although the consideration that plague outbreaks might be supported by parasites infesting humans and their environments is finding growing support in the scientific literature.[33]

While coordinated efforts consistently reduced the number of times plague was introduced to Europe during the Third Pandemic, one large (Oporto in 1899) and several small outbreaks still occurred over a period of about 50 years and were efficiently managed by the relevant local authorities. For example, the local sanitary authorities effectively controlled the spread of plague during an outbreak in

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places; and washing, ventilation, or use of antiseptic gases for parts of these premises. The doctor should also define if the vessels were "plague-free," "suspected," or "infected." The incubation time for plague was formally fixed at a maximum of 10 days, with the consequence that vessels with no cases in the last 12 days of voyage could not be considered "plague-free," but "suspected," thus they did not require disinfection if no rats that had died of plague were found on board. People suffering from plague were notified by the doctor on board, and had to disembark and isolate. Particular attention was paid to pilgrims travelling on vessels; besides the controls before boarding, the captain should ensure continuous controls and correcting measures, as well as sufficient prophylactic disinfection and food and water during the voyage. Particularly detailed instructions were also provided for maritime travel through the Suez Channel, the Red Sea, and the Persian Gulf to avoid the spread of plague into the Mediterranean Sea. For travelers on land, disinfection and controls were to replace quarantine for people and goods along well-travelled routes and railways. Only people with symptoms of plague should be isolated. Border closure should be enacted only in exceptional cases.

If, despite all the precautions, an infected ship arrived in Europe, people sick from plague should disembark and be isolated. The other persons on board should disembark as well and kept isolated or under surveillance for no more than 10 days. Every object should be considered contaminated and disinfected, as should all the locals on board visited by people with plague. The water stores should be replaced with fresh potable water. Before departure, the captain of the ship where plague was found should ensure that no person suspected of being infected had embarked, that the vessel is perfectly clean and disinfected, and that no belongings of the sick people remain on board. People eventually affected during the travel should be isolated, as well as their caregivers. Their belongings, as well as their secretions and body fluids should be collected in vases containing disinfecting solutions and immediately discarded in the toilet, which should be disinfected as well. Cadavers, previously wrapped in a shroud impregnated with a disinfecting solution, should be thrown into the sea. The logbook, containing any appropriate information, should be submitted to the health authority on arrival. On the "suspected" vessel, medical visits and disinfection should be implemented, but the people should remain on board and under surveillance for a period not longer than 10 days. A "plague-free" vessel should be admitted to free practice, whatever the nature of their license. The competent authority of the port of arrival should request a certificate from the physician on board attesting that no cases were present on vessel since departure.

Cases notified in Europe should also report most detailed information about the prophylactic measures used to avoid an epidemic, in particular in relation to health inspection or medical examination, isolation, and disinfection. Places where plague cases were found are considered "contaminated" until 12 days after the recovery or death of the last case, but only to ensure that appropriate disinfection was carried out. The erection of military *cordon sanitaires* and censorship of information concerning the epidemic was not recommended by the Conference of Venice in 1897 (Proust, 1897; Echenberg, 2010; Bramanti et al., 2019).

Although dead rats on vessels were mentioned in the cited opus of Proust (1897), because Ogata had already observed plague in rats, the first Conference aware of the role played by rats in transmitting plague on ships was that held in Paris in 1903. Nonetheless, it was not until after the International Sanitary Convention of 1926 that the Office International d'Hygiène Publique (founded in Paris in 1907) was required to publish "a *Relevé annuel* relating to the deratting of ships and ports" (World Health Organization, 1958).

32 Bramanti et al. (2019).

33 For example, Barbieri, Drancourt, & Raoult (2020); Dean et al. (2018).

Glasgow in 1900.[34] The investigation included rapid identification of cases, contact tracing, and quarantining, as well as sending plague cases to the hospital, preventing large gatherings at wakes, and sanitizing homes and clothing. The authorities also disseminated information about plague to healthcare providers and the public. The actions of the sanitary authorities in Glasgow appear to have stopped the outbreak, which resulted in only 35 cases in the city. Although Glasgow experienced more introductions of plague, local outbreaks could be effectively managed with the epidemiological interventions available to the authorities at that time.[35] The success of these interventions, by reducing contacts with cases, was demonstrated for Glasgow through the reconstruction of the transmission network.[36] At the beginning of the 20th century in Europe, introductions of plague were effectively managed by reducing contact between humans and vectors through better hygienic conditions and social distancing or isolation, ultimately stopping further spread of the contagion.

The timely discovery and notification of plague cases, and the implementation of various control measures like those in Glasgow, meant that further spread of the contagion within Europe was rare. Indeed, in Europe during the Third Pandemic, we could find only a few cases of plague that spread from ports to other regions, all mediated by infected people travelling over land, like the case in Liverpool in 1907 (imported from Glasgow), and a case in Reggio di Calabria imported from Taranto.[37] Effective control measures, along with better hygienic and sanitary conditions, could have all contributed to the end of the Third Pandemic in 1946, at least in Western Europe. Today, the endemicity of plague in some industrialized countries, like the USA and China, can be explained by the presence of local plague reservoirs. With stringent, internationally coordinated controls, only a few cases are notified every year, mostly due to accidental contact with infected rodents, cats, or dogs. Large outbreaks of plague, such as the one in Madagascar in 2017, are very rare indeed, as long as the rapid identification, reporting, and isolation of cases is possible. From the early stages of the Third Pandemic, control measures and interventions benefitted greatly from improved methods of communication, whereas in the modern era hygiene and medicine have had the most effect, together with other technological improvements. All these conditions, which have contributed to the end of the Third Plague Pandemic—at least in some parts of the world—were unimaginable in the previous centuries. Therefore, plague might have had its largest death toll during the First and Second Pandemics, even in places without wild rodent reservoirs, as quarantines, *cordons sanitaire*, and medical treatments were less efficient. A vibrant discussion about the presence of wild rodent reservoirs in ancient Europe is still ongoing, based on different interpretations of the phylogeny of the ancient strains, as well as on historical and ecological data. In order to settle this issue, there is a need for

34 Dean, Krauer, & Schmid (2019).
35 Bramanti et al. (2019).
36 Dean et al. (2019).
37 See Colvin (1907) for Liverpool, and Bramanti et al. (2019) for Reggio di Calabria.

additional environmental data, as well as additional genomes from different parts of Eurasia that can be accurately dated and for which historical information is present.[38]

The U.S. Centers for Disease Control and Prevention defines an epidemic as an often-sudden increase in the number of cases beyond what is expected in a given area. Since plague is primarily a rodent disease, human cases are often regarded as outbreaks, and this is especially true for cases in Europe, where no past or present reservoirs for plague have yet been demonstrated. Building on our previous biological research, we argue that there has never been a plague reservoir in Europe, a condition that can also explain the limited role of the rats in supporting plague epidemics in the past. Plague was frequently reintroduced to Europe via maritime shipping or overland from regions where the infection is endemic due to plague reservoirs. Rats on ships and goods and their fleas may have been responsible for the transportation of plague into Europe, but we argue that within Europe plague circulated among humans, without the needs of rats, as we have seen in Glasgow in 1900. Chains of transmission might have mainly been mediated by contact with contaminated persons or their body fluids, or clothes or rags containing ecto-parasites. Therefore, we suggest that the efforts made to track and record plague cases in Europe during the Third Pandemic made it possible to measure the beginning and end of the epidemic in an epidemiological context. From notifications of plague cases, we can see that the beginning of the Third Pandemic in Europe coincided with the spread of plague to many parts of the world, whereas repeated introductions of the disease led to cases in Europe until the 1950s. The International Sanitary Conferences convened through the 19th century were proven to be very efficient in proposing measures to contain and stop the contagion. Preventing and controlling pandemics is an imperative goal that can be achieved through cooperation between international authorities, with a clear understanding of the mechanisms of infectious disease transmission.

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References

Achtman, M. (2012). Insights from genomic comparisons of genetically monomorphic bacterial pathogens. *Philosophical Transactions of the Royal Society of London, Series B, 367*(1590), 860–867. https://doi.org/10.1098/rstb.2011.0303

Appleby, A. B. (1980). The disappearance of plague: A continuing puzzle. *The Economic History Review, 33*(2), 161–173.

38 Bramanti et al. (2021).

Barbieri, R., Drancourt, M., & Raoult, D. (2020). The role of louse-transmitted diseases in historical plague pandemics. *The Lancet Infectious Diseases*, *21*(2), e17–e25.

Ben Ari, T., Neerinckx, S., Gage, K. L., Kreppel, K., Laudisoit, A., Leirs, H. & Stenseth, N. C. (2011). Plague and climate: Scales matter. *PLoS Pathogens*, *7*(9), e1002160.

Bos, K. I., Herbig, A., Sahl, J., Waglechner, N., Fourment, M., Forrest, S. A. … & Poinar, H. N. (2016). Eighteenth century Yersinia pestis genomes reveal the long-term persistence of an historical plague focus. *ELife*, *5*, e12994. https://doi.org/10.7554/eLife.12994.001

Bos, K. I., Schuenemann, V. J., Golding, G. B., Burbano, H. A., Waglechner, N., Coombes, B. K. … & Krause, J. (2011). A draft genome of Yersinia pestis from victims of the Black Death. *Nature*, *478*(7370), 506–510. https://doi.org/10.1038/nature10549

Bramanti, B., Dean, K. R., Walløe, L., & Stenseth, N. C. (2019). The Third Plague Pandemic in Europe. *Proceedings of the Royal Society B*, *286*(1901), 20182429. https://doi.org/10.1098/rspb.2018.2429

Bramanti, B., Wu, Y., Yang, R., Cui, Y., & Stenseth, N. C. (2021). Assessing the origins of the European plagues following the Black Death: A synthesis of genomic, historical, and ecological information. *Proceedings of the National Academy of Sciences*, *118*(36), e2101940118. https://doi.org/10.1073/pnas.2101940118

Carducci, A. (2001). L'ultima peste in Europa: Taranto 1945. In M. L. Distante & A. E. Portulano-Scoditti (Eds.), *Atti del XLI Congresso Nazionale della Società Italiana di Storia della Medicina* (pp. 163–176). Mesagne, Italy: Giordano Editore.

Carmichael, A. G. (2014). Plague persistence in western Europe: A hypothesis. *The Medieval Globe*, *1*(1), 8.

Cohn, S. (2002). *The Black Death transformed: Disease and culture in early Renaissance Europe*. London, UK: Arnold.

Colvin, T. (1907). Is bubonic plague still lurking in the city of Glasgow? *The Lancet*, *170*(4396), 1522–1523.

Dean, K. R. , Krauer, F., & Schmid, B. V. (2019). Epidemiology of a bubonic plague outbreak in Glasgow, Scotland in 1900. *Royal Society Open Science*, *6*(1), 181695.

Dean, K. R., Krauer, F., Walløe, L., Lingjærde, O. C., Bramanti, B., Stenseth, N. C., & Schmid, B. V. (2018). Human ectoparasites and the spread of plague in Europe during the Second Pandemic. *Proceedings of the National Academy of Sciences of the United States of America*, *115*(6). https://doi.org/10.1073/pnas.1715640115

Echenberg, M. (2010). *Plague ports: The global urban impact of bubonic plague, 1894–1901*. New York, NY: NYU Press.

Eckert, E. A. (2000). The retreat of plague from central Europe, 1640–1720: A geomedical approach. *Bulletin of the History of Medicine*, *74*(1), 1–28.

Feldman, M., Harbeck, M., Keller, M., Spyrou, M. A., Rott, A., Trautmann, B. & Krause, J. (2016). A high-coverage Yersinia pestis genome from a sixth-century Justinianic plague victim. *Molecular Biology and Evolution*, *33*(11), 2911–2923. https://doi.org/10.1093/molbev/msw170

Guellil, M., Kersten, O., Namouchi, A., Luciani, S., Marota, I., Arcini, C. A., Bakanidze, L. … & Bramanti, B. (2020). A genomic and historical synthesis of plague in 18th century Eurasia. *Proceedings of the National Academy of Sciences*, *177*(45), 28328–28335. https://doi.org/10.1073/pnas.2009677117

Haensch, S., Bianucci, R., Signoli, M., Rajerison, M., Schultz, M., Kacki, S. … & Bramanti, B. (2010). Distinct clones of Yersinia pestis caused the Black Death. *PLoS Pathogens*, *6*(10). https://doi.org/10.1371/journal.ppat.1001134

Harbeck, M., Seifert, L., Hänsch, S., Wagner, D. M., Birdsell, D., Parise, K. L. … & Scholz, H. C. (2013). Yersinia pestis DNA from skeletal remains from the 6th Century AD reveals insights into Justinianic plague. *PLoS Pathogens*, *9*(5). https://doi.org/10.1371/journal.ppat.1003349

Immel, A., Key, F. M., Szolek, A., Barquera, R., Robinson, M. K., Harrison, G. F. … & Krause, J. (2021). Analysis of genomic DNA from medieval plague victims suggests long-term effect of *Yersinia pestis* on human immunity genes. Molecular Biology and Evolution, *38*(10), 4059–4076. https://doi.org/10.1093/molbev/msab147

Keller, M., Spyrou, M. A., Scheib, C. L., Neumann, G. U., Kröpelin, A., Haas-Gebhard, B. … & Krause, J. (2019). Ancient Yersinia pestis genomes from across Western Europe reveal early diversification during the First Pandemic (541–750). *Proceedings of the National Academy of Sciences*, *116*(25), 12363–12372.

Konstantinidou, K., Mantadakis, E., Falagas, M. E., Sardi, T., & Samonis, G. (2009). Venetian rule and control of plague epidemics on the Ionian Islands during 17th and 18th centuries. *Emerging Infectious Diseases*, *15*(1), 39.

Moll, A. A., & O'Leary, S. (1942). Plague in the Americas: The West Indies and certain European-African islands. *Boletín de la Oficina Sanitaria Panamericana*, *21*(10), 980–1000.

Morozova, I., Kasianov, A., Bruskin, S., Neukamm, J., Molak, M., Batieva, E. … & Schuenemann, V. J. (2020). New ancient Eastern European Yersinia pestis genomes illuminate the dispersal of plague in Europe. *Philosophical Transactions of the Royal Society B*, *375*(1812), 20190569.

Namouchi, A., Guellil, M., Kersten, O., Hänsch, S., Ottoni, C., Schmid, B.V. … & Bramanti, B. (2018). Integrative approach using Yersinia pestis genomes to revisit the historical landscape of plague during the medieval period. *Proceedings of the National Academy of Sciences*, *115*(50), E11790–E11797.

Panzac, D . (1985). *La peste dans l'Empire Ottoman : 1700–1850*. Leuven, Belgium: Peeters.

Pollitzer, R . (1951). Plague studies I: A summary of the history and a survey of the present distribution of the disease. *Bulletin of the World Health Organization*, *4*(4), 475.

Pollitzer, R. (1954). *Plague*. Geneva, Switzerland: World Health Organization.

Proust, A. (1897). *La défense de l'Europe contre la peste et la Conférence de Venise de 1897*. Paris, France: Masson. Retrieved from http://books.google.com/books?id=mmuFA-8TxpUC&oe=UTF-8

Rascovan, N., Sjögren, K.-G., Kristiansen, K., Nielsen, R., Willerslev, E., Desnues, C., & Rasmussen, S. (2019). Emergence and spread of basal lineages of Yersinia pestis during the Neolithic decline. *Cell*, *176*(1–2), 295–305.

Rasmussen, S., Allentoft, M., Nielsen, K., Orlando, L., Sikora, M., Sjögren, K.-G. … & Willerslev, P. (2015). Early divergent strains of Yersinia pestis in Eurasia 5,000 years ago. *Cell*, *163*(3), 571–582.

Schmid, B. V., Büntgen, U., Easterday, W. R., Ginzler, C., Walløe, L., Bramanti, B., & Stenseth, E. C. (2015). Climate-driven introduction of the Black Death and successive plague reintroductions into Europe. *Proceedings of the National Academy of Sciences*, *112*(10). https://doi.org/10.1073/pnas.1412887112

Scott, S., & Duncan, C. J. (2001). *Biology of plagues: Evidence from historical populations*. Cambridge, UK: Cambridge University Press.

Seguin-Orlando, A., Costedoat, C., der Sarkissian, C., Tzortzis, S., Kamel, C., Telmon, N. … & Orlando, L. (2021). No particular genomic features underpin the dramatic economic consequences of 17th century plague epidemics in Italy. *Iscience*, *24*(4), 102383.

Spyrou, M. A., Keller, M., Tukhbatova, R. I., Scheib, C. L., Nelson, E. A., Valtueña, A. A. … & Krause, J. (2019). Phylogeography of the second plague pandemic revealed through analysis of historical Yersinia pestis genomes. *Nature Communications*, *10*(1), 1–13.

Spyrou, M. A., Tukhbatova, R. I., Feldman, M., Drath, J., Kacki, S., Beltrán de Heredia, J., … & Krause, J. (2016). Historical Y. pestis genomes reveal the European Black Death as the source of ancient and modern plague pandemics. *Cell Host and Microbe*, *19*(6), 874–881. https://doi.org/10.1016/j.chom.2016.05.012

Spyrou, M. A., Tukhbatova, R. I., Wang, C.-C., Valtueña, A. A., Lankapalli, A. K., Kondrashin, V. V. & Krause, J.(2018). Analysis of 3800-year-old Yersinia pestis genomes suggests Bronze Age origin for bubonic plague. *Nature Communications*, *9*(1), 2234.

Stenseth, N. C., Atshabar, B. B., Begon, M., Belmain, S. R., Bertherat, E., Carniel, E. … & Rahalison, L. (2008). Plague: Past, present, and future. *PLOS Medicine*, *5*(1), e3. https://doi.org/10.1371/journal.pmed.0050003

Susat, J., Bonczarowska, J. H., Pētersone-Gordina, E., Immel, A., Nebel, A., Gerhards, G., & Krause-Kyora, B. (2020). Yersinia pestis strains from Latvia show depletion of the pla virulence gene at the end of the second plague pandemic. *Scientific Reports*, *10*(1), 1–10.

Susat, J., Lübke, H., Immel, A., Brinker, U., Macāne, A., Meadows, J. … & Krause-Kyora, B. (2021). A 5,000-year-old hunter-gatherer already plagued by Yersinia pestis. *Cell Reports*, *35*(13), 109278. https://doi.org/10.1016/j.celrep.2021.109278

Twigg, G. (1984). *The Black Death: A biological reappraisal*. New York, NY: Schocken.

Vallès, X., Stenseth, N. C., Demeure, C., Horby, P., Mead, P. S., Cabanillas, O. … & Baril, L. (2020). Human plague: An old scourge that needs new answers. *PLoS Neglected Tropical Diseases*, *14*(8), e0008251.

Valtueña, A. A., Mittnik, A., Key, F. M., Haak, W., Allmäe, R., Belinskij, A. … & Krause, J. (2017). The Stone Age plague and its persistence in Eurasia. *Current Biology*, *27*(23), 3683–3691.e8. https://doi.org/10.1016/J.CUB.2017.10.025

Wagner, D. M., Klunk, J., Harbeck, M., Devault, A., Waglechner, N., Sahl, J. W. … & Poinar, H. (2014). Yersinia pestis and the plague of Justinian 541–543 AD: A genomic analysis. *The Lancet Infectious Diseases*, *14*(4), 319–326. https://doi.org/10.1016/S1473–3099(13)70323–2

World Health Organization . (1958). *The first ten years of the World Health Organization*. Geneva, Switzerland: World Health Organization.

Xu, L., Stige, L. C., Leirs, H., Neerinckx, S., Gage, K. L., Yang, R. … & Zhang, Z. (2019). Historical and genomic data reveal the influencing factors on global transmission velocity of plague during the Third Pandemic. *Proceedings of the National Academy of Sciences, 116*(24). https://doi.org/10.1073/pnas.1901366116

Yersin, A. (1894). La peste bubonique à Hong-Kong. *Annales de l'Institut Pasteur, 2*, 428–430.