



A depiction of the Mongol siege of Girdkuh, an Ismaili stronghold in what is present-day Iran. Hayton of Corycus, *Fleur des histoires de la terre d'Orient*, ca. 1301–1400, parchment, 55 leaves with 2 columns, 300 x 210 x 35 mm, half-bound, 21v, Bibliothèque nationale de France, Département des Manuscrits, NAF 886.

The Four Black Deaths

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THE BLACK DEATH IS USUALLY DEFINED as the massive plague pandemic that struck the Black Sea, the Mediterranean, and adjacent land masses between 1346 and 1353, killing as much as half the total population.¹ It has been called the largest pandemic in human history. This

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¹ The standard account of the Black Death in Europe is Ole J. Benedictow, *The Black Death, 1346–1353: The Complete History* (Woodbridge, 2004). For the Middle East, see Michael W. Dols, *The Black Death in the Middle East* (Princeton, N.J., 1977). Although written prior to the new genetics synthesis, both of these remain valuable overviews. To them should now be added the opening chapters of Nükhet Varlık, *Plague and Empire in the Early Modern Mediterranean World: The Ottoman Experience, 1347–1600* (Cambridge, 2015). On the original and evolving meanings of the term “the Black Death,” see Nükhet Varlık, “Why Is Black Death Black? European Gothic Imaginaries of ‘Oriental’ Plague,” in Christos Lynteris, ed., *Plague Image and Imagination* (London, forthcoming). Historiography of plague in the Golden Horde can be found in T. F. Hajdarov [Khaydarov], “Istoriografija jepidemii ‘chernoj smerti’ na territorii Ulusa Dzhuchi (1814–2016)” [“Historiography of the Epidemic of ‘Black Death’ on the Territory of Ulus Juchi (1814–2016)”], *Zolotoordynskoe obozrenie* [*Golden Horde Review*] 5, no. 1 (2017): 164–192; on the Islamic world in Miri Shefer-Mossensohn, “A Historiography of Epidemics in the Islamic Mediterranean,” in Nükhet Varlık, ed., *Plague and Contagion in the Islamic Mediterranean* (Kalamazoo, Mich., 2017), 3–26. On the significance of the new genetics of plague, see Lester K. Little, “Plague Historians in Lab Coats,” *Past and Present*, no. 213 (November 2011): 267–290; J. L. Bolton, “Looking for *Yersinia pestis*: Scientists, Historians, and the Black Death,” in Linda Clark and Carole Rawcliffe, eds., *The Fifteenth Century XII: Society in an Age of Plague* (2013), 15–38; Monica H. Green, “Taking ‘Pandemic’ Seriously: Making the Black Death Global,” in *Pandemic Disease in the Medieval World: Rethinking the Black Death*, inaugural issue of *The Medieval Globe* 1, no. 1–2 (Fall 2014): 27–61; George Dameron, “Identificazione di un killer: Recenti scoperte scientifiche e storiche sulla natura della peste nera,” in Francesco Ciabattani, Elsa Filosa, and Kristina Marie Olson, eds., *Boccaccio, 1313–2013* (Ravenna, 2015), 57–70; and Pierre Toubert, “La Peste Noire (1348), entre histoire et biologie moléculaire,” *Journal des Savants*, no. 1 (2016), 17–31. In an important essay in 2019, Naomi Standen argued for the importance of avoiding modern nation-state terminology in referring to premodern global history.

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essay argues that it was even larger than previously imagined. An element of Black Death historiography connects the outbreak that started in 1346 in the Ulus of Jochi (the Golden Horde, which occupied lands north of the Caspian and Black Seas) to events farther east: as far as the Inner Asian Mountain Corridor (and specifically around Lake Issyk Kul, in modern-day Kyrgyzstan) according to some accounts, or as far as eastern China according to others.² Surprisingly, the plague events of either central or eastern Asia are dated to as little as a decade or so before the events several thousand kilometers away, in the Caucasus and Crimea. The implausibility of rapid long-distance movement by a disease incapable of sustained human transmission has aroused skepticism about the scenario of a single, westward-moving, late medieval, pan-Eurasian pandemic.³ Whether locating the origin of the Black Death in the Caucasus, the Inner Asian Mountain Corridor, China, or Tibet, these theories make inferences about the Mongol Empire, even though the historiography of that 150-year entity yields almost no information on plague at all.⁴

Until the late twentieth century, the various theories of the Black Death's origins and geography were all based on documentary or epigraphic evidence. However, the history of infectious diseases, and particularly the history of plague, has entered a new era. Since the 1980s, the field of paleogenetics (the study of fossil or "ancient" DNA [aDNA]) has developed (see Table 1, below, for the genetics vocabulary used in the

Naomi Standen, "Colouring Outside the Lines: Methods for a Global History of Eastern Eurasia, 600–1350," *Transactions of the Royal Historical Society*, 6th ser., 29 (2019): 27–63. In principle, I am sympathetic to these arguments. However, because I am attempting to make visible a biological history of Eurasia, in which national boundaries have no meaning whatsoever, I have retained modern geographic terminology to help orient the reader.

² For example, Wu Lien-teh (Liande), "The Original Home of Plague," in A. L. Hoops and J. W. Scharff, eds., *Far Eastern Association of Tropical Medicine, Transactions of the Fifth Biennial Congress Held at Singapore, 1923* (London, 1924), 286–304. William H. McNeill, *Plagues and Peoples* (Garden City, N.Y., 1976), posited an origin in the second half of the thirteenth century, when the Mongols advanced into southern China, but believed that plague remained in China until the outbreaks of the 1330s. Independently of McNeill, John Norris and Michael Dols famously debated in the late 1970s whether the Black Death (as conventionally defined) had come from central or eastern Asia (as Dols believed) or out of the Caucasus (as Norris believed). See John Norris, "East or West? The Geographic Origin of the Black Death," *Bulletin of the History of Medicine* 51, no. 1 (1977): 1–24; Michael W. Dols, "Geographic Origin of the Black Death: Comment," *Bulletin of the History of Medicine* 52, no. 1 (1978): 112–113; and Norris's reply, 114–120. I argue here that both Norris and Dols were right on the geography, but unaware of the proper timeframe in which these biological events should be placed. The case of Issyk Kul is discussed below.

³ The most recent extended case for a quick duration, pan-Eurasian transmission was made by Bruce Campbell, *The Great Transition: Climate, Disease and Society in the Late Medieval World* (Cambridge, 2016). I examined the problems with his compressed timeframe in Monica H. Green, "Black as Death" [essay review of Bruce Campbell, *The Great Transition* (2016)], *Inference: International Review of Science* 4, no. 1 (June 2018), <http://inference-review.com/article/black-as-death>.

⁴ Mongol studies has enjoyed a renaissance in the past few decades, so the avoidance of discussion of the Black Death seems deliberate. See, for example, these leading surveys, which never mention plague or the Black Death, or do so just once only to drop the subject immediately: Michal Biran, *Chinggis Khan, Makers of the Muslim World* (Oxford, 2007); Hodong Kim, "The Unity of the Mongol Empire and Continental Exchanges over Eurasia," *Journal of Central Eurasian Studies* 1 (December 2009): 15–42; Michal Biran, "The Mongol Empire in World History: The State of the Field," *History Compass* 11, no. 11 (2013): 1021–1033; Michal Biran, *Qaidu and the Rise of the Independent Mongol State in Central Asia* (New York, 2013); Michal Biran, "The Mongol Empire and Inter-Civilizational Exchange," in Benjamin Z. Kedar and Merry E. Wiesner-Hanks, eds., *The Cambridge World History*, vol. 5: *Expanding Webs of Exchange and Conflict, 500 CE–1500 CE* (Cambridge, 2015), 534–558; Marie Favereau, "The Mongol Peace and Global Medieval Eurasia," *Comparativ: Zeitschrift für Globalgeschichte und vergleichende Gesellschaftsforschung* 28, no. 4 (2018): 49–70. A notable exception has been Uli Schamiloğlu; see "The Impact of the Black Death on the Golden Horde: Politics, Economy, Society, and Civilization," *Zolotoordinskoe obozrenie [Golden Horde Review]* 5, no. 2 (2017): 325–343.

present essay). Paleogenetics can retrieve the aDNA of any (formerly) living organism; to date, the field has focused on humans, domesticated animals (such as dogs and horses), and human pathogens. Focus on *Yersinia pestis*, the bacterium long assumed to be the cause of both the First and Second Plague Pandemics (that is, the Justinianic Plague in the sixth century and the Black Death in the fourteenth) began in 1998.⁵ After several contentious years of debate and experimentation with methods and protocols, by 2011, aDNA work had established definitively that *Yersinia pestis* was the causal agent of the Black Death in Europe.⁶ But genetic analyses did more than simply confirm the pathogen involved. Phylogenetics (the study of the organism's evolution) yielded information that had not previously been suspected from historical records. It established that *Y. pestis* suddenly diverged in the later Middle Ages—shortly before the Black Death—into four new branches, a polytomy geneticists have called the “Big Bang” (see Figure 1, below).⁷ All four lineages have living descendants. Some strains are found only in isolated pockets of eastern and central Eurasia; one spread to East Africa, perhaps at the end of the Middle Ages; others, in modern times, have spread around the world. Only western Europe, where plague seems to have died out by the eighteenth century, has no living descendants of the late medieval Big Bang.

It is ironic, then, that we know so much about the experience of plague in late medieval western Europe and the Middle East, both of which would have been affected by Branch 1 of the new *Y. pestis* lineages, and almost nothing about plague's effects on other parts of Eurasia, where Branches 2–4 spread. By drawing on both paleogenetic and phylogenetic data, as well as the documentary historical record, it is possible not simply to resolve nagging inconsistencies in our historical narratives about the Black Death's origins, but also to pinpoint the likely location, timing, and cultural and environmental circumstances of the late medieval polytomy and the events that may have led to parallel, not sequential, pandemic outbreaks in China, central Eurasia, and the Black Sea/Mediterranean. Together, the documentary and genetic records support the idea that there were four Black Deaths: four explosive proliferations of *Yersinia pestis* into new environments. This epidemiological process started in the thirteenth century, not the fourteenth, as our standard Black Death narratives have suggested. The combined evidence from the biological and documentary archives calls for a reassessment of human events in late medieval Eurasia, including several that have never before been tied to plague.

LIKE ALL BACTERIA, *YERSINIA PESTIS*, the pathogen that causes plague, is a single-celled organism. It cannot be seen with the naked eye, and its existence was not posited until it

⁵ The concept of three pandemics of plague has been confirmed by phylogenetics, which shows that the prolonged plague disseminations usually identified from highly visible human outbreaks—that is, the Justinianic Plague (sixth to eighth century C.E.), the Black Death (discussed here), and the global Third Pandemic (late nineteenth to mid-twentieth century)—are indeed biologically distinguishable proliferations of different lineages of *Yersinia pestis*. A definition of the Second Plague Pandemic grounded on genetics would place its origin circa 1200, as argued here; technically, it has no end date, since lineages from the Big Bang (see below) still survive.

⁶ On the history of these developments, see Michael McCormick, “Toward a Molecular History of the Justinianic Pandemic,” in Lester K. Little, ed., *Plague and the End of Antiquity: The Pandemic of 541–750* (Cambridge, 2007), 290–312; Little, “Plague Historians”; and Monica H. Green, “Plague (*Yersinia pestis*),” *Encyclopedia of the History of Science* (Pittsburgh, forthcoming).

⁷ The Big Bang is discussed in detail below.

was discovered microbiologically at the end of the nineteenth century, thanks to the development of high-powered microscopy, staining techniques, and a laboratory infrastructure that supported such research as a public good. With the new techniques of paleogenetics, *Y. pestis*'s effects on human populations can currently be traced back nearly six thousand years; the organism itself (a clone of a relatively innocuous environmental pathogen, *Yersinia pseudotuberculosis*) may be only a few thousand years older than that.⁸ For most of *Y. pestis*'s history, humans have been unaware of its existence. But the effects of an infectious disease, especially one of such extreme virulence as plague, would have been unmistakable if circulating in human populations.⁹ Reckoning with invisible historical actors is a challenge unique to historians who research the pre-laboratory age. But the implications of *not* reckoning with such a powerful force as plague are considerable, as has recently been suggested by the Africanist archaeologist and historian Gérard Chouin:

Let us imagine, just for a moment, that the [late medieval and early modern] Second Plague Pandemic had as many overwhelming consequences in Sub-Saharan Africa as it had in Europe or across the Islamic world. Let us further imagine that the pandemic drastically affected the demography of the African continent, with unrecorded repercussions on land uses, urban landscapes, political economies, industrial and craft production, labour systems, and religious realms. If it did so, we have until now misread key historical events and ignored fundamental transformative processes.¹⁰

A similar point was made by sinologist Robert Hymes, who proposed in 2014 that late medieval China may have been struck by plague as well, though there were no Boccaccios or Ibn Khalduns in its rich documentary tradition to evocatively chronicle plague's effects.¹¹ The commonality is this: if we did not have all the *written* documentation for the trajectory, timing, symptoms, and effects of plague in late medieval and early mod-

⁸ On the history of the genetics research on plague that has allowed this new history to be constructed, see Green, "Plague (*Yersinia pestis*)."

⁹ An up-to-date overview on the science of *Yersinia pestis* can be found in Christian E. Demeure, Olivier Dussurget, Guillem Mas Fiol, Anne-Sophie Le Guern, Cyril Savin, and Javier Pizarro-Cerdá, "Yersinia pestis and Plague: An Updated View on Evolution, Virulence Determinants, Immune Subversion, Vaccination, and Diagnostics," *Genes and Immunity* 20, no. 5 (2019): 357–370; also published in *Microbes and Infection* 21, no. 5–6 (June–July 2019): 202–212. On plague ecology generally, see Ruifu Yang and Andrey Anisimov, eds., *Yersinia pestis: Retrospective and Perspective* (Berlin, 2016). Absent prompt antibiotic therapy (available only since the 1940s), plague is one of the most lethal infectious diseases in the world. Plague's clinical manifestations and case fatality rates (CFR) depend on the route of entry into the body. Bubonic plague enters the body via the lymphatic system (from an arthropod bite) and has a CFR of about 50 percent; pneumonic plague enters the body via the lungs (from bacteria coughed out by another victim of the disease) and has a CFR of close to 100 percent; septicemic plague enters the bloodstream directly (via a cut or animal bite, for example), and likewise has a CFR of close to 100 percent. Victims can also be infected by eating the meat of an infected animal. On the latter, see, for example, T. Leslie, C. A. Whitehouse, S. Yingst, C. Baldwin, F. Kakar, J. Mofleh, et al., "Outbreak of Gastroenteritis Caused by *Yersinia pestis* in Afghanistan," *Epidemiology and Infection* 139, no. 5 (2011): 728–735.

¹⁰ Gérard Chouin, "Reflections on Plague in African History (14th–19th c.)," *Afriques* 9 (2018), <http://journals.openedition.org/afriques/2228>. Because the disciplinary perspective of different contributors to plague history matters to the arguments presented here, I will introduce key figures by their principal disciplinary identity as well as their name.

¹¹ Robert Hymes, "A Hypothesis on the East Asian Beginnings of the *Yersinia pestis* Polytoymy," *The Medieval Globe* 1, no. 1–2 (2014): 285–308. Hymes's essay was part of the inaugural issue of the journal, on the theme "Pandemic Disease in the Medieval World: Rethinking the Black Death," ed. Monica H. Green and Carol Symes. Reissued as a book under the same title: Monica H. Green, ed., *Pandemic Disease in the Medieval World: Rethinking the Black Death* (Kalamazoo, Mich., 2015).

ern Europe, we would have had no knowledge of its devastating impact before paleogenetic methods (aDNA) began the laborious process in 1998 of documenting the presence of *Yersinia pestis*.¹² Indeed, European historiography was hamstrung between the 1970s and the early twenty-first century by a generation of “plague-denying” arguments that questioned whether the European Black Death, or subsequent incidents of “pestilence,” were in fact caused by the disease we now associate with the bacterium *Yersinia pestis*.¹³

Those debates are not completely over, of course, since aDNA is still an expensive and only sporadically successful technique; it is not possible, even when archaeological remains are recoverable, to document each and every “plague” outbreak paleogenetically. Nevertheless, because plague is an environmental disease, involving many more organisms and pathways than human bodies alone, it is possible to look for plague landscapes: places or populations that plague seems to have scarred in its characteristic ways. Chouin and colleagues have now laid out extensive evidence, mostly from archaeology, of what seems to be widespread population contraction and urban abandonment in late fourteenth- and fifteenth-century West Africa.¹⁴ Hymes, for his part, has done a close re-reading of chronicles and medical writings from China to suggest the possible impact that plague exerted there in the thirteenth century, pushing back by more than a century the beginning of the Black Death event. Marie-Laure Derat has done the same for Ethiopia, including religious writings in her research.¹⁵ At the very least, for both West and East Africa and for China, there are now questions on the table about major disease events, hitherto undocumented in our histories.

The most famous depiction of the Black Death as a trans-Eurasian event came from world historian William McNeill, whose 1976 *Plagues and Peoples* placed the origin of the late medieval pandemic in the Himalayas, and proposed that a sudden sweep of the continent followed, from east to west, in the fourteenth century.¹⁶ McNeill’s proposals were met with skepticism by historians of China; historians of the Mongol world have largely ignored his views. And indeed, current scientific studies of plague’s history offer no support for McNeill’s thesis, at least insofar as he posited a southeast Asian origin for the pandemic. As recently as 2017, a major study on the Islamic portion of the Mongol world expressed skepticism that the origins of the Black Death (and

¹² Plague kills too quickly to leave any perceptible trace on skeletal remains. Hence, the traditional methods of paleopathology (assessment of disease conditions via ocular analysis of physical remains) are of no help in documenting plague’s history. Lester K. Little summarizes the early development of the new field of molecular genetics research, and its internal controversies, in “Plague Historians.” A summary of developments since can be found in: Maria A. Spyrou, Kirsten I. Bos, Alexander Herbig, and Johannes Krause, “Ancient Pathogen Genomics as an Emerging Tool for Infectious Disease Research,” *Nature Reviews Genetics* 20, no. 6 (2019): 323–340. The pace of work in this field has increased exponentially. Throughput (the computer processing of data) has increased 100 million-fold in speed over the past fifteen years, with attendant reductions in cost.

¹³ The present journal published an essay in 2002 claiming that plague studies had reached the end of its paradigm. Unbeknown to that author, it had—but not for the reasons he ascribed. Samuel K. Cohn Jr., “The Black Death: End of a Paradigm,” *American Historical Review* 107, no. 3 (2002): 703–738. For the developments in paleogenetics that began in 1998, see previous note.

¹⁴ Chouin, “Reflections on Plague”; Daphne E. Gallagher and Stephen A. Dueppen, “Recognizing Plague Epidemics in the Archaeological Record of Burkina Faso and Mali, West Africa,” *Afriques* 9, <https://journals.openedition.org/afriques/2198>.

¹⁵ Marie-Laure Derat, “Du lexique aux talismans: Occurrences de la peste dans la Corne de l’Afrique du XIII^e au XV^e siècle,” *Afriques* 9 (2018), <http://journals.openedition.org/afriques/2090>.

¹⁶ McNeill, *Plagues and Peoples*. For corrections of his list of Chinese epidemics, see Hymes, “A Hypothesis on the East Asian Beginnings of the *Yersinia pestis* Polytoomy”; and Timothy Brook, *Great State: China and the World* (London, 2019).

the Mongols' role in it) could ever be resolved. That study, by Peter Jackson, rehearsed arguments put forward in Anglophone historiography, suggesting a "broad consensus" that the disease originated in the steppe but that the political or economic conditions created by the Mongols played no demonstrable role in its spread. At most, the Mongols probably contributed only in dispersing captives and refugees into inhospitable conditions, thereby lowering their resistance to disease.¹⁷ Given the poverty of sources for the Chaghadaid (central Asian) and Jochid (western Asian) Khanates from this period, Jackson concluded that "further progress on this question is unlikely."¹⁸

Recent work dispels even the one seemingly concrete staple of Mongol historiography that tied them to the Black Death. For nearly two centuries, a story documented by only one fourteenth-century non-eyewitness (and repeated in a slightly altered form by another) claimed that the Mongols of the Golden Horde (Ulus of Jochi) had catapulted plague-ridden bodies over the walls of Caffa during a siege, infecting the Italian merchants resident there. When the merchants fled home, they took the disease with them. Hannah Barker, through a deft use of old-fashioned diplomatic history, suggests that the sieges of Caffa (there were two, one in 1344 and another in 1345–1346) were unrelated to the dispersal of plague into the Black and Mediterranean Seas, which could not have started before 1347. Rather than being carried by catapulted bodies at Caffa, the disease was almost certainly transmitted across the Black Sea in 1347 by contaminated grain shipments coming out of Tana, a port farther north on the Sea of Azov.¹⁹ The Mongols, it would seem, have been vindicated of their last remaining causal association with the Black Death: they were hapless victims of the disease, just as were the Italians.

The final remaining datum linking the Mongols to a pan-Eurasian narrative of fourteenth-century plague comes not from a chronicle, but from epigraphic evidence. A gravesite near the lake Issyk Kul in modern Kyrgyzstan was excavated in the late nineteenth century. Though the site had already been disturbed by local farmers, archaeologists were able to retrieve a number of headstones, several of them dated and a handful indicating the cause of death: plague. When the plague ecologist for the World Health Organization, Robert Pollitzer, stumbled on the archaeological reports in 1951, he proposed the 1338–1339 Issyk Kul outbreak as the origin of the Black Death. It has figured in Black Death narratives ever since, most recently in a demographic study affirming that the pattern of mortality was "plague-like," both in the number of sudden deaths and in the distribution of mortality across age groups. Specifically, this study tied the outbreak to the environmental disruptions caused by the Mongols in the early fourteenth century.²⁰ Yet there is no *prima facie* evidence to link that particular outbreak in

¹⁷ Peter Jackson, *The Mongols and the Islamic World: From Conquest to Conversion* (New Haven, 2017), 408. Jackson's discussion is placed under the header "The Integration of Eurasia within a Single Disease Zone: The Black Death," begging the question.

¹⁸ Jackson, *The Mongols and the Islamic World*, 408: "Given the poverty of source material for the Jochid and the Chaghadaid territories alike, further progress on this question is unlikely. One wonders, in any case, how far the idea that the Mongols were responsible for the genesis and spread of the Black Death drew its appeal from a readiness to credit the conquerors with yet another category of deaths on an industrial scale."

¹⁹ Hannah Barker, "Laying the Corpses to Rest: Grain, Embargoes, and *Yersinia pestis* in the Black Sea, 1346–1348," BodoArXiv, May 2, 2020, doi:10.34055/osf.io/rqn8h (forthcoming in *Speculum*, 2021).

²⁰ The most thorough account of the Issyk Kul excavations is T. W. Thacker, "A Nestorian Gravestone from Central Asia in the Gulbenkian Museum, Durham University," *Durham University Journal* 59 (1967): 94–107. Robert Pollitzer, "Plague Studies 1: A Summary of the History and a Survey of the Present Distribution of the Disease," *Bulletin de l'Organisation mondiale de Santé* [*Bulletin of the World*

the late 1330s to the plague outbreak in the Golden Horde, three thousand kilometers away, just eight years later. After all, as we know now, Issyk Kul sits next to what has almost certainly been a plague reservoir for centuries. Why should that plague reservoir have been so uniquely active (and only in a westerly direction) in this particular period?

I contend that reservoir *was* uniquely active, and that it seeded new plague reservoirs elsewhere. But it did so starting in the thirteenth century, not the fourteenth, and it did so in a centrifugal pattern. The most important contribution of genetics is not that it verifies the pathogen (though it does that definitively), but that in so doing, it brings *Yersinia pestis*'s history up to the present day, to be scrutinized by the tools of modern science. Importantly, it also shifts the history of plague away from a litany of human outbreaks—or rather, *recorded* human outbreaks—as its measure, and follows the biological record of the bacterium instead, as it can now be reconstructed through both aDNA and the results of field studies of modern plague in the wild. As a single-celled organism that replicates, under ideal conditions, about a dozen times a day, *Y. pestis* needs to be understood from an ecological perspective: its modes of transmission, its rates of change. Most of those factors can never be known precisely for historical outbreaks that occurred hundreds of years ago. But they do suggest certain biological parameters that we, as historians of the human past, should go looking for, and the timeframe in which to do so. In what follows, I agree with Hymes that we should look closely at the thirteenth century, not the fourteenth, to find the Black Death's origins. Like Hymes (and other investigators before him), I agree that we need to look beyond the geographies explicitly flagged by our written sources, and seek *Y. pestis*'s history in its real, wild animal haunts. And like Hymes, I connect the expanded geography and chronology of this new Eurasian disease regime with a specific human vector: the Mongols. Where I go beyond Hymes is in using the new biological archive that genetics has opened—first, to zero in on the location where the Big Bang may have occurred, and then to lay out scenarios for the genesis of three of the four new *Y. pestis* lineages. In relating those results back to the archive of human documentation, it is necessary to remember that the *demographic* effects of plague may not have been comparable in all places. Plague is not a human disease. Its impact on human populations will not be the same everywhere, for the simple reason that plague amplifies in dense urban conditions but is far less visible to human perception when it passes through rural or wild terrain. Humans are responsible for plague's long-distance migrations, but that task in the epidemiological chain can be effectively accomplished by only a handful of people. Rather than looking only for dramatic epidemics or mass graves, we must track this killer in its quieter moments, too.

OUR FIRST TASK IS TO OPEN the biological archive. The archive for the study of plague's history is assembled from three types of data: (1) genetic data (aDNA) of *Yersinia pestis* retrieved from the physical remains of historical plague victims; (2) modern genetic

Health Organization] 4 (1951): 475–533. The Issyk Kul case has most recently been used to tell a Black Death story by Philip Slavin, “Death by the Lake: Mortality Crisis in Early Fourteenth-Century Central Asia,” *Journal of Interdisciplinary History* 50, no. 1 (Summer 2019): 59–90.

TABLE 1: Genetics Vocabulary

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- aDNA = ancient DNA, the genetic material obtained from palaeogenetic investigation of archaeological remains; it can be from any living organism, bacterial, plant, or animal
 - founder effect = reduced genetic variation caused by dispersal of a few identical organisms into a new environment^a
 - paleogenetics (“old genetics”) = the study of genetic material retrieved from archaeological materials
 - phylogenetics (“tree genetics”) = the study of genetic (evolutionary) relationships
 - phylogenetic tree = a diagram showing those relationships (“family tree”)
 - phylogeography = analysis of different strains of an organism according to their geographic distribution; the addition of dating information often helps to define biological events
 - polytomy (“many cuts”) = multiple genetic divergences in a single radiation
 - *the* polytomy = the “Big Bang,” a sudden divergence of *Yersinia pestis* into four new lineages, estimated to have occurred in the thirteenth century
 - SNP (single nucleotide polymorphism) = the substitution, at any position along the genome, of one nucleobase (A, C, G, or T) by another; if this mutation proves successful (replicated in many surviving progeny), it becomes a defining feature of a lineage
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^aA fuller definition of the founder effect can be found here: “Bottlenecks and Founder Effects,” Understanding Evolution (website), University of California Museum of Paleontology and the National Center for Science Education, accessed October 22, 2020, https://evolution.berkeley.edu/evolibrary/article/bottlenecks_01.

data of *Y. pestis* retrieved from living isolates of the organism collected over the course of the twentieth and early twenty-first centuries, which are descendants of medieval ancestors; and (3) data about the location and species of the animals from which these samples of the pathogen, “ancient” or modern, have been retrieved. As with most kinds of historical data, questions of dating and provenance are paramount, as are questions about how inferences can responsibly be drawn to connect one kind of data to another. Tracking the evolution of an organism implies acceptance of the double premise that, first, the organism’s biological character (here focusing on its molecular genetic structure) changes over time and, second, that those changes are inherited. Establishing the phylogenetic (“family tree”) connections between different strains of plague gives us the foundation to document the points at which the bacterium’s history has intersected with humans’.

As with any historical archive, we begin by asking: Is it complete? If not—if there are lacunae—can we know how big they are? How many years of records are missing? What geographic areas have no coverage? In other words, how does the archive we have compare to what existed in the past? For *Yersinia pestis*, the archive is extremely incomplete. But for the first time in history, we can begin to assess its dimensions. For the period from the later Middle Ages to the present day, it is an archive with four *fonds*.²¹ Because the four basic collections had a common origin, we know how old each is relative to the others. And all four still have living descendants, so we know both the origin and the endpoint (the modern-day whereabouts and character) of each lineage.

A physical analogy will help clarify these conceptual presumptions. A standard deck of playing cards has fifty-two cards, divided into four suits of thirteen cards each.

²¹ There is actually a fifth, since certain strains of *Y. pestis* that originated before the Black Death still survive (Branch 0) and, for the most part, are still capable of causing human infections. Their role in medieval outbreaks cannot be ruled out, but they will not factor into the present analysis. Evidence for their sudden dispersal in this period would, in the absence of aDNA (and none has yet been retrieved), be a deep divergence that could plausibly be assigned to the twelfth to fourteenth centuries, or a geographic distribution paralleling Mongol movements, which is what I am arguing here from the post-polytomy Branches 1, 3, and 4. From data currently available, I see no evidence of the virulent pre-polytomy lineages moving outside limited geographic niches.

Let those four suits—diamonds (Branch 1), hearts (Branch 2), clubs (Branch 3), and spades (Branch 4)—correspond to our four lineages of *Y. pestis*. The ace is low; the king, high. Now let time, circumstance, and decay do their work, until we have only a fraction of the deck left. Still, because we know what a “complete” deck should look like, we know (approximately) where the lacunae are. For every suit, we have the “king”—the modern isolates of *Y. pestis* collected over the course of the twentieth and twenty-first centuries, since Alexandre Yersin first isolated *Yersinia pestis* microbiologically in Hong Kong in 1894. Modern isolates are samples collected from field studies of plague foci in the wild and from human patients diagnosed with plague.

All these isolates of *Y. pestis*, all these modern “kings” at the top of each suit, had ancestors. Those are the rest of the suit, from the queen down to the ace—the origin of each lineage. But they all died before the laboratory revolution could document them. That’s where aDNA (“ancient DNA”) comes in: the retrieval of “molecular fossils,” fragments of the genomes of bacteria that lived in the past. These molecular fragments for *Yersinia pestis* are obtained from inside the teeth of persons suspected of having had plague.²² New, highly sophisticated computerized technologies can reconstruct up to 99 percent of the ancient genomes of *Yersinia pestis*, totaling several million base pairs. With the results of aDNA, we now have, for the suit of diamonds (Branch 1), the ace, the 2, and the 3, in addition to the king. For the other suits, we still have nothing but the king (the most recent samples). But because we know that every lineage—all four suits—had a common origin, obtaining early samples from even one of the lineages tells us something important about all four.²³ If we start with the presumption that anything alive today has ancestors, the task becomes to document as much as we can about those ancestors and to determine how much change has occurred between them and their known descendants.

One way to trace ancestry genetically is to track SNPs: single nucleotide polymorphisms, where one nucleobase on the genome (“A,” “C,” “G,” or “T”) is replaced by a different one. Every SNP begins in a single bacterium’s cell. When *Y. pestis* is circulating in say, a mouse, that mammalian host may have been infected by as few as ten bacteria. By the time of death, the mouse could have as many as twenty-five million plague bacteria circulating in its body.²⁴ If a SNP occurred in that mouse early in the infectious process, the newly altered bacterium would dominate its current ecological niche: the individual host’s body. For one nucleotide mutation to “take”—to become characteris-

²² Just before a mammalian host dies of plague, the bacterium passes into the bloodstream, replicating endlessly. The teeth are vasculated, so the bacterium ends up there, too. After the host succumbs, the hard enamel of the tooth can help to preserve bits of blood. All *Y. pestis* aDNA that has been retrieved to date has come from human teeth.

²³ The most recent contribution to the paleogenetics of the Second Plague Pandemic is Maria A. Spyrou, Marcel Keller, Rezeda I. Tukhbatova, Christiana L. Scheib, Elizabeth A. Nelson, Aida Andrades Valtueña, Gunnar U. Neumann, Don Walker, Amelie Alterauge, Niamh Carty, Craig Cessford, Hermann Fetz, Michaël Gourvenec, Robert Hartle, Michael Henderson, Kristin von Heyking, Sarah A. Inskip, Sacha Kacki, Felix M. Key, Elizabeth L. Knox, Christian Later, Prishita Maheshwari-Aplin, Joris Peters, John E. Robb, Jürgen Schreiber, Toomas Kivisild, Dominique Castex, Sandra Lösch, Michaela Harbeck, Alexander Herbig, Kirsten I. Bos, and Johannes Krause, “A Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes,” *Nature Communications* 10, no. 1 (2019): 1–13. All prior paleogenetics studies on *Y. pestis* can be found cited there.

²⁴ I have calculated the number of bacteria from data in Florent Sebbane, D. Gardner, D. Long, B. B. Gowen, and B. Joseph Hinnebusch, “Kinetics of Disease Progression and Host Response in a Rat Model of Bubonic Plague,” *American Journal of Pathology* 155, no. 5 (May 2005): 1427–1439.

tic of a whole strain of plague—it has to successfully replicate many millions of times over. In terms of population dynamics, such a mutation would have the best chance to replicate freely and precipitously when just a few identical bacteria come into a new environment, with fresh hosts. Here, the introduced strain is not competing against other strains, so its characteristic genetic sequence defines the organism in the new region through what is called a *founder effect*. Paleogenetics became capable of identifying specific historical strains in 2011, when field-defining new work produced the complete genome sequence from two strains of plague from fourteenth-century London.²⁵ Subsequent work on samples from all over Europe and western Russia now allows us to confirm that the Black Death and plague as it was experienced in western Eurasia for the next four hundred years was the result of a founder effect: the transmission of a single new strain of *Yersinia pestis* (Branch 1) to the Caucasus/Volga region.²⁶

I propose that in establishing that the Black Death was indeed a founder effect, the introduction of a new strain of *Y. pestis* into western Eurasia, the new plague genetics has made it possible to historicize—to pinpoint in space and time—an equally significant event in global plague history: the “Big Bang.”²⁷

THE “BIG BANG” IS A phylogenetic abstraction (Figure 1). Although the term was not invoked until 2013, the phenomenon it refers to was first noted by biologists in 2004, as *Yersinia pestis*’s phylogenetic tree was being sketched out on the basis of the first three whole modern genomes to be sequenced.²⁸ Even as this work was being done with modern isolates of *Y. pestis*, work by other labs was advancing paleogenetic work. In 2011, the complete sequence of *Y. pestis* aDNA was announced from an exceptionally well-dated mass grave in London known as the East Smithfield Black Death Cemetery. Added to analyses of modern genomes, the Black Death genome could now be used to

²⁵ Kirsten I. Bos, Verena J. Schuenemann, G. Brian Golding, Hernán A. Burbano, Nicholas Waglechner, Brian K. Coombes, Joseph B. McPhee, Sharon N. DeWitte, Matthias Meyer, Sarah Schmedes, James Wood, David J. D. Earn, D. Ann Herring, Peter Bauer, Hendrik N. Poinar, and Johannes Krause, “A Draft Genome of *Yersinia pestis* from Victims of the Black Death,” *Nature* 478, no. 7370 (2011): 506–510. It was discovered several years later that the genomes were from two different burial sites, of different dates; see Green, “Plague (*Yersinia pestis*).”

²⁶ Spyrou et al., “A Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes.” As of 2019, Black Death genomes, all identical down to the last SNP, have been retrieved from Barcelona, Saint-Laurent-de-la-Cabrerisse, London, Cambridge, Oslo, and Nabburg (Bavaria). As indicated in Figure 1, Branch 1 splits after the Black Death into what I have called branches 1A (which is only documented from aDNA, all of it—up through the eighteenth century—from within Europe) and 1B (which is documented from aDNA in Bolgar City and from modern samples everywhere in the world). Most recently on Branch 1A, see Karen Giffin, Aditya Kumar Lankapalli, Sushanna Sabin, Maria A. Spyrou, Cosimo Posth, Justina Kozakaitė, Ronny Friedrich, Žydrūnė Miliauskienė, Rimantas Jankauskas, Alexander Herbig, and Kirsten I. Bos, “A Treponemal Genome from an Historic Plague Victim Supports a Recent Emergence of Yaws and Its Presence in 15th Century Europe,” *Scientific Reports* 10, no. 1 (2020): 1–13.

²⁷ Monica H. Green, “Putting Africa on the Black Death Map: Narratives from Genetics and History,” *Afriques* 9 (December 2018): para. 26, <http://journals.openedition.org/afriques/2125>.

²⁸ Mark Achtman, Giovanna Morelli, Peixuan Zhu, Thierry Wirth, Ines Diehl, Barica Kusecek, Amy J. Vogler, David M. Wagner, Christopher J. Allender, W. Ryan Easterday, Viviane Chenal-Francois, Patricia Worsham, Nicholas R. Thomson, Julian Parkhill, Luther E. Lindler, Elisabeth Carniel, and Paul Keim, “Microevolution and History of the Plague Bacillus, *Yersinia pestis*,” *PNAS* 101, no. 51 (2004): 17837–17842. Importantly, this work also drew on partial sequences of several hundred other *Y. pestis* isolates.

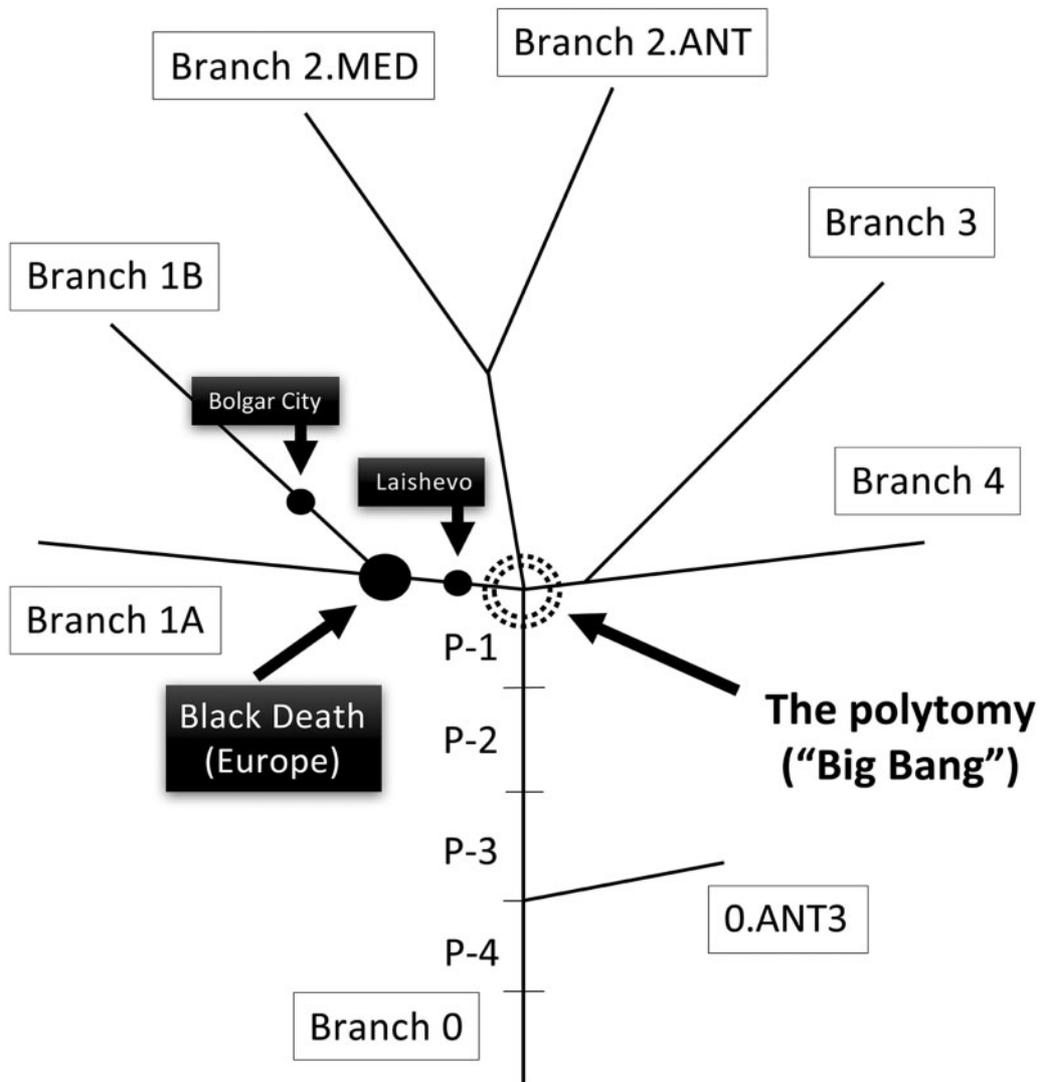


FIGURE 1: Diagram of the polytomy, showing the relationship between the ancient *Yersinia pestis* lineage, Branch 0, and the four new Branches, 1–4, created at the polytomy (the Big Bang). The most recently acquired SNPs (single nucleotide polymorphisms) that the post-polytomy lineages share are marked P-1 through P-3; P-4 is also shared with the extant pre-polytomy lineage, 0.ANT3, found in the Tian Shan range. The Laishevo genomes (one SNP beyond the polytomy), Black Death genomes (two SNPs beyond), and the Bolgar City genomes have been recovered from aDNA.

tether the *Y. pestis* phylogenetic tree to specific chronological parameters, showing definitively that the major dichotomy, which had already been postulated, fell just before the late medieval pandemic. By 2013, two more branches were added to the divergence, turning the dichotomy into a polytomy. This sudden and marked divergence was labeled the “Big Bang.”²⁹ The Black Death as documented for Europe was not itself the Big Bang but merely the aftershock.

²⁹ Yujun Cui, Chang Yu, Yanfeng Yan, Dongfang Li, Yanjun Li, Thibaut Jombart, Lucy A. Weinert, Zuyun Wang, Zhaobiao Guo, Lizhi Xu, Yujiang Zhang, Hancheng Zheng, Nan Qin, Xiao Xiao, Ming-

In terms of its date, the paleogenetics study of 2011 placed the divergence “two SNPs” before the Black Death, whose *terminus ante quem* was indisputable: 1348, the date the London Black Death cemetery was consecrated. But how much time is “two SNPs”? The computerized estimates of the emergence date of Branch 1 do not place its origin confidently in the fourteenth century but suggest that it may have emerged as a distinct lineage in the thirteenth or even the twelfth century.³⁰

The Laishevo genome (taken from a burial site that could only roughly be dated to the fourteenth century on the basis of archaeological indications) proves that the pre-Black Death lineage afflicted humans in the Volga River region.³¹ Where the local reservoir of the Laishevo strain was, or how long it had been present in the region, is as yet unclear. But the genetic proximity of the Laishevo strain to the Big Bang means that we are close to zeroing in on the plague reservoir that produced such a pronounced ecological event. Except: the rest of the biological archive doesn’t point anywhere near to the Volga or the Pontic steppe. The rest of the biological archive points half a continent away.

The rest of the biological archive also takes us, for the moment, out of the Middle Ages and asks us to look at *Yersinia pestis*’s present distribution. In adopting geneticists’ genotyping method to classify modern strains of *Y. pestis*, plague ecologists have laid the foundation for phylogeographical analysis: plotting specific (living) strains to specific locations. Of course, where these strains are now is not necessarily where they were eight hundred years ago. But the possibility that they might have remained locally in host populations that had grown to tolerate the organism is a premise from which to start. Previous work had suggested that *Y. pestis*’s likely point of origin as a new species, and place of long-term development, was China. But that work was based largely on isolates from China, which allowed ascertainment bias to narrow its focus to the Tibetan plateau.³² Subsequent studies have shown that the living strains most closely

shou Wu, Xiaoyi Wang, Dongsheng Zhou, Zhizhen Qi, Zongmin Du, Honglong Wu, Xianwei Yang, Hongzhi Cao, Hu Wang, Jing Wang, Shusen Yao, Alexander Rakin, Yingrui Li, Daniel Falush, François Balloux, Mark Achtman, Yajun Song, Jun Wang, and Ruifu Yang, “Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*,” *Proceedings of the National Academy of Science* 110, no. 2 (2013): 577–582. See Green, “Plague (*Yersinia pestis*),” for details on this process of discovery.

³⁰ The most recent molecular clock estimates proposed are circa 1196 (95 percent confidence interval, 961 C.E. to 1347 C.E.) or even circa 1170 C.E. (921 C.E. to 1346 C.E.). Maria A. Spyrou, Rezeda I. Tukhbatova, Chuan-Chao Wang, Aida Andrades Valtueña, Aditya K. Lankapalli, Vitaly V. Kondrashin, Victor A. Tsybin, Aleksandr Khokhlov, Denise Kühnert, Alexander Herbig, Kirsten I. Bos, and Johannes Krause, “Analysis of 3800-Year-Old *Yersinia pestis* Genomes Suggests Bronze Age Origin for Bubonic Plague,” *Nature Communications* 9 (June 8, 2018): article number 2234, Supplementary Table 9. The two different estimates come from different modes of statistical analysis.

³¹ Spyrou et al., “A Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes.” Unfortunately, no more precise dating of the Laishevo genome is possible than roughly “fourteenth century”; no carbon-dating was done on the remains, and the dating assessment is based solely on cultural artifacts found at the gravesite. Laishevo lies on the northern bank of the Volga River, at its confluence with the Kama River.

³² Achtman et al., “Microevolution and History of the Plague Bacillus, *Yersinia pestis*.” The preliminary assessment adopted in my 2014 overview of plague history (Green, “Taking ‘Pandemic’ Seriously”) should now be rejected. China (in its current borders) still harbors a significant range of *Y. pestis* lineages, including 0.PE7, considered the oldest living strain, with an origin dating back at least five thousand years. But as the discoveries of Late Neolithic and Bronze Age strains of plague have documented, plague has always been an extraordinarily mobile disease. The living strain that is next in age to 0.PE7 is 0.PE2, currently found thousands of kilometers away in the Caucasus Mountains. A number of documented strains thereafter display zigzag patterns across the Eurasian steppe. On the Late Neolithic and Bronze

basal to (evolutionarily older than) the lineages created by the late medieval polytomy come, in fact, from a landmass farther west than the Tibetan plateau: the Tian Shan Mountains, which form the border between modern China and Kyrgyzstan and extend into China's Xinjiang Uygur Autonomous Region up to the Junggar Basin.³³ This ecologically distinct landscape is home to the four extant strains in the 0.ANT group of *Y. pestis*, all of which are distributed in adjacent areas and overlapping marmot populations (see Figure 2). These, in turn, are the group out of which came the strains that caused the two pandemic events that frame the Middle Ages: the Justinianic Plague—normally dated from 541 to ca. 750, with a lineage branching off the phylogenetic tree between the extant 0.ANT1 and 0.ANT2/0.ANT5 lineages—and the Black Death.³⁴

The most derived (evolutionarily youngest) extant strain of the 0.ANT group is 0.ANT3. The 0.ANT3 strains are “cousins” of all the post-polytomy strains; most recently, they share a common ancestor with the SNP we could call “P-4” (“polytomy-minus-4”) on the phylogenetic tree (Figure 1). The pre-polytomy lineage acquired three more SNPs before the Big Bang caused three and then four distinct lineages to emerge.³⁵

Whether that divergence started in the Tian Shan or in a place to which the pre-

Age strains of *Y. pestis*, see most recently He Yu, Maria A. Spyrou, Marina Karapetian, Svetlana Shnaider, Rita Radzevičiūtė, Kathrin Nägele, Gunnar U. Neumann, Sandra Penske, Jana Zech, Mary Lucas, Petrus LeRoux, Patrick Roberts, Galina Pavlenok, Alexandra Buzhilova, Cosimo Post, Choongwon Jeong, and Johannes Krause, “Paleolithic to Bronze Age Siberians Reveal Connections with First Americans and across Eurasia,” *Cell* 181, no. 6 (June 2020): 1232–1245.e20.

³³ Field studies over the course of the twentieth century have documented hundreds of cases of infected marmots in Kyrgyzstan; Gulmira Sariyeva, Gulnara Bazarkanova, Ravshambek Maimulov, Sabirzhan Abdikarimov, Berzhan Kurmanov, Aigul Abdirassilova, Anton Shabunin, Zaurbek Sagiyev, Aigul Dzharparova, Ziyat Abdel, Raikhan Mussagaliyeva, Serge Morand, Vladimir Motin, and Michael Kosoy, “Marmots and *Yersinia pestis* Strains in Two Plague Endemic Areas of Tien Shan Mountains,” *Frontiers in Veterinary Science* 6 (July 2019): 1–13, article no. 207. Only a few of these collected isolates can yet be accessioned into the biological archive being drawn on here, because only a few have been genotyped—classified in a way that allows us to assign them a position on the *Y. pestis* phylogenetic tree. The publication in 2017 of a country-wide study of genotyped *Y. pestis* isolates from Kyrgyzstan was decisive in allowing attention to shift from the Tibetan Plateau to China's western neighbor. See Galina A. Eroshenko, Nikita Yu Nosov, Yaroslav M. Krasnov, Yevgeny G. Oglodin, Lyubov M. Kukleva, Natalia P. Guseva, Alexander A. Kuznetsov, Sabyrzhan T. Abdikarimov, Aigul K. Dzharparova, Vladimir V. Kutyrev, “*Yersinia pestis* Strains of Ancient Phylogenetic Branch 0.ANT Are Widely Spread in the High-Mountain Plague Foci of Kyrgyzstan,” *PLOS ONE* 12, no. 10 (2017): e0187230; Green, “Putting Africa on the Black Death Map,” especially paras. 11, 20, and 32; and G. A. Eroshenko, A. K. Dzharparova, E. G. Oglodin, Zh. V. Al'khova, L. M. Kukleva, A. A. Kuznetsov, Ya. M. Krasnov, S. T. Abdikarimov, V. V. Kutyrev, “Phylogeny of *Yersinia pestis* Strains Belonging to 0.ANT Branch, Isolated in Tien-Shan and Pamir-Alay in the 20th–21st Centuries” [in Russian], *Проблемы особо опасных инфекций* [*Problemy osobo opasnykh infektsiy*] [*Problems of Particularly Dangerous Infections*], no. 1 (2020): 76–84.

³⁴ On the Justinianic Plague, see Marcel Keller, Maria A. Spyrou, Christiana L. Scheib, Gunnar U. Neumann, Andreas Kröpelin, Brigitte Haas-Gebhard, Bernd Pfüffgen, Jochen Haberstroh, Albert Ribera i Lacomba, Claude Raynaud, Craig Cessford, Raphaël Durand, Peter Stadler, Kathrin Nägele, Jessica S. Bates, Bernd Trautmann, Sarah A. Inskip, Joris Peters, John E. Robb, Toomas Kivisild, Dominique Castex, Michael McCormick, Kirsten I. Bos, Michaela Harbeck, Alexander Herbig, and Johannes Krause, “Ancient *Yersinia pestis* Genomes from across Western Europe Reveal Early Diversification during the First Pandemic (541–750),” *PNAS* 116, no. 25 (2019): 12363–12372. On the estimated date of this emergence, see the data collected in Monica H. Green, “When Numbers Don't Count: Changing Perspectives on the Justinianic Plague,” *Eidolon* (November 18, 2019), <https://eidolon.pub/when-numbers-dont-count-56a2b3c3d07>.

³⁵ All the genotyped isolates of 0.ANT3 published to date (Cui et al., “Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*,” n=5, and Eroshenko et al., “Phylogeny of *Yersinia pestis* Strains Belonging to 0.ANT Branch, Isolated in Tien-Shan and Pamir-Alay in the 20th–21st Centuries,” n=8) belong to a single polytomy, which must predate its oldest extant isolate (from 1928). How much further back the documented clade might date is at the moment unclear. Only about ten SNPs sepa-

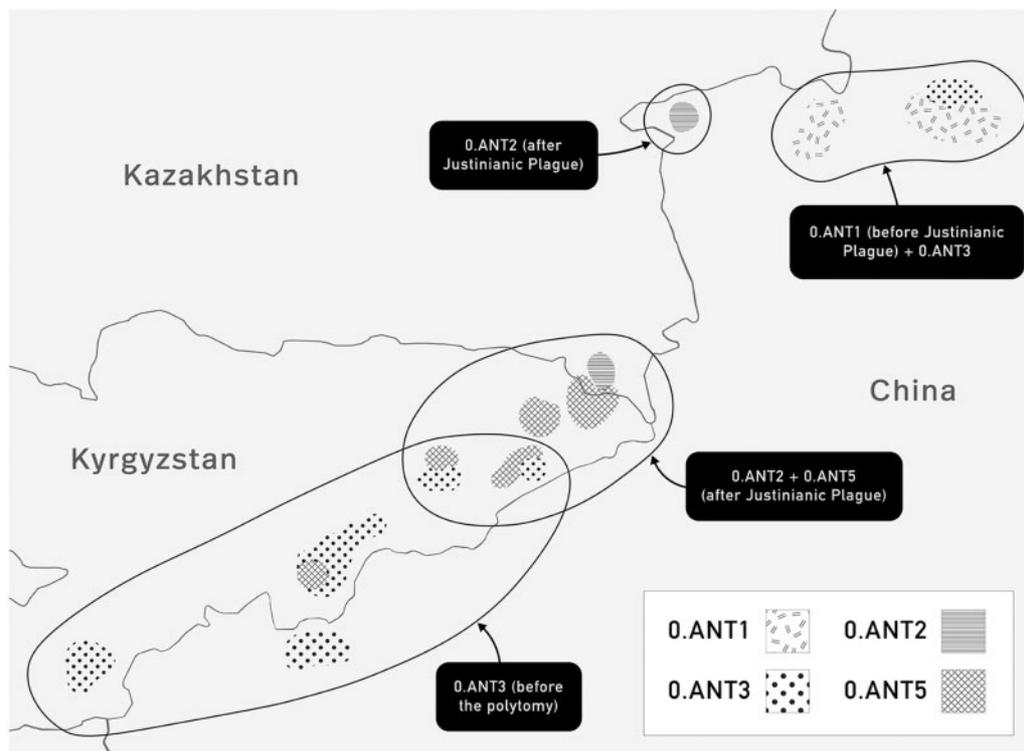


FIGURE 2: Current distribution of pre-polytomy marmot strains of *Yersinia pestis* (0.ANT1, 0.ANT2/0.ANT5, and 0.ANT3) in the Tian Shan Mountains and Junggar Basin. Based on Eroshenko et al., “*Yersinia pestis* Strains of Ancient Phylogenetic Branch 0.ANT Are Widely Spread in the High-Mountain Plague Foci of Kyrgyzstan”; map design: Erica Fagin.

polytomy lineage had already been displaced cannot now be determined. What *can* be determined is where all the known descendants of the Big Bang are now. As with the Laishevo strain, the next documentable phases of *Y. pestis*'s evolution—the strains of *Y. pestis* that occupy the next derived (evolutionarily later) positions on the phylogenetic tree—are nowhere near the Tian Shan range. Instead, the closest surviving strains—the modern isolates of Branches 3 and 4, the “kings” of their respective suits—are found at least one thousand kilometers away, in Siberia, Mongolia, and China.³⁶ Branch 2 tells a different geographic story. After persisting in a single reservoir (or at least, surviving as a single lineage) for some period (long enough to acquire seventeen SNPs in common), Branch 2 split: one sub-branch (2.ANT) moving in a north-south pattern from Inner Asia to Tibet and India; and a second sub-branch (2.MED) extending east-west across the entire width of China to the Caspian Sea.³⁷

rate the most recent common ancestor of the extant clade from the polytomy. It is possible that the present geographic distribution of 0.ANT3 only dates from the past century or two.

³⁶ When Cui et al., “Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*,” originally described the Big Bang, they were working with only one isolate of Branch 4. Since several additional isolates have now been sequenced, it has become clear that Branches 3 and 4 shared a common ancestor (marked by a single SNP) before diverging.

³⁷ On the distribution of 2.MED and 2.ANT strains, see Green, “Putting Africa on the Black Death Map,” paras. 4, 23 n. 38, and 38. Three isolates of 2.MED1 have been documented in the Tian Shan range

Unlike its cosmological namesake, plague's Big Bang is unlikely to have happened as a single event on a single day. The Big Bang—the four founder effects created by the final stage of the pre-polytomy lineage—may have been an “event” spread out over months or even decades as each particular new population of *Y. pestis* developed in a new niche. But with the exception of Branch 2 (whose early history is the most opaque), each of the newly formed branches gives a clue to how quickly it moved into new terrain. While Branch 1 has only one SNP separating the Laishevo genome from its presumed Tian Shan ancestor, Branches 3 and 4 (documented in the twentieth and twenty-first centuries, rather than from fourteenth-century isolates) have as little as fifteen or eighteen SNPs separating them from the polytomy, approximately eight hundred years ago.³⁸ The minimal amount of genetic change in certain strains of Branches 3 and 4 indicates that, although now found in different marmot host species from their presumed medieval ancestor, these new lineages show no signs of having undergone extensive epidemic (or epizootic) proliferation owing to continued cross-species transmission. As far as the biological archive can inform us now, in other words, the distribution of *Y. pestis* into at least three (and then four) completely new, non-adjacent ecological niches was not a drawn-out process. Rather, it seems to have been quick. I propose that it was caused by human activity.

IF THE WIDESPREAD DISTRIBUTION of the four lineages created by the Big Bang was due to human activity, then it must have happened on a human timescale, through documentable events in human archives. In what follows, acknowledging that I am working without corroborating genetic data, I identify a series of historical circumstances that might account for the Big Bang. I propose that the pattern of spread of the post-polytomy lineages overlaps geographically with the extent of the Mongol Empire. Moreover, I argue that certain events in the period of Mongol expansion (when examined closely, as Hymes has done for the sieges in Song China) yield evidence suggestive of the presence of plague, or at least of mechanisms by which it might have propagated. Three scenarios capture phases of this process. They are meant to advance three assertions: (1) that the Mongols, having stumbled upon a population of plague-infected marmots in their initial incursions into the area around the Tian Shan mountains, went on to create a connected marmot enzootic landscape, seeding the disease in marmot populations in areas quite distant from the Kyrgyzstan range; (2) that the transmission of plague westward to the regions south and north of the Caucasus can be plausibly

in the same high-mountain plague foci as many isolates of the pre-polytomy lineage, 0.ANT3; see Table 1 of Eroshenko et al., “*Yersinia pestis* Strains of Ancient Phylogenetic Branch 0.ANT Are Widely Spread in the High-Mountain Plague Foci of Kyrgyzstan.” However, of the four major sublineages of 2.MED that have now been identified, 2.MED1 is the most derived, the furthest evolutionarily from the polytomy; it may not have evolved until the nineteenth century. Its presence in the Tian Shan is therefore likely a reintroduction, not an echo of a medieval event.

³⁸ On a faster rate of genetic change during epidemic proliferations, see Cui et al., “Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*”; on a case of acquisition of new SNPs as the disease moved repeatedly across several host species, see Ruixia Dai, Baiqing Wei, Haoming Xiong, Xiaoyan Yang, Yao Peng, Jian He, Juan Jin, Yumeng Wang, Xi Zha, Zhikai Zhang, Ying Liang, Qingwen Zhang, Jianguo Xu, Zuyun Wang, Wei Li, “Human Plague Associated with Tibetan Sheep Originates in Marmots,” *PLOS Neglected Tropical Diseases* 12, no. 8 (2018): e0006635.

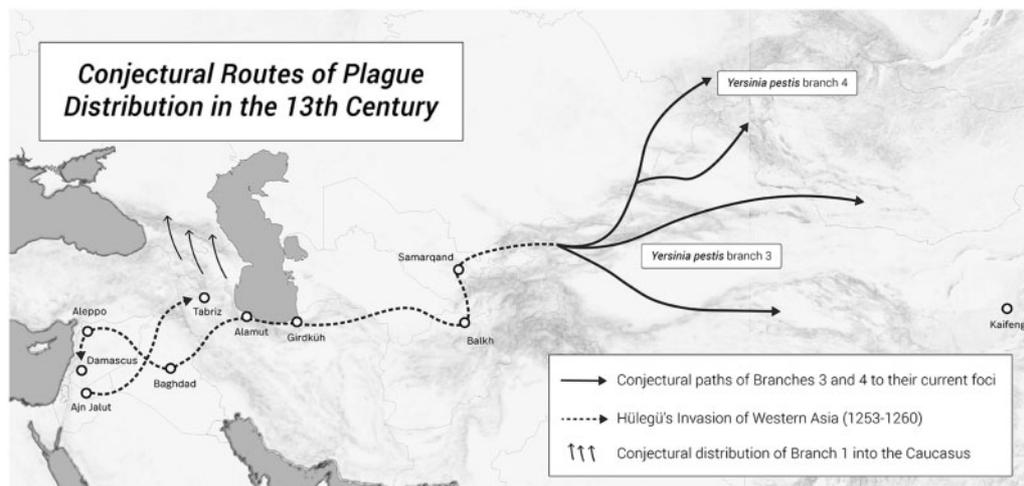


FIGURE 3: Conjectural routes of plague distribution in the thirteenth century, including the route of Hülegü's campaign through central and west Asia, 1253–1260. Data on Hülegü's route adapted from Robert Marshall, *Storm from the East: From Genghis Khan to Khubilai Khan* (Berkeley, Calif., 1993). Map design: Erica Fagin.

assigned to the thirteenth century, instead of the traditional chronology tying it to the Black Death outbreaks of the 1340s; and finally (3), that a specific scenario of political and economic disruption in the Altai region (between Siberia and Mongolia) in the later thirteenth century takes on a new dimension if *Yersinia pestis* is added to the mix of historical actors. I call these “scenarios” to stress that the evidence is only circumstantial. But all, I believe, are plausible reconstructions that gather scattered documentary evidence (very little of which has yet been connected to plague narratives) with elements of the biological story of *Yersinia pestis*'s development gleaned thus far from phylogenetics. Collectively, they explain why a map of plague's hypothesized spread in the thirteenth century (Figure 3) overlaps with the main trajectories of Mongol conquest (Figure 4).

IN HIS 2014 HYPOTHESIS about the Mongols' role in bringing plague to thirteenth-century China, Hymes postulated a mechanism by which Mongols encountered the disease: eating plague-ridden marmots.³⁹ We can add to the evidence usefully gathered by Hymes with the observation that Mongols used marmots not only for food and fur, but also as a source of leather. The description of marmots' properties in the 1330 *Yin-shan cheng-yao* (*Proper and Essential Things for the Emperor's Food and Drink*) by the chief

³⁹ Hymes, “A Hypothesis on the East Asian Beginnings of the *Yersinia pestis* Polytomy.” There are several species of rodents that play roles as primary hosts for plague. All extant strains of *Yersinia pestis* prior to those found in the Tian Shan primarily persist in voles. Gerbils and jerboas are the primary hosts for strains scattered now across central Asia (primarily strains in the Branch 2 lineage). Any number of animals can be secondary hosts, especially animals like field mice that graze around marmot burrows, and predators that prey on marmots or on other small rodents that happen to pick up their fleas. However, marmots of the Tian Shan are currently the primary hosts for the 0.ANT strains leading up to the polytomy. In the online supplementary material, “Marmots and Their Plague Strains,” I have collected data from several dozen field studies and genetics papers—itemizing the genotype, host species, date of collection, and locale of all species of marmots documented with plague. As explained there, all other marmot species known to harbor *Y. pestis* carry post-polytomy strains.

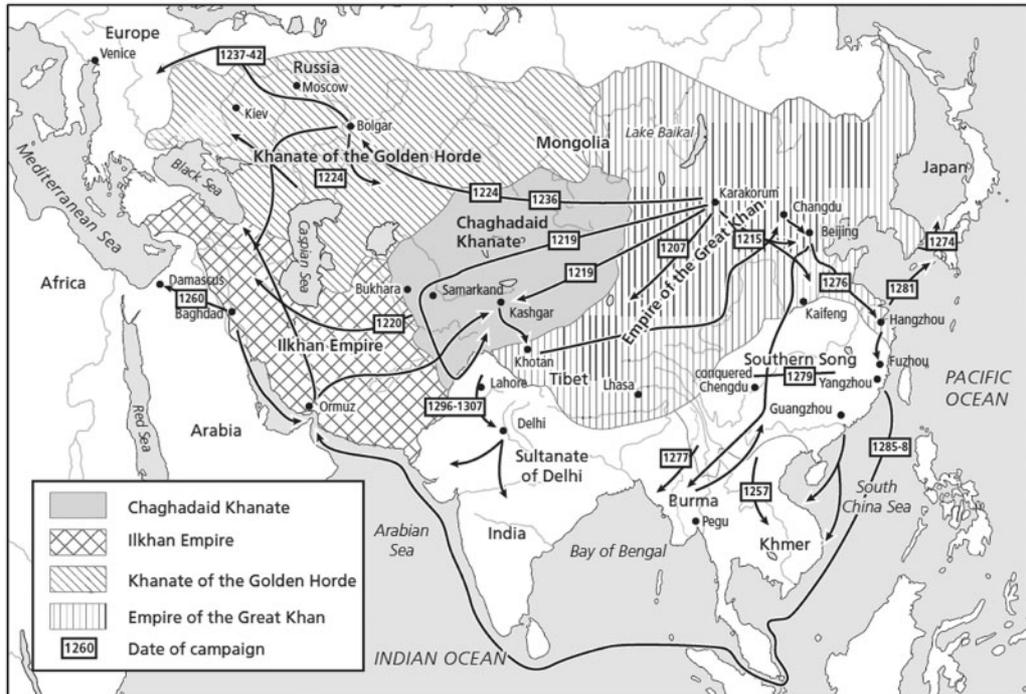


FIGURE 4: The Mongol conquests and the Four Khanates. Source: Biran, “The Mongol Empire,” map20.1. Used with permission.

dietary physician of the Yuan court of China, Hu Sihui, makes note not only of marmots’ utility as a source of meat and broth, but also as a good source of *fanpi*, or “nomad leather,” which is impermeable to water, Hu explains, and very warm.⁴⁰ Just how often the Mongols consumed marmots, for any purpose, is not clear. Chinggis Khan is described consuming marmots during his period of exile, and foreign observers of the Mongols—Islamic, Christian, and Buddhist alike—noted the special predilection the Mongols had for consuming rodents.⁴¹ Many of the new terrains that the Mongols moved into in the course of their thirteenth-century campaigns were inhabited by marmots, as was their homeland (see online supplemental file, “Marmots and Their Plague Strains”), and it would be reasonable to assume (as did John Masson Smith in his 1984 survey of Mongols’ food supplies) that they were regularly exploiting local marmot populations in each new region they entered.⁴² However, since Hu was still recom-

⁴⁰ Paul D. Buell and Eugene N. Anderson, eds. and trans., *A Soup for the Qan: Chinese Dietary Medicine of the Mongol Era as Seen in Hu Sihui’s ‘Yinshan Zhengyao’; Introduction, Translation, Commentary, and Chinese Text* (rev. ed., Leiden, 2010), 513. My thanks to Timothy Brook for this translation of *fanpi*. John Masson Smith, “Mongol Campaign Rations: Milk, Marmots, and Blood?,” *Journal of Turkish Studies* 8 (1984): 223–228, usefully collected a range of evidence, from Mongol sources and those of foreign visitors, attesting to the Mongols’ regular use of rodents as a protein source.

⁴¹ Igor de Rachewiltz, trans., *The Secret History of the Mongols: A Mongolian Epic Chronicle of the Thirteenth Century* (Leiden, 2006), 25, sec. 89 and 90.

⁴² On current marmot classifications, see Boris Kryštufek and Vladimír Vohralík, “Taxonomic Revision of the Palearctic Rodents (Rodentia). Part 2. Sciuridae: *Urocitellus*, *Marmota* and *Sciurotamias*,” *Lynx, series nova* 44 (2013): 27–138. Their maps in figures 12, 23, 27, 33, 37, 41, and 44 show the distribution of all eight extant Eurasian species of marmots. Most species’ ranges have contracted, due to over-

mending consumption of marmot meat in the Yuan imperial court in 1330, there seems not to have been any general assumption by that point that marmots were (or had become) dangerous. The biological archive supports that assumption, giving no indication that virulent strains of plague were present in Mongolia before Chinggis Khan's reign.⁴³ Rather, the introduction of virulent strains of *Y. pestis* into marmot populations outside of the Tian Shan seems limited to the post-polytomy period. Once the plague-ridden marmot populations of the Tian Shan had been encountered, the work of hunting, cooking, and tanning marmot hides would have put every hunter, cook, and tanner at potential risk of infection. If the advancing troops carried with them previously slaughtered carcasses or prepared skins (which might have harbored infectious fleas or dung, which can also transmit *Y. pestis*), then we get a scenario favoring long-distance transmission.⁴⁴

Hymes's hypothesis was also crucial for a new conception of plague's history, positing a much earlier *terminus ante quem* for the beginning of the pandemic. The Mongols seem to have been involved in epidemic outbreaks at the sites of several sieges between 1218 and 1232, suggesting that they brought plague with them as they advanced into China. These were major military events, led by Chinggis Khan's top generals. For the Kaifeng siege, we have the exceptionally valuable testimony of a royal physician, Li Gao. According to Hymes, Li first recognized the inadequacy of the old category of "cold damage" disease (what might be called epidemic diseases) to explain the most striking symptom of this new affliction: its exceptional fatality rate. Li formulated a new category, "internal damage," reporting deaths in excess of one thousand a day for almost three months at the height of the epidemic.⁴⁵

Where Hymes's hypothesis needs adjustment is in tying this scenario of the Mongols' encounters with plague to their initial raids on the Xi Xia (western Xia state) and their subsequent campaigns into northern China via the Gansu corridor. This scenario is not supported by the biological archive. As with Mongolia itself, the only strains of plague documentable in marmots in Gansu province today are post-polytomy

hunting and deliberate eradication programs. It has not yet been investigated whether the highly virulent Tian Shan strains of *Yersinia pestis*, whose primary hosts are gray and long-tailed marmots (*M. baibacina* and *M. caudata*), might have caused considerable die-offs among other species of the open steppe. I find it notable that neither Menzbier's marmot (*Marmota menzbieri*, found in limited areas of Kyrgyzstan and eastern Kazakhstan) nor the bobak (*M. bobaka*, found widely in Kazakhstan and western Russia, though formerly ranging also into Ukraine) are currently found to be infected with plague.

⁴³ As Julia Riehm and colleagues observed in 2012, the strains of plague currently documented in Mongolia are either of considerable antiquity (first or second millennium B.C.E.) or they are post-polytomy strains. Importantly, the former are found in voles, not marmots; some of them, indeed, are avirulent in humans. Julia M. Riehm, Gilles Vergnaud, Daniel Kiefer, Tserenorov Damdindorj, Otgonbaatar Dashdavaa, Tungalag Khurelsukh, Lothar Zöller, Roman Wölfel, Philippe Le Flèche, and Holger C. Scholz, "Yersinia pestis Lineages in Mongolia," *PLOS ONE* 7, no. 2 (2012): e306242012; and K. A. Nikiforov, L. M. Kukleva, Zh. V. Al'khova, E. A. Naryshkina, N. P. Guseva, G. A. Eroshenko, E. G. Tokmakova, S. V. Balakhonov, and V. V. Kutyrev, "Phylogeographic Analysis of *Yersinia pestis* Subspecies *ulegeica* Strains," *Russian Journal of Genetics* 56, no. 7 (2020): 802–809. Since Riehm et al. published in 2012, new discoveries of Bronze Age plague have pushed back the likely chronology of all the extant lineages on the 0 Branch of the *Y. pestis* phylogeny. See most recently Spyrou et al., "Analysis of 3800-Year-Old *Yersinia pestis* Genomes Suggests Bronze Age Origin for Bubonic Plague."

⁴⁴ This scenario is more plausible than the idea that sick soldiers or camp attendants were continually transmitting it to each other. Primary pneumonic plague (inhalation of bacteria coughed out by a person already suffering from an advanced infection) kills within two to three days, and would likely leave an individual too sick to travel within one day.

⁴⁵ Hymes, "A Hypothesis on the East Asian Beginnings of the *Yersinia pestis* Polytomy."

strains.⁴⁶ A viable scenario would need to connect the Mongols to the Tian Shan, more than one thousand kilometers southwest of their homelands in Mongolia, and do so at a date before 1218, which marks the first siege in China that seems to have involved a plague outbreak. In fact, there is ample evidence for such an encounter. A little less than a century before the Mongol expansion began in 1206, another group, the Qara Khitai, had moved into the area out of which the pandemic strains of plague emerged: modern-day Xinjiang Autonomous Region and Kyrgyzstan. The Mongols' conquest of the Qara Khitai began in 1216 and was completed by 1218. Indeed, we cannot rule out the possibility that it was the Qara Khitai who initially dislodged plague from the Tian Shan and set in process the Big Bang. The Qara Khitai were themselves an invading force: refugees from the Liao state in northern China who had only arrived in the region in 1124 and would have had no prior experience with these particularly lethal strains of the disease.⁴⁷ But the Mongols would have facilitated plague's long-distance migrations, since all the post-polytomy lineages follow the tracks of their conquests.

With some rare exceptions, the different marmot species of central and east Asia are not populations that intermix or migrate into each other's territory.⁴⁸ Yet unlike in the Tian Shan range, where sequential evolutionary stages of *Y. pestis* are found in adjacent or overlapping marmot population niches (Figure 2), post-polytomy *Y. pestis* strains are found in widely dispersed marmot populations. Thus, for example, tarbagan marmots in Mongolia (*Marmota sibirica*) carry the post-polytomy lineages 3.ANT2 and 4.ANT, neither of which are much genetically changed beyond their common ancestor at the time of the Big Bang. The Himalayan marmots (*M. himalayana*) in central China, Tibet, and Nepal, which likewise currently have no contiguous or overlapping ecological ranges with the Tian Shan marmots, harbor 3.ANT1, 2.MED3, 2.ANT1, and 2.ANT2. The distribution of plague in Asia's marmot populations thus seems to show that the disease has moved between marmot populations that themselves show no sign of overlapping or intermixing. The time-depth during which these disease transfers occurred cannot now be confirmed with any accuracy, save that they must have happened after the Big Bang.⁴⁹ Nor can we yet rule out that other species of rodents, or other mechanisms of transmission, were involved in carrying plague into these various marmot populations. Nevertheless, if we juxtapose the evidence from the biological archive with the evidence that Hymes brought forward of plague-like outbreaks at the major sieges in

⁴⁶ Christos Lynteris, "Tarbagan's Winter Lair: Framing Drivers of Plague Persistence in Inner Asia," in Christos Lynteris, ed., *Framing Animals as Epidemic Villains: Histories of Non-Human Disease Vectors* (Cham, 2019), 65–90, implies that the association of marmots with plague is a recent phenomenon, within the past century or so. My argument would push the association with Mongolian marmots back to the thirteenth century. Again, see the online supplemental file, "Marmots and Their Plague Strains."

⁴⁷ Michal Biran, *The Qara Khitai Empire in Eurasian History: Between China and the Islamic World* (Cambridge, 2005), is the foundational study of the Qara Khitai empire, though it mentions plague as a possible factor in the Qara Khitai history not even once.

⁴⁸ R. P. Zimina, I. P. Gerasimov, Jingyan Yan, Hongjian Chen, Gonghua Lin, Qian Li, Jiarui Chen, Wen Qin, Jianping Su, and Tongzuo Zhang, "Genetic Evidence for Subspecies Differentiation of the Himalayan Marmot, *Marmota himalayana*, in the Qinghai-Tibet Plateau," *PLOS ONE* 12, no. 8 (2017): e0183375.

⁴⁹ The marmots of central and southern China also carry the following post-polytomy strains, which are now understood to be Branch 1B strains that formed after the west Eurasian Black Death (probably in the area where the Bolgar City genome was retrieved; see Figure 1) and then spread eastward across central Asia: 1.IN1, 1.IN2, and an unnamed strain intermediate between 1.IN2 and 1.IN3. With that next stage of Branch 1's development, 1.IN3, *Y. pestis* moves out of marmot hosts entirely and into other rodent populations.

Song China between 1218 and 1232, both phenomena might be explained circumstantially by the early thirteenth-century Mongol campaigns through what seem to have been ecosystems rife with plague.

THE CAUCASUS HAD BEEN HOME to both marmots and earlier lineages of plague for several thousand years.⁵⁰ Nevertheless, the direct involvement of marmots in the westward transmission of plague after the Big Bang seems unlikely, not simply because it is unclear whether marmots still existed in the Caucasus/Volga region in the Middle Ages, but also because none of the intervening marmot species across the steppe have been documented harboring plague.⁵¹ Rather, it seems that other mechanisms were involved in plague's westward spread. Three episodes in early Mongol history, each tied to a different generation of Mongol leadership, connect the landscapes of the Tian Shan with the Caucasus. Having taken the eastern portion of the Qara Khitai empire by 1218, Chinggis Khan himself headed the 1218–1225 campaign against the Khwarazmshah, in the territory to the west of the Tian Shan formerly controlled by the Qara Khitai. This campaign ended with the destruction of the Khwarazmian state in what is now Kazakhstan and Uzbekistan.⁵² Under the second generation of Chingissid leadership, the Mongols pressed farther west. Chinggis Khan's eldest son, Jochi, was charged with one detachment, while his generals Jebe and Sube'etai (who would later lead the 1232 siege of Kaifeng) passed through the territory of modern Iran and turned northward through the Caucasus. They destroyed Georgia and defeated the Alans and other rival groups in what is now southern Russia. After Chinggis Khan's death, in 1227, his third son, Ögödei, was elected his successor in 1229. Once again, he sent Mongol forces westward. Again, there was a focus on subjugating Georgia and Armenia, but attention also turned farther north to several forest peoples of the Volga-Ural region.⁵³ Importantly, it was on this campaign that the Mongols established permanent claims of pasturage on the rich lands of Azerbaijan (now northern Iran).

There seem to be no plague-like events reported for either of these campaigns, but that need not be decisive. On a military campaign, death (among all parties) was common—even expected—and the death of, say, a camp attendant charged with preparing

⁵⁰ The Greater Caucasus Mountains, whose steep landscape closely resembles the Tian Shan's, had already proved a viable home for plague in previous millennia; there is strong circumstantial evidence that it or the Volga basin became a new long-term reservoir for Branch 1. On plague's earlier history in the region, see Aida Andrades Valtueña, Alissa Mittnik, Felix M. Key, Wolfgang Haak, Raili Allmäe, Andrej Belinskij, Mantas Daubaras, Michal Feldman, Rimantas Jankauskas, Ivor Janković, Ken Massy, Mario Novak, Saskia Pfrengle, Sabine Reinhold, Mario Šlaus, Maria A. Spyrou, Anna Szécsényi-Nagy, Mari Törv, Svend Hansen, Kirsten I. Bos, Philipp W. Stockhammer, Alexander Herbig, and Johannes Krause, "The Stone Age Plague and Its Persistence in Eurasia," *Current Biology* 27, no. 23 (December 2017): 3683–3691.e8. An ancient species of marmot formerly inhabited the Caucasus. See Aija Macāne, Kerkko Nordqvist, and Elena Kostyleva, "Marmot Incisors and Bear Tooth Pendants in Volosovo Hunter-Gatherer Burials: New Radiocarbon and Stable Isotope Data from the Saktysh Complex, Upper-Volga Region," *Journal of Archaeological Science: Reports* 26 (August 2019): 101908; and Gennady Baryshnikov, John F. Hoffecker, and Robin L. Burgess, "Palaeontology and Zooarchaeology of Mezmaiskaya Cave (Northwestern Caucasus, Russia)," *Journal of Archaeological Science* 23, no. 3 (1996): 313–335. There is not yet any evidence, however, that those marmots were involved in plague transmission.

⁵¹ See the online supplemental file, "Marmots and Their Plague Strains."

⁵² Carl Fredrik Sverdrup, "Sübe'etei Ba'atur, Anonymous Strategist," *Journal of Asian History* 47, no. 1 (2013): 33–49; Christopher Atwood, "Jochi and the Early Western Campaigns," in Morris Rossabi, ed., *How Mongolia Matters: War, Law, and Society* (Leiden, 2017), 35–56.

⁵³ Jackson, *The Mongols and the Islamic World*, 39–40.

a *boodog*-like meal (a kind of marmot barbecue), or a tanner making marmot hides into *fanpi* (“nomad leather”) might not be investigated or even noticed by anyone in a position to record it.⁵⁴ Again, plague need not have caused massive death to have successfully moved long distances.

However, a third scenario of pre-fourteenth-century transmission to the west has yet to register in plague historiography. After the campaigns of the 1220s, the 1230s, and another in the 1240s, which pushed across the Pontic steppe as far west as Hungary, one of Chinggis Khan’s grandsons, Hülegü, was charged in 1253 by his brother Möngke Khan (who had by then been installed as the Great Khan) to lead a massive army to conquer the remaining Muslim states in southwestern Asia. Hülegü spent nearly two years traveling between Qaraqorum (the seat of the Great Khan) and Samarkand, assembling his forces and the provisions needed to sustain them. After setting up a new headquarters at Hamadan (western Iran) in 1256, Hülegü successfully besieged the Ismaili holdout at Alamut. The Mongol troops then moved on to Lanbasar, another Ismaili fortress, which they proceeded to besiege. The defenders held out for a full year, and *wabā* (“epidemic” or “pestilence”) broke out at the end of the siege, in 1257, oddly paralleling the circumstances of the plague outbreaks in the sieges in China, as noted by Hymes.⁵⁵ *Wabā* and *ranjūrī* (“pestilence and sickness”) were also reported to have broken out during an earlier siege of Girdkuh in 1254 (though in that case, the siege recommenced and continued for years afterward).⁵⁶ Several months later, in October 1257, Hülegü’s forces set out for Baghdad; there, too, “pestilence” broke out after what became a very short siege. According to one account, copied between 1282 and 1285, Hülegü arrived on January 19, 1258, after his advance troops:

When Hülegü reached Baghdad the rest of his army, who were already in the city, was standing on the ramparts. Because a great assemblage of people, namely all the people of the Sawād, had come to the city before the Mongol army arrived [in other words, the city’s population was already swollen because of the refugees], there was a great dearth, want, and scarcity of provisions in Baghdad. Pestilence [*wabā*] struck and many people died. The number of deaths reached the point that the Ministry’s priority was to prepare the corp-

⁵⁴ *Boodog* is a special preparation method employing hot stones inserted into the animal’s whole carcass for cooking. Encounters with both the animal’s blood and fleas would be expected.

⁵⁵ The siege of Lanbasar was reported by both Quṭb al-Dīn al-Shīrāzī (d. 1311) and Rashīd al-Dīn (d. 1318), both connected to Hülegü’s court in Tabriz. For the former, see Quṭb al-Shīrāzī, *The Mongols in Iran: Quṭb al-Dīn al-Shīrāzī’s Akhbār-i Moghūlān*, trans. and ed. George Lane (Abingdon, 2018), 52/82 [28]; Lane provides a “straight translation” (46–75) and, separately, an annotated version (76–109); I cite page numbers of the English translations and (in square brackets) the Persian edition pagination, which Lane uses to divide the text. For Rashīd al-Dīn’s account, see W. M. Thackston, trans., *Rashiduddin Fazlullah’s Jami’u’tawarikh (Compendium of Chronicles)* (Cambridge, Mass., 1999), 485: “He [Hülegü] sent Sadruddin to supervise the handing over of all the strongholds and fortifications of his fathers and forefathers had acquired over time in Quhistan, Rodbar, and Qumis, and which were filled to the brim with vessels and treasures. The number came to a hundred. The castellans were made to come out, and all the fortresses were razed except for Gird Koh and Lammasar. His kinsmen and adherents held Lammasar for a year. After that, pestilence broke out and many died. Those who were left came out and joined the others [i.e., were killed].” The name of the fortress is spelled “Lamsar” and “Lamasar” in other sources. My thanks to Sean Anthony for confirming the original Arabic term used by Rashīd al-Dīn, and to Matthew Melvin-Koushki for confirming the Persian text of al-Shīrāzī.

⁵⁶ Al-Shīrāzī, *The Mongols in Iran*, on the siege at Girdkuh, 51/80 [26] (*wabā*) and 52/82 [28] (*wabā* and *ranjūrī*). Again, my thanks to Matthew Melvin-Koushki for confirming the original Persian text. In the former case, Lane translates *wabā* as “cholera.” This surely is unwarranted, though it is commonly found in English translations from Arabic and Persian made after the cholera pandemics of the nineteenth century, when the classic term *wabā* (in Persian, or *waba’* in Arabic) shifted to a new use.

ses and bury them. Meanwhile the situation deteriorated so much that the people of Baghdad could no longer cope with ablutions and burial of the dead, so bodies were thrown into the Tigris. In the end, it became so bad that they could not even carry the dead to the Tigris on account of the number who were dying, as the porters did not have time and were no longer turning up. At this point the caliph commanded that an empty property be assigned for each area and the dead be put there and as it filled and as they found the opportunity, they would bury them. Even when the army arrived, they were unable to cope with the situation.⁵⁷

This account of the epidemic at Baghdad comes from a recently rediscovered text, called *Akḥbār-i Moghūlān*, or *Mongol News*. The manuscript in which it was found was copied by Quṭb al-Dīn al-Shīrāzī (d. 1311), who worked as a physician and medical teacher before taking up a position as an astronomer and philosopher at the Ilkhanate court, in 1262. Whether al-Shīrāzī was the text's author or merely its copyist is unclear, but we know of his interest in the topic of plague because of his detailed comments on the disease's symptoms in his commentary on Avicenna's medical encyclopedia, the *Qanun*.⁵⁸ In *Mongol News*, the pestilence is the most striking aspect of the horrific scene and the singular distinction of al-Shīrāzī's account vis-à-vis other contemporary narratives of the Baghdad siege.⁵⁹

This account is important for a second reason. Al-Shīrāzī offers a detail that we might understand (with our modern conceptions of the ecology of plague) as providing the "means and opportunity" for plague's transmission to Baghdad: the Mongols brought provisions with them from their other conquered territories, laid out in linen bags at various stations along their route. These included "pounded millet (*gāvrus-i kufteh*) from the provinces of Khitai and Uyghuristan," lands that had come under the control of the Mongols with the conquest of the Qara Khitai.⁶⁰ In other words, the Mongols shipped their own grain supplies all the way from Tian Shan territory up to the walls of Girdkuh, Lanbasar, and Baghdad, creating the ideal conditions to bring tag-along rodents and their fleas.⁶¹ This would also account for a further detail that

⁵⁷ Al-Shīrāzī, *The Mongols in Iran*, 54/84/85 [32].

⁵⁸ Lane remains undecided on the issue of al-Shīrāzī's authorship; see al-Shīrāzī, *The Mongols in Iran*, 9–11. My thanks to Nahyan Fancy for information on al-Shīrāzī's commentary. On al-Shīrāzī's biography, see Sayyed 'Abd-Allāh Anwār, "Qoṭb-al-Dīn Shīrāzī," *Encyclopedia Iranica*, originally published July 20, 2005, accessed June 25, 2020, <http://www.iranicaonline.org/articles/qotb-al-din-sirazi>; and Leigh Chipman, "The 'Allāma and the Ṭabīb: A Note on Biographies of Two Doctors, Rashīd al-Dīn and Quṭb al-Dīn al-Shīrāzī," in Anna Ayse Akasoy, Charles Burnett, and Ronit Yoeli-Tlalim, eds., *Rashid Al-Din: Agent and Mediator of Cultural Exchanges in Ilkhanid Iran* (London, 2013), 115–126.

⁵⁹ For example, Hend Gilli-Elewy, "*Al-Hawādīṭ al-ḡāmi'a*: A Contemporary Account of the Mongol Conquest of Baghdad, 656/1258," *Arabica* 58, no. 5 (2011): 353–371, here 368, likewise mentions an epidemic, but does so almost in passing. In forthcoming work, Nahyan Fancy and I have assembled various witnesses to this "epidemic" in Baghdad and the surrounding region in 1258.

⁶⁰ Al-Shīrāzī, *The Mongols in Iran*, 19. Here I am quoting from the translation given in Lane's introductory chapter. In his two versions of the main translation, 50/80 [24], Lane instead renders Khitai as "northern China." My thanks to Matthew Melvin-Koushki for confirming that the original Persian reads "*Khajā*" and "*Yughuristān*." The anonymous *al-Hawādīṭ al-ḡāmi'a* also confirms how intensely Hülegü outfitted the expedition with provisions: he arrived in Baghdad "with soldiers whose numbers cannot be counted and whose supply lines never ended" (Gilli-Elewy, "*Al-Hawādīṭ al-ḡāmi'a*," 364).

⁶¹ These rodents were unlikely to have been marmots, whose presence (because of their size) would not have gone unnoticed. Rather, it was more likely small commensal rodents, already habituated to human spaces, which are documented as the main transitional hosts involved in human outbreaks of plague. Millet is well-documented among the grains that attract rodents. Millet (particularly the glutinous millet grown in this area of northern China and central Asia) was specially prized as the basis for a gruel that, according to the Franciscan observer of the Mongols, John of Piano Carpini, writing in the late 1240s,

al-Shīrāzī adds. It was not simply the besieged inhabitants of the city who became ill, but also the Mongol army itself: “and many died of the disease.” Among those stricken (though he later recovered) was Hülegü himself.⁶² The epidemiological scenario seems to be this: once the siege was over, provisions stockpiled by the Mongols were shared between conquerors and conquered alike. Hence, the mutual risk. This outbreak has the same epidemiological profile as the outbreaks Hymes found among the Chinese sieges earlier in the century, where the epidemics only started with the lifting of the siege.⁶³

After Baghdad, satellite outbreaks of “epidemics” (and what is now specifically called “plague”: *tā'ūn*) are reported by other chroniclers, writing in Arabic and Syriac, in Syria, Anatolia, and even Egypt.⁶⁴ It is significant that as *wabā'* and *tā'ūn* were breaking out in Syria, Hülegü, by then ill himself, retreated into Azerbaijan (northern Iran) after the siege of Baghdad in March 1258.⁶⁵ If Hülegü's campaign bore responsibility for bringing plague into western Eurasia from the lands surrounding the Tian Shan, this complex biological web may have also been the circumstance that seeded the Laishevo strain of *Y. pestis*, one SNP away from the Big Bang, into northern Iran, and thence to the Caucasus.

A SERIES OF EVENTS IN THE 1270s and 1280s provides a possible scenario for the northward transmission of Branches 3 and 4. Two generations after Chinggis Khan absorbed the Qara Khitai empire into his own, the main route that armies, emissaries, merchants, and pilgrims took between Mongolia and Almaliq—the center of the Chaghadaid Ulus to the northwest of the Tian Shan range—had barely changed. A route that had once taken travelers to the west of the Tian Shan, and then around the southern rim of the Jungghar Basin (a large desert region), shifted around the time of Chinggis Khan, to rounding the northern rim of the basin instead. In 1275–1276, the land routes in central Asia were still functioning between Talas and Almaliq—and then beyond Almaliq up to Mongolia—and were also improved by the installation of new post stations. That situation changed drastically after the revolt of Shiregi (a son of Möngke Khan, grandson of Chinggis Khan), which broke out in the fall of 1276. The collapse of the Mongol

could satiate a soldier for much of the day. Glutinous millet was also used to brew beer. In other words, this was a grain worth importing, since it could not be obtained locally. See Buell and Anderson, *A Soup for the Qan*, 45, 49–51. For some suggestive hints of consumption of millet (a C4 food that leaves distinctive isotopic traces) as a regular part of the Mongol diet, see Jack N. Fenner, Tumen Dashtseveg, and Khatanbaatar Dorjpurev, “Food Fit for a Khan: Stable Isotope Analysis of the Elite Mongol Empire Period Cemetery at Tavan Tolgoi, Mongolia,” *Journal of Archaeological Science* 46 (2014): 231–244.

⁶² Al-Shīrāzī, *The Mongols in Iran*, 55/86 [34]. Hülegü's sickness is also reported by Rashīd al-Dīn; see John Masson Smith, “Hülegü Moves West: High Living and Heartbreak on the Road to Baghdad,” in Linda Komaroff, ed., *Beyond the Legacy of Genghis Khan* (Leiden, 2006), 111–134, here 122. Hülegü has also been reported getting sick while proceeding to Tun in Spring 1256, and he would be severely ill again later in life (al-Shīrāzī, *The Mongols in Iran*, 61/93 [43]). Aside from something termed “depression,” no symptoms are specific enough to connect his condition to plague.

⁶³ Hymes, “A Hypothesis on the East Asian Beginnings of the *Yersinia pestis* Polytoymy.”

⁶⁴ For Anatolia, al-Shīrāzī reports that one of the next cities to be besieged was Mayyafariqin (Silvan), but he reports no sickness (al-Shīrāzī, *The Mongols in Iran*, 55/86 [34]). Rather, that detail, and reports that Mardin was also afflicted by plague, comes from Bar Hebraeus; see Feda Şamil Arık, “Selçuklular Zamanında Anadolu'da Veba Salgınları” [“The Plagues in Anatolia at the Time of the Seljuks”], *Tarih Araştırmaları Dergisi* 15, no. 26 (1991): 27–57. My thanks to Nükhet Varlık for bringing this study to my attention. Nahyan Fancy and I discuss other chroniclers who attest to outbreaks in Syria in a separate study.

⁶⁵ Reuven Amitai-Preiss, *Mongols and Mamluks: The Mamluk-Ilkhanid War, 1260–1281* (Cambridge, 1995), 22.

garrisons in Almaliq and the abduction of Qubilai Khan's son, Nomuqan, provoked what Mongolist historian Hosung Shim has called a "serious tumult" from Almaliq up to Mongolia via the Altai Mountain range, which lies on the border between present-day Siberia and western Mongolia: precisely where Branch 4 of the *Y. pestis* polytomy is now found (Figure 3).⁶⁶ Could the collapse of the garrison at Almaliq and the revolt led by Shiregi have been due in part to the effects of plague?

Whatever the cause of the tumult, it was serious enough that Qubilai completely changed the postal route connecting the Chaghadaid Khanate with the Yuan court in China. Shim reports that from 1278 on, the Yuan government constructed new postal stations in Uighuristan, the area around Beshbaliq and the eastern part of the modern Xinjiang Autonomous Province, which includes the easternmost spur of the Tian Shan, allowing them to avoid the Altai region. Beshbaliq, in turn, was linked to the Hexi (Gansu) Corridor, and thence to the Yuan court.⁶⁷ At almost the same time, the princes of neighboring groups in Mongolia also rebelled against Qubilai, ostensibly as a result of disloyalty fostered by Qubilai's rival (and nephew) Qaidu. Qubilai's general, Bayan of the Barin, was sent twice to Mongolia to impose order, first in 1277–1278 and again in 1285. As the Yuan lost control over Mongolia, they retreated to Uighuristan (whence the millet for Hülegü's expedition in the 1250s had come). The winter of 1278–1279 was particularly difficult, and it is said that Bayan and his troops, being undersupplied, had to supplement their meals with roots and their clothing with marmot skins. The extent of the "marmot economy" can be seen in this anecdote:

He [Bayan] also ordered that, whenever a soldier caught and ate a *tarbagan*, he should save the skin. When [the skins] reached ten thousand [in number] people were puzzled. Then he dispatched a messenger to bring them to the Capital. The Emperor laughed, saying, 'Po-yen (Bayan), because the borderland is cold and the soldiers have not enough clothing, wants to exchange [this] for our [allotted] fabrics.' As a result the Emperor granted clothing.⁶⁸

Whether any of the marmots Bayan and his troops encountered in Uighuristan were affected by plague is unclear; certainly, there is nothing in this story to suggest that the troops suffered from epidemic disease in addition to severe cold. Yet it tells us about the geographical connections of the period (the abandoning of Almaliq to the northwest of the Tian Shan, trouble in the Altai and Mongolia, and the connections to the Yuan court via Gansu) as well as the kinds of human-driven environmental disruptions that, had *Y. pestis* been present, could contribute to the large-scale spread of the pathogen.

THE COMBINED EVIDENCE OF phylogenetics and paleogenetics has demonstrated conclusively that the pandemic strains of *Yersinia pestis*, those involved in the Justinianic Plague and then in the Black Death(s), emerged from what must have been the ancestors among the Branch 0 strains. The living descendants of Branch 0 are currently

⁶⁶ Hosung Shim, "The Postal Roads of the Great Khans in Central Asia under the Mongol-Yuan Empire," *Journal of Song-Yuan Studies* 44 (2014): 405–469.

⁶⁷ *Ibid.*

⁶⁸ Francis Woodman Cleaves, "The Biography of Bayan of the Bārin in the *Yüan shih*," *Harvard Journal of Asiatic Studies* 19, no. 3/4 (1956), 185–303, here 263–265. See also C. C. Hsiao, "Bayan (1237–1295)," in Igor de Rachewiltz et al., eds., *In the Service of the Khan: Eminent Personalities of the Early Mongol-Yuan Period (1200–1300)* (Wiesbaden, 1993), 584–607.

found in or near the Tian Shan range at the modern border between the Uyghur Autonomous Province and Kyrgyzstan; this is not in and of itself proof of the medieval whereabouts of Branch 0, but it provides data from which further inquiries could build. Further, the centrifugal pattern of dissemination of the four documented post-polytomy lineages of *Y. pestis*—the four “Black Deaths”—together with the geographically restricted survival of the most recent pre-polytomy lineages (0.ANT2/0.ANT5 and 0.ANT3) points to a region in or near the Tian Shan as the possible site of the Big Bang. In terms of the Big Bang’s timing, not only does “molecular clock” analysis point to a chronological range that would encompass the period of Mongol dominance of central Asia, but the pattern of geographic spread of the post-polytomy lineages also accords with the emergence of the Mongols out of Mongolia in 1206; their conquest of the Qara Khitai empire by 1218; and their military expeditions into China, central Asia, Rus’, Iran, and Iraq between 1218 and 1258. Furthermore, I have suggested that the revolts that disrupted the region from Almaliq up to the Altai and Mongolia itself in the 1270s and 1280s may have been accompanied and exacerbated by plague. Although definitive evidence linking these bacterial and human events together is still lacking, the linkages are biologically possible. As circumstantial evidence, the biological and documentary archives collectively point to the thirteenth century, not the fourteenth, as the beginning of the Four Black Deaths.⁶⁹

Even as circumstantial evidence, these findings suggest that it is time to set aside assumptions that have been at the foundation of plague historiography for more than a century: that the Black Death involved either a single continent-wide, east-to-west sweep of plague across Eurasia, or that it was a limited outbreak confined to western Eurasia. Both scenarios are assumed to have happened within the fourteenth century. The biological archive insists not simply that we broaden our geography and chronology of plague’s history; to track a disease as complicated in its ecology as plague requires us also to reconstruct the circumstances that would have allowed its long-distance transmission. Despite its moniker, the Big Bang may have been a quiet event, largely unnoticed by human populations amid the far more visible chaos caused by the early Mongol conquests and the repeated displacements of peoples across central Eurasia. But amid that visible chaos, much may have been happening at a microbial level. And happening at great speed: the polytomy is not a story of the creeping spread of disease, brought by wave after wave of traveling merchants making their way along the too-often-invoked “Silk Road” during an alleged *Pax Mongolica*. Least of all is it a story of “rodents on the march” across vast landscapes.⁷⁰ Such scenarios can explain

⁶⁹ There is growing need to revisit the reported incidents of *wabā’* and *ṭā’ūn* in Arabic sources that fall between the conventional end-date of the Justinianic Plague (ca. 750 C.E.) and the Black Death. Many of these are listed in A. von Kremer, *Grossen Seuchen des Orients nach arabischen Quellen* (Vienna, 1880), and more could be cited. On a case that seems to be plague in Ethiopia in or before 1242, see Nahyan Fancy, “Knowing the Signs of Disease: Plague in Arabic Medical Commentaries Between the First and Second Pandemics,” in Lori Jones and Nükhet Varlık, eds., *Death and Disease in the Medieval and Early Modern World* (York, 2021, forthcoming).

⁷⁰ The analysis I am presenting here differs diametrically from that proposed, most recently, by Amine Namouchi, Meriam Guellil, Oliver Kersten, Stephanie Hänsch, Claudio Ottoni, Boris V. Schmid, Elsa Pacciani, Luisa Quaglia, Marco Vermunt, Egil L. Bauer, Michael Derrick, Anne Ø. Jensen, Sacha Kacki, Samuel K. Cohn Jr., Nils C. Stenseth, and Barbara Bramanti, “Integrative Approach Using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period,” *PNAS* 115, no. 50 (December 11, 2018): E11790–E11797. Namouchi and colleagues posit repeated waves of plague imported into Europe from the time of the Black Death until plague’s eventual disappearance in the eigh-

local spread of the disease, as with a recent, closely analyzed case that used genetics to show how plague moved through a herding community in China between marmots, sheep, dogs, and humans.⁷¹ But such slow, sequential transmissions leave their mark in the genome.⁷² No, the evidence of genetics suggests that, at least in the thirteenth century, plague moved with sudden rapidity, only to burrow into new host populations where its novelty, virulence, and isolation allowed it to flourish anew.

If the phylogeography of *Yersinia pestis*—the genetic history of the organism plotted onto a map—paints such a compelling picture of a centrifugal plague phenomenon starting in the realm of the Qara Khitai in the early thirteenth century, why has our historiography been so unsuccessful, to date, in seeing it? The biggest reason is the one stated at the beginning of this paper: no recorder of human history prior to the late nineteenth century could have factored bacteria into their understanding of events they witnessed or heard about from their contemporaries. Still, the Black Death (as traditionally defined as a Mediterranean/European phenomenon) is so abundantly *visible* in our sources from western Eurasia that the contrasts—the absences, the silences—in the rest of the continent are puzzling. Admittedly, the account of Quṭb al-Dīn al-Shīrāzī was only retrieved for historical analysis a few years ago, and Hymes’s detailed reconstruction of the evidence for plague in Song China is also quite recent. But how could the (alleged) role of plague in a crisis of such profound consequence for world history as the Fall of Baghdad have gone so long unnoticed?⁷³

To these questions, I would answer, first, that perhaps it is time to stop taking the

teenth century. At most, the palaeogenetic evidence published thus far supports, in my reading, only two introductions into Europe: the Black Death, in 1347 (see Spyrou et al., “A Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes”); and Giffin et al., “A Treponemal Genome from an Historic Plague Victim Supports a Recent Emergence of Yaws and Its Presence in 15th Century Europe”), and the *pestis secunda* strain, in 1358 or 1359, which (if it is not of European origin) may have come into western Europe via a northern route (Green, “Putting Africa on the Black Death Map”). I suspect that the broad dissemination of Branch 2’s sublineages, 2.ANT and 2.MED, paint a somewhat different story, one that takes us into the early modern period.

⁷¹ Dai et al., “Human Plague Associated with Tibetan Sheep Originates in Marmots.” This case involved a local strain of 1.IN, the lineage of Branch 1B that migrated across central Asia from Russia, sometime between the fifteenth and seventeenth centuries.

⁷² That same study, comparing samples collected over a period of thirty years, captured over two hundred SNPs in thirty-six isolates. Dai et al., “Human Plague Associated with Tibetan Sheep Originates in Marmots,” S3 Table. This is probably a normal level of genetic change. As explained above, what matters historically is which SNPs survive to create new lineages. Most mutations, as was likely the case in this study, die with their hosts. On this phenomenon of purifying selection, see Mark Achtman, “Insights from Genomic Comparisons of Genetically Monomorphic Bacterial Pathogens,” *Philosophical Transactions of the Royal Society B: Biological Sciences* 367, no. 1590 (2012): 860–867.

⁷³ Mona Hassan, *Longing for the Lost Caliphate: A Transregional History* (Princeton, 2017), 47 and 279 n. 94, lists plague as a compounding factor in the devastation of Baghdad, citing six historians from the thirteenth to fifteenth centuries. Thanks to the generous investigations of Nahyan Fancy and Matthew Melvin-Koushki, I have been able to assess the content of these works to confirm the references to “epidemics/plague.” We found that only three identified an epidemic (*wabā’*, not specifically “plague,” *tā’ūn*) at Baghdad: *Hawādith al-jāmi’ a*, by an anonymous Baghdad writer working at least as late as 1301; Muhammad Ibn Shākir al-Kutubī’s (d. 1363) *Uyūn al-Tawārīkh* (written before 1363 in Damascus); and Ibn Kathir’s (d. 1373) *Bidayya wa-l-nihaya*. The other sources refer to *wabā’* and/or *tā’ūn* in Damascus or Aleppo specifically, or in Syria generally, but not in Baghdad. Nahyan Fancy and I address the significance of this distinction in another study. Al-Shīrāzī’s account is not cited in Hassan’s study, which was based on a 2006 Ph.D. dissertation written prior to the publication of al-Shīrāzī’s Persian account in 2010. However, see also Michal Biran, “Violence and Non-Violence in the Mongol Conquest of Baghdad,” in Robert Gleave and István Kristó-Nagy, eds., *Violence in Islamic Thought from the Mongols to European Imperialism* (Edinburgh, 2018), 15–31, who cites, and therefore was well aware of, al-Shīrāzī’s account.

urban European experience of the Black Death as the model against which all manifestations of plague must conform. The narratives now coming from both genetics and documentary history confirm that plague's arrival in the Black Sea and the Mediterranean was swift; although the mechanisms of onward transmission remain to be determined, the initial spread of plague, in 1347–1348, was clearly a function of maritime transport and urbanized networks of trade. In contrast, it is likely that plague outbreaks in Mongol areas were only rarely *epidemic*, let alone *pandemic*. At least in the thirteenth century, this was still by and large a nomadic culture. If plague reached a Mongol encampment—a veritable city on wheels—the inhabitants could pull up stakes and move out of landscapes that had become morbid with plague. That may, in fact, be what Hülegü himself did in the spring and summer of 1257, when he is known to have moved camp at least five times independent of any military operations.⁷⁴ Of the plague-like events that are recorded in some fashion, these seem to have struck both Mongol and non-Mongol alike.

Additionally, we may have been looking for the wrong symptoms. Plague has different manifestations depending on its route into the body. Gastrointestinal plague is the least researched of all modes of ingress. But a study of a case of ingested camel meat provides a list of symptoms: fever, vomiting, diarrhea, and in some cases pharyngeal lesions and lymphadenitis.⁷⁵ Only the last (swollen lymph glands, presumably in the neck) would echo the buboes we have become accustomed to look for in traditional accounts of plague. For the rest, the symptoms echo those of poisoning, and Mongol sources certainly record the latter.⁷⁶

Surely, if we are looking for epidemics of massive mortality as our measure, the scale of devastation at Kaifeng in 1232 (as described by Li Gao) or at Baghdad and surrounding areas in 1258 (as implied by Qutb al-Dīn al-Shīrāzī and other sources) could rival any Black Death account we have from Europe or the Islamic Mediterranean.⁷⁷ But the fact remains that Mongol sources record nothing *systematically* catastrophic for the thirteenth century in the way that western Eurasian sources will do at the onset of the Black Death. There is no Mongol equivalent of an Ibn al-Wardī, an Ibn Khatīma, or

⁷⁴ Smith, "Hülegü Moves West," 124.

⁷⁵ Leslie et al., "Outbreak of Gastroenteritis Caused by *Yersinia pestis* in Afghanistan"; in this case, the infected meal was a camel. See also Jan Kehrmann, Walter Popp, Battumur Delgermaa, Damdin Otgonbayar, Tsagaan Gantumur, Jan Buer, and Nyamdorj Tsogbadrakh, "Two Fatal Cases of Plague after Consumption of Raw Marmot Organs," *Emerging Microbes and Infections* 9, no. 1 (2020): 1878–1880, <https://doi.org/10.1080/22221751.2020.1807412>. Michelle Ziegler, "The Black Death and the Future of the Plague," *The Medieval Globe* 1–2 (2014): 259–284, comments on the unrecognized significance of gastrointestinal plague.

⁷⁶ For example, George Lane, *Early Mongol Rule in Thirteenth-Century Iran: A Persian Renaissance* (New York, 2003), 49, 101, 141, 156, and 174.

⁷⁷ I have not attempted in this essay to assess mortality levels or case fatality rates, but it would be well to point out that the early strains of each of the four post-polytomy lineages would have been virtually identical to the others genetically, meaning they likely had comparable virulence in humans. If strains very similar to the (western) Black Death strain reached the cities of central Asia and eastern China, we can well imagine comparable effects to what Ibn al-Wardī described in Aleppo, what Boccaccio described in Florence, what Henry Knighton described in England, and what Gilles li Muisit described in Tournai. Li Gao (per Hymes, "A Hypothesis on the East Asian Beginnings of the *Yersinia pestis* Polytomy") reports fatalities numbering in the thousands during the epidemic in Kaifeng in 1232, and his report echoes quite plausibly the level of devastation documented by Borsch and Sabraa for a number of cities in the Middle East, which most likely would have been struck by the same strain that hit western Europe. See Stuart Borsch and Tarek Sabraa, "Refugees of the Black Death: Quantifying Rural Migration for Plague and Other Environmental Disasters," *Annales de démographie historique* 134, no. 2 (2017): 63–93.

a Boccaccio, who presents a narrative of plague's origin or path; there is no thirteenth-century chronicler of plague as a pandemic disease. Then again, none of the western chroniclers of the fourteenth-century Black Death were facing a simultaneous onslaught of plague and Mongol attackers. A wealthy musician in Baghdad in 1258, for example, who, like some other chroniclers of the sack, makes no mention of "pestilence" as a factor, had clearly hoarded his own food supplies, saving him from reliance on food the Mongols brought with them. Concerned only to save his family and his wealthy quarter of the city, he may have had little awareness of (or desire to see or remember) the devastation happening elsewhere.⁷⁸

The evolutionary understanding of *Yersinia pestis*'s transmission into western and central Asia that I have proposed here, building on Hymes's evidence for the spread of plague into Song China, allows us to assess the connection between the spillover and seeding events of the thirteenth century, and the explosive fourteenth-century proliferation of plague in old haunts (the Chaghadaid Khanate and Yuan China) and new (the Black Sea and Mediterranean basins). My proposal is falsifiable, should aDNA demonstrate the presence of specific strains of *Y. pestis* at times or places that contradict the scenarios I have proposed. In the meantime, if all four Black Deaths are to be integrated into the history of the Mongol Empire and the global history of disease, the challenge ahead is to reinterrogate the multiple episodes of structural breakdown in Mongol territories that have thus far been described as "political," particularly the chaos in the Chaghadaid Khanate in the 1270s and 1280s, the collapse of the Ilkhanate in the 1330s, and what has been called "total chaos" in the Chaghadaid Khanate (again) as it fell apart in the 1340s.⁷⁹ David O. Morgan, an expert on the Ilkhanate, famously suggested that polity, "in barefaced defiance of 'Gibbon's Law,' fell without having previously declined." But Morgan did not believe plague was a problem in Iran and the Caucasus.⁸⁰ That assumption should now be challenged: the Ilkhanate collapsed right when plague was likely reemerging in the Caucasus in the 1330s, and the Golden Horde likewise collapsed in the Volga region upon a second wave of plague in 1359.⁸¹ As Marie-Laure Derat has recently noted with respect to late medieval Ethiopia (where plague is also reported in the Muslim sultanate of Sawa [Shewa] in the later thirteenth century), perhaps these sequences of short reigns and structural chaos hide the fact that

⁷⁸ Michal Biran, "Music in the Mongol Conquest of Baghdad: Šafī al-Dīn Urmawī and the Ilkhanid Circle of Musicians," in Bruno De Nicola and Charles Melville, eds., *The Mongols' Middle East: Continuity and Transformation in Ilkhanid Iran* (Leiden, 2016), 133–154.

⁷⁹ This depiction of the end of the Chaghadaid Khanate comes from Shim, "The Postal Roads of the Great Khans in Central Asia under the Mongol-Yuan Empire," 457. Shim is a rare Mongolist who addresses the presence of plague, but he places its origin in Mongolia itself and suggests that it broke out for the first time in 1331–1332, being entirely a phenomenon of the fourteenth century (458). In contrast, plague is never mentioned in Charles Melville, "The End of the Ilkhanate and After: Observations on the Collapse of the Mongol World Empire," in Bruno De Nicola and Charles Melville, *The Mongols' Middle East*, 307–335. Jackson, *The Mongols and the Islamic World*, as noted above, is a rare study that attempts to wrestle with plague's possible presence, though as noted, he gives up, deeming the question insoluble.

⁸⁰ David Morgan, "The Decline and Fall of the Mongol Empire," *Journal of the Royal Asiatic Society* 19, no. 4 (2009): 427–437.

⁸¹ Uli Schamiloğlu, "The Impact of the Black Death on the Golden Horde: Politics, Economy, Society, and Civilization," *Zolotoordinskoe obozrenie [Golden Horde Review]* 5, no. 2 (2017): 325–343. Valuable primary sources have been identified for further investigation in Timur Khaydarov, "Epidemics and Mass Famine in Russia in the Territory of Rus' and the Golden Horde (10th–the First Half of the 14th Century)" [in Russian], *Туркологические исследования [Turkological Studies]* 1, no. 2 (2018): 73–100.

people were dying at unprecedented rates.⁸² At the other end of the Mongol Empire, Timothy Brook confirms the likelihood that plague played a precipitating role in the collapse of the Yuan dynasty.⁸³ Severe social disruption may itself be a signature of plague that should prompt us to investigate further.⁸⁴

That investigation may push us beyond the biological archive altogether to engage with the geologic. One factor of the Baghdad siege may have distinguished it from all others before or after. In 1257, the earth witnessed one of the largest volcanic explosions of the Holocene. The volcano Samalas, in Indonesia, ejected some forty cubic kilometers of rock and hundreds of megatons of volcanic gases into the atmosphere, with worldwide effects. But whereas other ultra-Plinian volcanic eruptions have resulted in pronounced cooling phenomena, Samalas's effects on global temperatures seem to have been minimal. It may have contributed to ozone depletion and increased ultraviolet exposure, thereby producing long-term effects such as higher incidence of skin cancers. Whether there may have also been effects on the plague bacterium or its rodent hosts is as yet unknown.⁸⁵ Nevertheless, it is notable that winds or "stench" are reported in Baghdad, Damascus, and even Tunisia by both contemporaries and later commentators; moreover, there is a notable rise in Arabic sources in this period of appeals to miasmas as the source of plague.⁸⁶ Little would come of this new train of thinking about plague since, after the events in Baghdad and Syria in 1258, plague gradually disappeared from current awareness in western Asia. It was only three generations later, after the eyewitnesses to the events of the 1250s were dead, that a new experience with plague would raise these questions anew.

In 2012, it was predicted that new research in paleogenetics would likely produce evidence for disease events "for which we lack a good historical record (or any awareness at

⁸² On Ethiopia, see Derat, "Du lexique aux talismans," para. 8.

⁸³ Brook, *Great State*.

⁸⁴ Sara Nur Yıldız, "Mongol Rule in Thirteenth-Century Seljuk Anatolia: The Politics of Conquest and History Writing, 1243–1282" (Ph.D. diss., University of Chicago, 2006), for example, noted (without being able to give an explanation for it) the odd replacement of the Mongol commanders assigned to the campaign against Anatolia right after the sack of Baghdad where, as noted above, plague also broke out among the Mongol soldiers (pp. 291, 296, and 353). (My thanks to Nükhet Varlık for bringing this study to my attention.) Likewise, the anonymous Baghdad text *Kitāb al-Hawādīṭ* notes three commanders who died very soon after being given new assignments in mid-1258; see Gilli-Elewy, "*Al-Hawādīṭ al-ḡāmi'a*," 369.

⁸⁵ The potential connection between the Big Bang and the 1257 Samalas explosion, which ranks as a 7 on the 8-level scale of volcanic explosivity, has been debated for several years. Campbell has concluded that Samalas's immediate effects were likely negligible in *The Great Transition* and Bruce M. S. Campbell, "Global Climates, the 1257 Mega-Eruption of Samalas Volcano, Indonesia, and the English Food Crisis of 1258," *Transactions of the Royal Historical Society*, 6th ser., 27 (2017): 87–12. Henry Gillies Fell, "Contribution of Volcanism to the Initiation of Plague Pandemics" (M.A. thesis, Durham University, 2018), suggests that the unusual intensity of that event may have itself caused the Big Bang. Martin Bauch, "Chronology and Impact of a Global Moment in the Thirteenth Century: The Samalas Eruption Revisited," in Andrea Kiss and Kathleen Pribyl, eds., *The Dance of Death in Late Medieval and Renaissance Europe: Environmental Stress, Mortality and Social Response* (New York, 2019), 214–232, collects various data on possible effects. David C. Wade, Céline M. Vidal, N. Luke Abraham, Sandip Dhomse, Paul T. Griffiths, James Keeble, Graham Mann, Lauren Marshall, Anja Schmidt, and Alexander T. Archibald, "Reconciling the Climate and Ozone Response to the 1257 CE Mount Samalas Eruption," *PNAS*, first published October 12, 2020, <https://doi.org/10.1073/pnas.1919807117>, confirms the limited effects on temperature, but raises the question of ozone depletion. For other evidence of climatic disruptions in this period, see Tana Li, "The Mongol Yuan Dynasty and the Climate, 1260–1360," in Martin Bauch and Gerrit Jasper Schenk, eds., *The Crisis of the 14th Century: 'Teleconnections' between Environmental and Societal Change?* (Berlin, 2019), 153–168.

⁸⁶ This evidence is assembled in a new study by Nahyan Fancy and myself.

all).⁸⁷ As I noted in 2014, the scientists making this prediction had exempted the Black Death, thinking its story already well documented. Yet even that oft-told narrative has now been transformed. The combined approaches of evolutionary genetics—working from modern isolates of *Yersinia pestis* and the retrieved genetic fragments of the bacterium reclaimed from its premodern victims—have given new parameters to the history of plague. The question “What was the disease?” has been set aside, replaced by questions this new method is uniquely equipped to answer. What *strains* were involved in particular outbreaks? To which other strains are they most closely related, and when can these be documented? Currently, the biological archive—which has now yielded over three dozen complete *Yersinia pestis* genomes in evidence of Europe’s late medieval and early modern experience of plague—supports the idea that one specific strain of *Yersinia pestis*, only two SNPs removed from the Big Bang, entered the Black Sea and the Mediterranean, and from there into Europe, in 1347–1348. In other words, aside from Gabriele de Mussi’s fiction about the siege of Caffa, the biological archive provides support for both the traditional narratives about the western Eurasian experience of the Black Death and the new evolutionary history of *Y. pestis*. Plague was indeed suddenly and terrifyingly *visible* in the Ilkhanate, the Golden Horde, the Black Sea, and the Mediterranean in the 1330s and 1340s. The climate crises and grain shortages of the early fourteenth century may well explain the intensity of that outbreak, independent of any particular virulence of the early Branch 1 strains. But the (west Eurasian) Black Death, as traditionally defined, was preceded by the terrors experienced at the sieges in Song China and western Asia in the thirteenth century. Although recorded by Li Gao, Quṭb al-Dīn al-Shīrāzī, and a handful of other sources, these early plague experiences did not cohere into narratives that passed into modern historiography; rather, they were subsumed or even buried within narratives of the Mongol attacks. The new reservoirs of *Y. pestis* that were likely seeded in China, Mongolia, Siberia, and the Caucasus regions in the thirteenth century by the Mongol conquests were forgotten, or never perceived by human eyes.

Paleogenetics investigations into the evolutionary history of *Yersinia pestis* will clearly continue, and may soon yield evidence for other regions of western, central, and eastern Asia (and even Africa) that seem to have been afflicted by plague in the late Middle Ages.⁸⁸ The instigating circumstances of Branch 2’s two-pronged proliferation in and through the Inner Asian Mountain Corridor remain to be investigated.⁸⁹ Already

⁸⁷ D. M. Altmann, F. Balloux, and R. J. Boyton, “Diverse Approaches to Analysing the History of Human and Pathogen Evolution: How to Tell the Story of the Past 70,000 years,” *Philosophical Transactions of the Royal Society of London B: Biological Sciences* 367, no. 1590 (2012): 765–769, as cited in Green, “Taking ‘Pandemic’ Seriously,” 30.

⁸⁸ There are many older field studies of plague conducted in Africa and Eurasia, but unless they employ the methods of genotyping pioneered by Achtman and others from 2004 on (see the online supplementary material, “Marmots and Their Plague Strains”), they cannot be used for the kind of evolutionary analysis employed here. The in-progress CARTO-PEST project, referenced above, will quickly expand the number of sequences available. Additionally, it will likely be possible to track both millet production and millet consumption through studies of the distinctive C4 isotopic signatures millet leaves in human and animal bones. See Taylor R. Hermes, Michael D. Frachetti, Paula N. Doumani Dupuy, Alexei Mar’yashev, Almut Nebel, and Cheryl A. Makarewicz, “Early Integration of Pastoralism and Millet Cultivation in Bronze Age Eurasia,” *Proceedings of the Royal Society B: Biological Sciences* 286, no. 1910 (2019): 2019.1273.

⁸⁹ What is generally called the Third Plague Pandemic, usually dated from the mid-nineteenth to the mid-twentieth century, involved strains that issued from the Yunnan Province in southwest China; these, in turn, were the descendants of what is referred to above as Branch 1B. See Giovanna Morelli, Yajun Song, Camila J. Mazzoni, Mark Eppinger, Philippe Roumagnac, David M. Wagner, Mirjam Feldkamp, Barica Kusecek, Amy J. Vogler, Yanjun Li, Yujun Cui, Nicholas R. Thomson, Thibaut Jombart, Raphael

underway are efforts to assess the biological pressures this organism has placed on the human populations continually exposed to it, and to track the rodent populations involved in its transmission.⁹⁰ The historian, working with documentary sources, will need to track the humans who are now implicated in plague's spread. In so doing, historians would do well to adopt epidemiologists' neutral stance toward the task of tracking infectious disease: this is not about assigning "blame."⁹¹ It is about documenting humans doing what humans do.

Leblois, Peter Lichtner, Lila Rahalison, Jeannine M. Petersen, François Balloux, Paul Keim, Thierry Wirth, Jacques Ravel, Ruifu Yang, Elisabeth Carniel, and Mark Achtman, "Yersinia pestis Genome Sequencing Identifies Patterns of Global Phylogenetic Diversity," *Nature Genetics* 42, no. 12 (2010): 1140–1145. There was, however, other plague activity in the late nineteenth and twentieth century, caused by Second Pandemic strains still active in central Asia (2.MED) and East Africa (1.ANT).

⁹⁰ Yong Hwan Park, Elaine F. Remmers, Wonyong Lee, Amanda K. Ombrello, Lawton K. Chung, Zhao Shilei, Deborah L. Stone, Maya I. Ivanov, Nicole A. Loeven, Karyl S. Barron, Patrycja Hoffmann, Michele Nehrebecky, Yeliz Z. Akkaya-Ulum, Erdal Sag, Banu Balci-Peynircioglu, Ivona Aksentijevich, Ahmet Gül, Charles N. Rotimi, Hua Chen, James B. Bliska, Seza Ozen, Daniel L. Kastner, Daniel Shriner, and Jae Jin Chae, "Ancient Familial Mediterranean Fever Mutations in Human Pyrin and Resistance to *Yersinia pestis*," *Nature Immunology* 21, no. 8 (2020): 857–867; Emily E. Puckett, David Orton, and Jason Munshi-South, "Commensal Rats and Humans: Integrating Rodent Phylogeography and Zooarchaeology to Highlight Connections between Human Societies," *BioEssays* 42, no. 5 (2020): <https://doi.org/10.1002/bies.201900160>.

⁹¹ Richard McKay, "Patient Zero: Why It's Such a Toxic Term," *The Conversation*, April 1, 2020, <https://theconversation.com/patient-zero-why-its-such-a-toxic-term-134721>.

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