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Putting Africa on the Black Death map: Narratives from genetics and history

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Putting Africa on the Black Death map: Narratives from genetics and history

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Monica H. Green

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I. Introduction

- 1 Because of a recent revolution facilitated by genetics, the field of microbiology has presented to the field of historical epidemiology a great boon: it has reconstructed the outlines of the evolutionary history of *Yersinia pestis*, the causative organism of the infectious disease, plague, which was perhaps the agent of more acute epidemic deaths than any other infectious organism in human history. This is a unified, global history,

stretching from the steppes of Bronze Age Eurasia, through the late medieval pandemic known as the Black Death, up to outbreaks of plague still seen today on four of the five inhabited continents. Any sample of *Y. pestis*, wherever it is found and whatever its date, can now be fitted into an integrated history of the organism, a narrative that shows the relations of that one sample to every other one that has ever existed.¹

- 2 It is customary now to identify three extended pandemics in plague history. The First Plague Pandemic is also known as the Justinianic Plague and is usually dated from 541 CE to ca. 750 CE, even though it is clear now that it had earlier genetic antecedents.² The Second Plague Pandemic encompasses the Black Death of the mid-14th century as it is usually described in historical literature, which sees it as a massively lethal plague outbreak that struck the Black Sea, the Mediterranean Basin, and adjacent landmasses between 1346 and 1353. However, as will be explained here, the Second Plague Pandemic should now be understood as an epidemiological event starting in the 13th century and continuing to the present day—encompassing not simply the Mediterranean littoral and Europe, but much of Eurasia and, as we will see, Africa. The Third Plague Pandemic began with new plague activity in southeast China in the 17th or 18th century. These new strains then emerged out of Yunnan Province in the mid-19th century and spread globally via steamships, starting in 1894 out of Hong Kong. The Third Plague Pandemic also has many still-living descendants, accounting for all of plague's new introduction into the Americas, Australia, and Madagascar.
- 3 The genetics of *Y. pestis* ties all these stories together. This universal narrative is as powerful in its implications for the history of Africa as it is for plague anywhere else in the world. This is true despite the fact that no plague aDNA—"ancient DNA", molecular fossils retrieved from pre-modern remains—has thus far been retrieved for *Y. pestis* from the African continent. For medieval and early modern Europe, the retrieval and, by 2011, complete sequencing of *Y. pestis* aDNA has been transformative. Samples of *Y. pestis* have now been retrieved from plague outbreaks from the 14th through the 18th centuries in Europe.³ These confirm not simply that, yes, *Y. pestis* did indeed play a role in the major devastations that Europe suffered at the time of the Black Death and in subsequent outbreaks. More importantly, these studies also prove that plague's many different strains can be tracked back into the past, creating—just as a modern epidemiologist would do—a dynamic map of where and when different strains struck different populations. That work is important not simply for Europe itself, but also for other parts of the world since, as has now been documented, plague has moved historically both trans- and intercontinentally.
- 4 For Africa, few modern *Y. pestis* genomes have been fully sequenced and, as noted, no historical traces of the plague bacillus have been recovered.⁴ But such studies as have been published show that Africa harbors strains of plague that entered the continent in different historical periods. Thus, for example, strains found in Madagascar, South Africa, Senegal, and Morocco clearly belong to Third Pandemic strains, which issued out of Hong Kong at the end of the 19th century.⁵ Samples found in Libya have been identified with the 2.MED strains (Branch 2), most of which are found in Eastern and Central Eurasia, and might possibly date from a period when the region was under Ottoman control.⁶ The strains found from the 1940s to the present day in Kenya, Uganda, the Democratic Republic of Congo, and Zambia—that is, the areas surrounding the Great Lakes District—are distinct from the foregoing. That subgroup, called 1.ANT, is not modern at all. Rather, 1.ANT can be shown to be a descendant of the strain now

known to have caused the largest pandemic in human history, the 14th-century Black Death. The genetics, therefore, very firmly puts Africa on the map of the Second Plague Pandemic, a role only barely recognized in prior histories of plague and not recognized at all in prior histories of the East African environment or cultural development.⁷

- 5 In 2014, I published an essay arguing that the new evolutionary history of *Y. pestis* recently forged by geneticists called for a complete rethinking of plague's global history. I worked through the implications of the genetics narrative as it was then understood from aDNA and phylogenetic analysis of modern isolates to argue that the *Y. pestis* sub-branch 1.ANT was the oldest living strain of plague to have taken its origin after the Black Death in the mid-14th century. Specifically, I suggested that it may have arrived in East Africa as early as the 15th century. It was, in other words, a legitimate part of the story of the Black Death, even though Sub-Saharan Africa had never previously been mentioned in Black Death narratives. I suggested, furthermore, that it might have arrived via the Indian Ocean littoral, though I demurred from suggesting a particular geographic origin of the transmission.⁸ In 2016, historian George Sussman, making no mention of my 2014 study, suggested instead that plague had not arrived in East Africa until the 19th century. He suggested, moreover, that plague had come to East Africa from India. As he put it:

the most likely explanation for the presence of the plague in the Great Lakes region of Central Africa before the third pandemic is infective fleas from Gujarat [western India] trapped in bundles of cloth in the early to mid-nineteenth century, transported by Indian merchants to Zanzibar and carried by porters led by Arab merchants inland.⁹

- 6 The differences in these two interpretations have major implications for African history, since they connect the continent to Eurasia in very different ways and posit significantly different chronologies for the entrance into East Africa of one of the most lethal infectious diseases in the world. My scenario linked East Africa to phenomena of the later Middle Ages, tying the transmission of plague into Africa directly to the Black Death as a Mediterranean or Middle Eastern phenomenon and the economic and demographic disruptions of that period. Sussman, in contrast, by shifting the focus to the 19th century and the context of British colonialism in India and East Africa, made plague's intrusion into East Africa a function of very different forces. From the perspective of African history itself, the two interpretations imply drastically shorter or longer periods during which the peoples of East Africa would have been wrestling with plague as a disease threat.
- 7 Since those two studies appeared, additional genetics work has been published that helps refine the story of the 1.ANT lineage further. I argue here that this new evidence, together with a re-reading of documentary evidence that was already available, complicates both my earlier interpretation and that of Sussman. Although the dating of its arrival remains to be confirmed, the evidence from both genetics and circumstantial historical accounts suggests that plague came to East Africa through the Middle East and not from India or, as had earlier been proposed, China.
- 8 I offer both a semi-global and a consilient approach that asks that Africa be integrated into Eurasian history and that multiple kinds of evidence and methodologies be drawn on to produce a coherent (or at least not openly conflicting) narrative.¹⁰ The argument is laid out in four stages. First, in Part II, I make the general argument (A) that the evolution of organisms—in this case, a pathogenic bacterium—is a kind of history and therefore can be used by historians for historical argument. A brief account of the

development of the discipline of genetics as it relates to the evolutionary history of *Y. pestis* is offered. I then argue more specifically for the relevance of the results of plague science to history. Because they carry distinct molecular signatures, different strains of plague create, in effect, “archaeological” layers that can be separated and studied sequentially. Because plague is now a globally distributed disease, evidence can be drawn from a global pool of data. Thus, even if they do not bear absolute time-stamps, these archaeological layers allow relational (B) geographic and (C) chronological arguments to be constructed. In the present case, the global evolutionary history of *Y. pestis* not simply allows an outline of the African strain’s early history to be sketched, but it also permits certain earlier, unsubstantiated hypotheses to be ruled out. In this case, it is shown that it is implausible, perhaps even impossible, that 19th-century India could have been the source of the strains of plague now found in East Africa.

- 9 In Part III, I then turn to several questions specifically about the history of plague in Africa. First (A), I suggest some ways that a new understanding of the late medieval, or possibly early modern strain that is of most interest here, the distinctive sub-branch of plague in East Africa, 1.ANT, can be informed by what is known of an earlier, though distinct, African experience with plague during Late Antiquity (the Justinianic Plague). Then (B), I examine the Second Plague Pandemic, whose most devastating episode was the 14th-century Black Death, as an epidemiological phenomenon. How badly and how extensively the African continent was hit in the mid-14th century is a question other contributions to the present volume will address. 1.ANT, the strain of concern here, is a somewhat later offshoot of the strain of *Y. pestis* that struck Central Eurasia, the Middle East, and Western Europe. This strain stands near the head of the *Y. pestis* Branch 1 lineage, a family of strains created in the “Big Bang”, a sudden evolutionary divergence in the later Middle Ages that pushed *Y. pestis* into at least four new geographic locales: the Caucasus, Mongolia, Siberia, and China and Tibet. I suggest that the ancestor of the 1.ANT strains emerged from a probable reservoir of plague that had been newly established in the region of the Caucasus, then under Mongol rule, in the 13th century. In section (C), I ask *how long* this Black-Death derived lineage has been present in East Africa. Finally, in section (D), I turn to the related question: *by what route* did this central Eurasian strain reach equatorial East Africa?
- 10 For these latter two questions—the timing of 1.ANT’s entry into equatorial East Africa and the route by which it entered—I can offer no definitive answers, only informed suppositions. But this exercise in circumscribing the timing and geography of what may have been major environmental upheavals should establish definitively that the experience of East Africa needs to be factored into all future narratives of the Second Plague Pandemic. Conversely, this analysis will suggest that plague now needs to be factored into the social and perhaps even political and economic history of early modern equatorial East Africa.

II. Evolution as history: The evidence from genetics

- 11 The bacterium *Y. pestis* developed its current genetic character, including its extraordinary lethality, in Central Eurasia.¹¹ Diverging about 28,000 years ago from a relatively harmless pathogen found in soil and water called *Yersinia pseudotuberculosis*, *Y. pestis* likely first acquired the ability to cause fulminating infections in the lungs of mammalian hosts thousands of years ago. Later, by about 6,000 years ago, it became

transmissible by fleas, thereby entering the body of its mammalian hosts via the lymphatic system and causing the swollen and infected lymph nodes we now associate with its most characteristic manifestation, bubonic plague. Ancestral strains of *Y. pestis* have been found in Bronze Age Eurasia dating back to the 3rd millennium BCE.¹² Similarly, extant strains very closely related to what must have been the progenitors of both the First and Second Plague Pandemics can still be found in the mountain ranges at the borders of what are now eastern Kyrgyzstan and western China.¹³

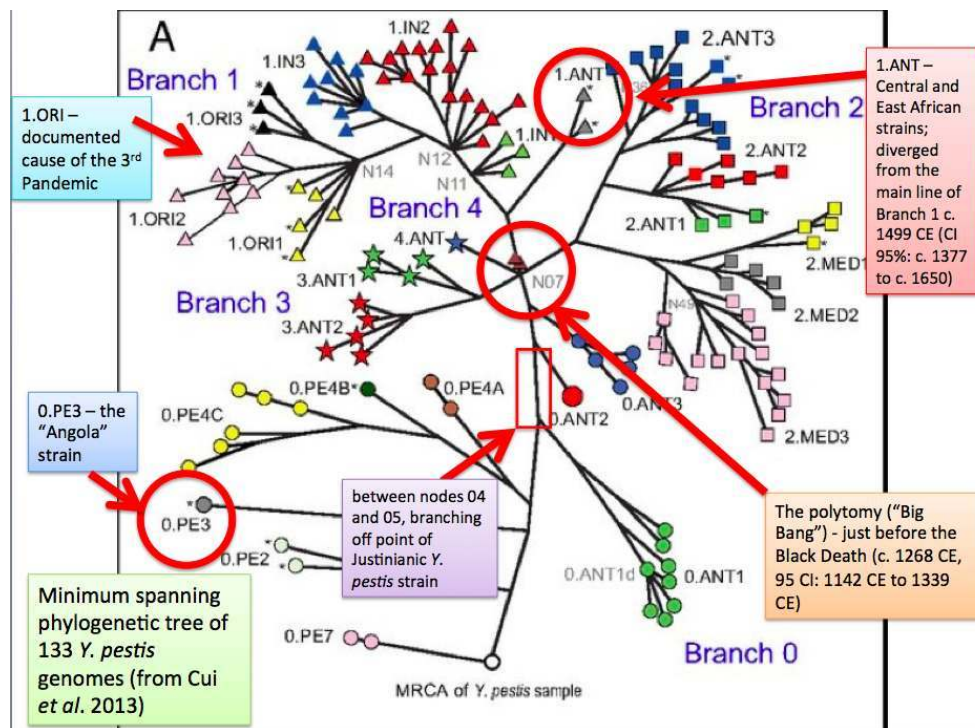
- 12 We know, then, that *Y. pestis* is a pathogen of Eurasian, not African, origin. And we can now confirm its historic role in causing both the late antique Justinianic Plague and the medieval Black Death. But, as noted above, the real value of genetics as a historical tool is the information it can give in tracking infectious diseases over space and time. Such an analysis is particularly important for an imported disease, such as plague is with respect to Africa. Having some basic background on how genetics provides historical data will therefore be helpful.

A. The recent history of molecular genetics and its emergence as a “historical” discipline

- 13 For approximately four decades, starting in the 1970s and continuing until about 2010, there was debate in some circles whether or not the Black Death had been caused by *Y. pestis*, the microorganism associated with the disease plague since it was first identified by Alexandre Yersin in 1894. Even before Yersin identified the bacterium, it was assumed that plague—with its characteristic buboes and rapid disease progression—was the same disease that had caused the Black Death of the 14th century and most subsequent outbreaks variously called, in European languages, *peste*, *pestilence*, *pestilencia*, etc. Skepticism about this disease–pathogen correlation arose in the later 20th century because of perceived discrepancies in the epidemiological patterns of medieval and modern plague outbreaks. Specifically, it was puzzling why the mortality of medieval and early modern outbreaks was so high, when in contrast the early 20th-century outbreaks, though devastating, produced only a fraction of the number of fatalities. Doubts about the pathogen’s identity were gradually put to rest between 1998 and 2010 by molecular identification of key elements of the *Y. pestis* genome in human remains from different burial grounds in Western Europe dating from the 14th to the 18th century.¹⁴
- 14 The new state of understanding of *Y. pestis*’s evolutionary development and historical spread is due to microbiological work in two different domains, both building on work done in the 1990s to fully sequence the *Y. pestis* genome.¹⁵ On the one hand, scientists collected *Y. pestis* isolates from laboratories around the world and tested them genetically to confirm their distinct lineages.¹⁶ On the other hand, another group of scientists who now style themselves “palaeogeneticists”, developing techniques to capture and study aDNA (DNA from historical or “ancient” remains), were able not simply to retrieve fragments of *Y. pestis* DNA from the bodies of victims of historical plague outbreaks (this work had first commenced in 1998), but, in 2011, they were able to complete the first sequencing of the entire historical *Y. pestis* genome from two strains of *Y. pestis* from mid-14th-century London.¹⁷
- 15 In 2013, another major genetics study sequenced an additional 121 modern *Y. pestis* isolates and supported what still stands as the consensus view in the field: *Y. pestis*’s

evolutionary development has a clear pre-Black Death and post-Black Death configuration (Fig. 1).¹⁸ At Node 07 on the tree (circled in red), a sudden polytomy (divergence or branching out) occurred in what had hitherto been a single main lineage (Branch 0). Although the timing of this “Big Bang” in *Y. pestis* dissemination is still debated, it can clearly be situated several decades before the actual Black Death described in European and Middle Eastern sources in the mid-14th century.¹⁹ From that point, four new lineages, each with distinctive genetic characteristics, would develop, creating new sub-branches that are represented by strains of *Y. pestis* still found in the world today. Although strains from Branches 3 and 4 have left a geographically limited footprint (they have currently been found only in western China, Mongolia, and southern Siberia), Branch 2 would eventually be distributed across Eastern and Central Eurasia, reaching from North Africa almost to the Pacific. Branch 1 would have the most widespread effects on human populations, being the cause not simply of the Black Death devastation of the Middle East and Europe, but (as subsequent research has shown) also the continuing devastation in Europe up through the 18th century, outbreaks in southern Russia and western and southwestern China, and ultimately, the entire global pandemic of the late 19th and early 20th century. Along the way, it sent a long offshoot into East Africa, where it thrives to the present day.

Figure 1: Minimum spanning phylogenetic tree of 133 *Yersinia pestis* genomes, with major historical events marked



From Y. cui *et al.*, 2013, fig. 1A, with additions by M.H. Green. Reproduced with permission.

- 16 The 2011 study sequencing *Y. pestis* from 14th-century London was widely hailed as being confirmatory: yes, *Y. pestis* could be retrieved from victims of the Black Death, implicating it, and not some other microorganism, in the largest mortality event in human history. This was no small achievement, of course, given the previous decades of doubt and contention about the cause of the Black Death and other major historical epidemics. Obscured in such an assessment, however, was the fact that the 2011 study

also signaled the foundation of a new, hitherto unrealized line of research.²⁰ The complete sequencing of two different strains of *Y. pestis* from 14th-century London opened up the possibility of beginning to research not simply the history of plague, but the history of *strains* of plague—that is, to track the organism’s development over space and time, and to link evolutionary changes to different outbreaks. Just as had already happened with molecular tracking of different subgroups of the retrovirus HIV-1, and as would happen in tracking several other infectious diseases, such as the outbreak of cholera that struck Haiti in 2010 and the 2013–15 Ebola outbreak in West Africa,²¹ whole genome studies brought the prospect of precise tracking of chains of transmission in time and space to historical epidemics as well. The number of complete genomes of *Y. pestis* that have been reconstructed from historical remains has grown significantly in recent years—70 at this writing. These can be compared with modern strains of the organism which, because they have geographical data tied to them, allow us to correlate specific strains of plague with specific geographies and chronological periods. Even partially sequenced samples can, if they are of sufficient length, be compared with whole genomes and situated on a phylogenetic tree that shows their relations to other strains.²²

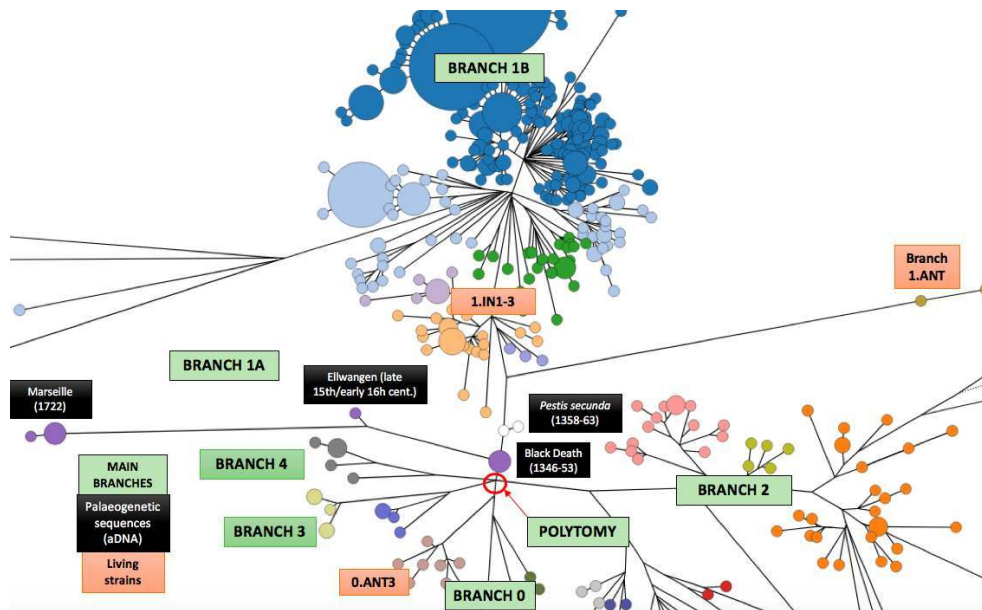
B. Genetics as a source for geographical information

- 17 For observers in Africa and everywhere else in the world prior to the mid-20th century (whose testimony we will examine in section III.C below), plague was just “plague”. There was no meaningful way to distinguish different strains clinically, let alone postulate how different strains might have had different patterns of circulation in the past. A biochemical means of classifying different strains was developed in the middle decades of the 20th century, and certain historical narratives were attached to different strains. That system itself has now proven to be otiose and has been replaced by the molecular system of identification adopted in the present study.²³ This new, evolutionary genetics of *Y. pestis* establishes that there were three separate arrivals of plague into Africa, intrusions that correspond to what are conventionally called the First, Second, and Third Plague Pandemics, described above.²⁴ The first intrusion may have had longer-term effects than we can yet document, but no descendants of the First Pandemic are known to survive, in Africa or anywhere else. The second and third arrivals were separate events from the first intrusion, and both have left genetic descendants that survive in Africa to this day.
- 18 The present study argues that evolutionary genetics—or more specifically, phylogenetic analysis—can do more than simply differentiate the signatures of different pandemic events. Phylogenetic analysis allows detailed tracking of disease transmission and pathogen evolution at the level of localized outbreaks. That is because all genetic data has two kinds of valuable information: information on where strains are circulating, and information on when new strains arise. Analysis of the “archaeological” layers of the genetics archive, together with information on the geographic distribution of strains, can rule out certain epidemiological scenarios just as much as it makes others plausible. The specific task here is to address Sussman’s 2016 claim that the strains of plague now found in East Africa were imported from India early in the 19th century. Approaching that assertion from the perspective of genetics, we would expect (a) that the strains found in India must be in the same lineage as the strains now found in East Africa; and (b) that the strains of plague found in India would

be ancestral to (evolutionarily older than) the strains now found in East Africa, or at least share a recent common ancestor that would make evolutionary derivation from an Indian source plausible. In fact, neither expectation holds.

- 19 The geographical distribution of individual strains is indicative of specific biological histories. Where strains are found now is not a certain indicator of where they have always been, but it at least serves as an endpoint in mapping historical trajectories. All properly curated modern biological isolates carry geolocation data.²⁵ In the case of *Y. pestis*, samples taken from human patients may suffer from a “migratory bias”, since humans are notoriously mobile. In contrast, samples taken from animals, particularly if captured in the wild, likely give reliable information on plague’s natural habitats. Whether human or animal sources, there is inherent sampling bias: the data will show where samples have been found, but should not be assumed to reflect the full extent of a strain’s range. That is also true for the few samples of aDNA that have been retrieved from historical burial sites, all of which (to date) come from human samples and are constrained by the added factors of where archaeological digs have been done and whether remains are available for analysis. Nevertheless, the geographic data are positive evidence for where certain strains are (or were) found in nature. Since phylogenetic analysis can now prove when, in an evolutionary chronology, specific strains took their origin, the geographic data from both aDNA retrievals and modern isolates provide evidence for the geographic and ecological patterns of *Y. pestis*’s circulation since the late medieval polytomy, which, within a very short time period, sent the organism into at least four quite different geographic zones.²⁶
- 20 As more and more genomes of *Y. pestis* are sequenced, we have a greater understanding of how, and where, the different lineages developed. Figure 2 provides an updated view of how the late medieval polytomy, the sudden evolutionary divergence of *Y. pestis* now called the “Big Bang”, is now understood when both extant isolates and aDNA reconstructions are brought together. The polytomy likely occurred in or near the southwest Tian Shan Mountains, which extend along what is now the border of modern Kyrgyzstan and China.²⁷ Two post-polytomy lineages seem to have had very limited geographical distributions radiating out from that center. Branch 4 has been documented in a tarbagan marmot (*Marmota sibirica*) in Mongolia and in a gray marmot (*M. baibacina*) and other hosts in southern Siberia (4.ANT).²⁸ Branch 3 is found only in Himalayan marmots (*Marmota himalayana*, 3.ANT1) in Gansu province, in north-central China, or in the tarbagan marmot and the midday jird (*Meriones meridianus*) in Mongolia (3.ANT2).²⁹ In both these cases, there is no reason to associate these lineages with either India or Africa.

Figure 2: Detail of *Yersinia pestis* phylogenetic tree marked to show the later medieval polytomy (the “Big Bang”) and the new branches it created, both those retrieved from aDNA (black labels) and those from modern isolates (peach-colored labels)



The African 1.ANT lineage is on the upper right; strains involved in the Third (modern) Pandemic are in the upper center. Circle size reflects the number of samples per strain; branch lengths are proportional to the actual number of single nucleotide polymorphisms (SNPs) between nodes.

Modified detail from *Yersinia pestis* data from *EnteroBase* (N.F. ALIKHAN *et al.*, 2018), based on *GrapeTree* (Z. ZHOU *et al.*, 2018); accessed 13 June 2018.

- 21 Branch 1 is the most widely distributed of all the post-polytomy lineages, and it is the one to which the African strain under discussion here, 1.ANT, belongs. The original Branch 1, created by the 13th-century polytomy, may have established its first long-term reservoir in an area near the Caucasus Mountains or Volga River basin. Palaeogenetic research has established that, at the time of the Black Death, it split into two separate sub-branches. Genetics data, combined with historical (documentary) sources, currently support a narrative as follows. In the 14th century, this fulminating strain of *Y. pestis* proliferated quite suddenly, possibly dislodged from a reservoir near the Caspian Sea by an earthquake in 1336. It swept over nearly the whole of the territory of the Golden Horde in 1346.³⁰ Passing then into the Black Sea and then the Mediterranean, it there caused the Black Death as described by countless European and Arabic sources. It also established a new lineage, possibly within Western Europe itself, which I refer to as Branch 1A. That lineage, now extinct, seems to have been responsible for most of the subsequent outbreaks of plague in Europe, up to and including the last major outbreak in continental Europe, the Plague of Provence (1720–22).³¹
- 22 Branch 1B, which includes all the extant strains of the Branch 1 lineage, including the African 1.ANT, was the long-term product of this postulated plague reservoir in southern Russia. Branch 1B sent a second offshoot into Europe, which I here call the *pestis secunda*, which may have traced the same path that the original Black Death took, or maybe took a northern route. It has been retrieved from burial sites in London, from a plague cemetery likely dating to 1361–63, and in the Netherlands, from an undated mass burial in Bergen op Zoom, from the second half of the 14th century. That strain seems to have died out in Europe. But Branch 1B survived in southern Russia, where it

has been documented in a burial in Bolgar City dating from the 1360s or just afterwards.³² No modern descendant of Branch 1B persists now in the western half of Eurasia, but modern strains are documented in East Africa, western China,³³ central China (Yulong),³⁴ southern China, and in all the places where steamships carried the Third Pandemic strains globally out of Hong Kong. Branch 1B is documented in India only in Third Pandemic strains, i.e., strains that could not have arrived there before ca. 1894.³⁵

- 23 Thus far, therefore, three of the four lineages of *Y. pestis* created by the late medieval polytomy either have nothing to do with India or, in the case of Branch 1B, arrived there only very late, beyond the period discussed by Sussman. Strains of Branch 2, on the other hand, have likely been in India, or at least north India, for quite some time. As will be noted from the phylogenetic tree (figs. 1 and 2), right after the polytomy, Branch 2 seems to persist in a single reservoir for a considerable period of time. Then, Branch 2 suddenly split. Where that long-term reservoir was, or where the point of divergence into sub-branches occurred, is yet to be determined. With the split into two sub-branches, however, we begin to have strains that have survived to the present day: 2.MED and 2.ANT. The 2.MED strains are currently spread out horizontally across a vast landscape of the whole steppe zone from the Caucasus over to the west of China, and on to Hebei and Jilin in the east.³⁶ Partial genome studies have established its presence as far west as Turkey and Libya, and south into India.³⁷ The most ancestral strains (closest to the polytomy) are found in both the Caucasus and in northwest China, suggesting diffusion along routes used by the Mongols for military expeditions, communications, and trade.³⁸ Strains in the 2.ANT sub-branch, in contrast, leaving aside samples from urban rats, are found in a north-south range from Mongolia down to Tibet, Nepal, and India. The strain sequenced in India in 2014 caused an outbreak in 2002, affecting 16 people. That outbreak happened in the very far northwest of the country, in Shimla, Himachal Pradesh, near the point of juncture with northern Pakistan, Afghanistan, and Tajikistan, areas long afflicted with plague.³⁹
- 24 To summarize: of the four post-polytomy lineages, only one is associated with East Africa (Branch 1B, specifically the 1.ANT sub-branch) and only one is associated with India prior to the late 19th-century Third Pandemic (Branch 2). These have no documented shared genetic history after the 13th century. On these grounds alone, there is no reason to assume that the widely dispersed sub-branch 1.ANT now found in East Africa came from India.

C. Genetics as a source for chronological information

- 25 It would seem that Sussman's theory of an Indian source for East Africa's plague strain can thus be set aside simply on the grounds of the geography of phylogenetics: the strain(s) found in India are not the same as the strain found in East Africa. But *Y. pestis* has been nothing if not mobile throughout its history, and present location is not a sufficient guarantee of past location. We have ample evidence from political and economic history of India's ties throughout the late medieval and early modern periods to the ecological plague zones of the Eurasian steppe.⁴⁰ In fact, India *could* have been the source of plague transmission to East Africa even as early as the 17th century. One of our earliest persuasive written descriptions of plague in India is Emperor Jahangir's account of a widespread plague outbreak, centered in the Punjab, comes from 1615 or 1617.⁴¹ Jahangir (or rather, the author of this portion of the memoirs, Mu'tamad Khān)

records that plague was reported at this time as a *new disease*. This would accord with the absence of references to plague by any European visitors to India in the 16th or early 17th century, a silence which is notable since both the Portuguese and the Dutch would be well acquainted with the distinctive manifestations of the disease by this time.⁴² So the evidence from India shows that there was likely some kind of plague in India by the early 17th century. Lacking aDNA, we cannot say what strain it was. Ideally, therefore, to confidently track plague strains through time and space, we would like to have as much consilient data as possible.

- 26 Here, I would like to argue why genetics can contribute important chronological data that, even if far less precise than dated documentary records, can be persuasive in epidemiological tracking. Evolution is by its very definition a kind of change over time. Examined at the population level, there is no inherent regularity with which that change occurs. There have been arguments in favor of the existence of “molecular clocks”, consistent rates at which change in the genome of an organism will occur. “Molecular sundials” might be more accurate. Just as the shadow marking an hour on a sundial is of variable size depending on the season of the year, so too are the units that contribute to estimating dates in phylogenetic reconstructions variable. For plague, the most commonly invoked unit of change is the SNP: a single nucleotide polymorphism, the presence at a given location of a different piece of “code” on the genome of one lineage in comparison with others. On the genome, these involve the substitution of, say, a T nucleobase (thymine) at a given genomic position for a C (cytosine), or a C for an A (adenine). Since there are only four nucleobases in DNA (G, guanine, being the fourth), the number of possible changes is limited. What matters, rather, is *where* on the genome those changes occur. Since even a single-celled bacterium like *Y. pestis* will have several million base pairs on its core genome (*Y. pestis* has, on average, over 4 million), the possibility for genetic change is potentially huge.
- 27 An SNP originates as a mutation in a single cell—what would be called a single nucleotide variant (SNV). That change in a single cell, however, must be replicated many million times over to become the “signature” of a lineage, which is what an SNP is.⁴³ What makes an SNP historically important is not that it has occurred in an isolated lineage, but that the lineage with this new, unique genetic marker has become characteristic of the strain of *Y. pestis* found in a plague focus, a reservoir where plague persists from year to year in local rodent populations.
- 28 That so few changes actually become fixed (stable) in *Y. pestis* lineages is one of the distinctive features of this organism. Indeed, *Y. pestis*, along with a few other organisms, has been called “genetically monomorphic”.⁴⁴ That is because, with such a successfully lethal organism, much of that variation happens in what end up being evolutionary cul-de-sacs, dead-ends where the organism runs out of suitable hosts. These bottlenecks, on the level of populations, create what is called purifying selection: by eliminating a lot of diversity, these near extinction events reset the molecular clock back to a standard genotypic norm. Over longer periods of time, purifying selection functions to keep a species’ genotype fairly stable.⁴⁵
- 29 This, then, is why SNPs are such a variable measure of time. Branch 4, one of the direct products of the late medieval polytomy, has acquired, in one sample that has been fully sequenced, only 17 SNPs in the 800 years since the lineage emerged.⁴⁶ Or consider the number of SNPs separating Node 04 (fig. 1), the point on the main phylogenetic tree where 0.ANT1, a strain pre-existing the Justinianic Plague strain, emerges, and Node 07,

the later medieval polytomy. 0.ANT1 likely emerged in its Asian reservoir in the 5th or 6th century BCE, whereas the pre-Black Death polytomy occurred about 1,700 years later. Yet there are only 40 SNPs that the main *Y. pestis* population acquired along Branch 0 in that interval. Other lineages have acquired more than three times as many SNPs in less than half that time.⁴⁷ In the entire history of *Y. pestis* that has now been documented genetically, only a few thousand SNPs have survived what seems to be a repeated process of purifying selection to define the lineages that persist in the world today.⁴⁸ It is therefore not single genetic profiles that we should be looking for to track epidemics, but plague reservoirs.

III. Plague in Africa: The pre-modern pandemics

- 30 Plague researchers in the 19th and 20th centuries speculated about plague's long presence in Africa, a few even suggesting (on the basis of inference rather than evolutionary theory) that Africa may have been plague's original home. It has only been since 2004, and then very gradually, that genetics analyses have begun to untangle historical and classificatory confusions and recognize that while Africa has indeed had multiple encounters with plague, it was not its original home.⁴⁹ There is little reason to rehearse those previous views here, except that this altered perspective on Africa as the intermittent recipient of plague rather than its perpetual home means that a chronology of introduction and spread now needs to be established. In 2016, Sussman denied entirely that plague had existed in later medieval East Africa. Instead, he assumed that the suspected presence of plague in late antique northeast Africa, although the likely source of the Justinianic Plague, had died out, leaving Africa plague-free until the disease was reintroduced, according to him, in the mid-19th century. Other than my own brief speculations about the possible presence of plague in late medieval East Africa published in 2014, the possibility that the Black Death, the most devastating epidemic disease of Europe and the Mediterranean, might also have affected lands further south has been a mere possibility, whispered in footnotes or summary conclusions, but not the object of sustained research to date.⁵⁰
- 31 Distinguishing the "archaeological layers" of plague in East Africa is important not simply to avoid confusing evidence from different time periods, but also so that any larger patterns that transcend specific outbreaks might be discerned. The narrative of genetics, whose basic outlines have already been recounted, makes clear that the strains involved in the First and Second Pandemics, although coming out of the same central Eurasian reservoirs, were separate and otherwise unrelated transmissions. The Justinianic Plague can be understood as an important rehearsal of Africa's experiences with the Second Plague Pandemic for two reasons: first, because the path taken to reach the Mediterranean may have included northeast Africa; and second, because if plague did focalize in Africa in the late antique period, those areas might be amenable to focalization again later. The bulk of our attention in this section, however, will go to the distinctive Second Plague Pandemic strain in East Africa, 1.ANT, whose relationship with the larger global history of plague has begun to become clear only in light of new phylogenetic work on *Y. pestis*. The innovation to be presented here is that, when juxtaposed to other sources of African history, the genetics narrative suddenly becomes alive with new meaning, offering a skeletal framework on which other elements of East Africa's history with epidemic disease might be structured.

A. Africa's role in the Justinianic Plague

- 32 The historical (documentary) record for the Justinianic Plague (c. 541 to ca. 750) paints it as a phenomenon centered on the Mediterranean, stretching from Persia to Spain.⁵¹ The basic narrative historians have often told is that the plague first broke out, in 541, in the Egyptian port town of Pelusium (on the eastern edge of the Nile Delta), and spread from there to other cities surrounding the Mediterranean. From these coastal introductions, it then spread further inland. Recent work in climate history suggests a more than coincidental shift in climate in the period from 536 on, initiated in part by a series of major volcanic eruptions which brought markedly cooler temperatures for several decades.⁵² The genetic (biological) record suggests, in contrast, a far more expansive geography, connecting the Mediterranean with the Tian Shan mountains in Kyrgyzstan, and a deeper chronology for this epidemiological event. *Y. pestis* genomes have been successfully retrieved from 6th-century Bavaria and, as recently reported, Valencia, southern France, and England as well.⁵³ The strains involved in the Justinianic Plague are most closely related to the extant strains about midway on the 0 Branch of the consensus phylogenetic tree, falling between extant strains now known as 0.ANT1 and 0.ANT2/0.ANT5.⁵⁴ After branching off from this main lineage, however, what would become the Justinianic Plague strain underwent considerable additional genetic change. The Bavarian genomes have, at minimum, 43 SNPs differentiating them from the main Branch 0 lineage.⁵⁵ This considerable evidence of genetic change corresponds with current estimates that the Justinianic Plague strain diverged from the main *Y. pestis* lineages at the beginning of the Common Era.⁵⁶ Field survey data on animal hosts and the strains of *Y. pestis* they carry suggest that that event likely occurred along or near the Tian Shan mountain range.⁵⁷ The organism clearly must have passed through multiple ecological zones before making its way to Bavaria. The question for African history is whether, in the centuries separating its movement out of Central Asia and its explosive debut on the Egyptian coast in 541, it established a temporary reservoir in Africa.
- 33 Neither historical sources nor the biological record can yet provide an answer to the question of the route plague took to reach the Mediterranean. Many historians favor a Red Sea route, which in turn implies passage through the Indian Ocean.⁵⁸ One might envision either a swift passage of plague through the region, moving with the substantial amount of goods traded through the area, or a slow passage, which may be supported by hints in the historical record that plague focalized in the region of the Red Sea. Procopius's report of plague breaking out in 6th-century Pelusium is not, in fact, our first report of what seems to be plague in ancient Mediterranean sources. Writing in the 1st century CE, the medical writer Rufus of Ephesus repeated the reports of earlier medical writers describing a plague-like disease in Libya, Egypt, and Syria. A close examination of records from Egypt found no evidence of regular encounters with plague-like epidemics prior to the onset of the Justinianic Plague.⁵⁹ Nevertheless, in 200, and in 250–53, epidemics (λοιμωδη νοσων or λοιμος) were reported in “Ethiopia”, here understood to mean Nubia.⁶⁰ Whether these were episodes of plague is unclear. In texts describing the Justinianic Plague, contemporary sources suggest “India” or “Kush” as the source of the disease, both terms at the time being understood for the region we would consider modern Ethiopia and Eritrea.⁶¹ The Aksumite Kingdom—and its large metropolitan center at Aksum—collapsed by the 7th century, whereas Egypt,

while also hard hit, was able to survive and soon thrive again.⁶² The reasons for the differential survival of these two polities may have been several, but the well-documented affinity of *Y. pestis* for high elevations may have been a factor if, in fact, the organism did temporarily focalize in northeast Africa.⁶³

B. Africa and the Second Plague Pandemic

- 34 As noted in the Introduction, one of the principal revelations of the new evolutionary approach to plague's history is to force our view from the dramatic initial reports of plague outbreaks—whether those from Procopius in the 6th century or Ibn al-Wardi in the 14th—and focus on the processes that allow plague to persist and spread, most frequently in circumstances when no human is present to record its movement. Given the vastness of the expanses that plague traversed in the late medieval and early modern centuries, the story of the Second Plague Pandemic will clearly be made up of narratives fractured by large gaps in our evidence. The story of 1.ANT—its genesis, its transference across the Middle East, and its eventual penetration of the highlands of East Africa—is such a story of fractures. And it may not be the only story that needs telling.
- 35 The normally cited end date for the Justinianic Plague is ca. 750. Reports of what may be outbreaks of plague come from the Maghreb, Egypt, and Mesopotamia as late as the 11th and 12th centuries, but whether these are late-surviving offshoots of Justinianic Plague strains, signs of new emissions out of Central Asia, or even not “plague” at all, is impossible to tell at the present time.⁶⁴ Scattered reports of plague outbreaks in the second half of the 13th century are, I believe, likely part of a new, separate plague phenomenon, part of the new “seeding” of post-polytomy strains of *Y. pestis* that would explode, starting in the 1330s and 1340s, into the Black Death as experienced in Western Eurasia and North Africa.⁶⁵ And explode it did. To take just the example of Cairo, undoubtedly the largest African city at the time, current estimates of mortality from the Black Death suggest that at least 52% of the city's inhabitants (probably including recent refugees) died in the space of just a few weeks. On the two days of highest mortality, 13,800 people died. Only two times in Cairo's history were the required prayers for the dead, the *Janaza*, waived because of the impossibility of coping logistically with the number of the dead; the Black Death strike of 1348 was one of them.⁶⁶
- 36 Given that so much of the Islamic world was affected by the first wave of the Black Death, it is more than reasonable to ask whether its effects on Africa might have been even greater than we have yet been able to document. The other essays in this special issue of *Afriques* are addressing this question for West and Northeast Africa.⁶⁷ For East Africa, as I noted already in 2014, the question has been hinted at but never pursued in a rigorous fashion. Signs of demographic collapse or abandonment in the 14th and 15th century along the East African littoral, which of course was connected to the Mediterranean via Red Sea trade into the Indian Ocean, might be attributable to plague. Such hints of demographic disruption have been noted for Gede, Kilwa, the Ruvu communities in the area of modern Tanzania, and perhaps even Madagascar, whose northern town of Mahilaka seems to have undergone decline around this same time, too. Perhaps there is even some connection to be drawn with the decline of Great Zimbabwe some distance from the Indian Ocean littoral.⁶⁸

- 37 Even if the East African littoral was struck by outbreaks of plague starting in the mid-14th century, however, the genetics would argue against a connection between any strains of *Y. pestis* that might have reached the coast then and the genesis of 1.ANT, the focus of the present analysis—since, as we have seen, the latter strain must have come out of a reservoir that the main Branch 1B shared for some time in Central Eurasia, perhaps as much as a century or more after the Black Death. Keeping these “archaeological layers” in mind is crucial if we are to fully understand Africa’s experiences with plague. For example, the well-documented case of Cairo’s assault by the Black Death would suggest that it was struck in 1348 by the same transmission of plague that reached other Mediterranean ports in 1347–48. Logically, therefore, that would make the original Black Death strain (the progenitor of both Branch 1A and Branch 1B) the likely culprit of Cairo’s catastrophic epidemic that year, not a more derived strain from the Branch 1B lineage. (This distinction will prove relevant when we look at 1.ANT’s possible routes of entry into East Africa in the next section.) There is, moreover, some evidence of economic recovery along the coast in the 15th century, which might be taken as evidence that, if plague did indeed strike, the infestation was temporary.⁶⁹
- 38 It is possible, therefore, that there are multiple stories to tell about East Africa’s engagements with plague in the late Middle Ages. The focus of the rest of the present essay will be on strains in the 1.ANT lineage, the oldest living cousin of the Black Death lineage, which takes us beyond the Middle Ages into the early modern period. On Figure 2, off to the far right, there are two isolates separated from the rest of Branch 1B by several dozen SNPs. The 1.ANT sub-branch is now found exclusively in East and Central Africa. It has been documented genetically from modern samples, collected since the 1940s, in Kenya, Uganda, the eastern part of the Democratic Republic of Congo, and Zambia. The pattern of geographic spread suggests it may also exist in neighboring areas of East Africa, such as Tanzania, Rwanda, and Burundi, which are part of the same ecological and climatic landscape of the Great Lakes District, and where plague persists to the present day.⁷⁰ According to a calculation performed by geneticists in 2013, 1.ANT diverged from the main line of Branch 1B at some point between c. 1377 and c. 1650.⁷¹ That estimate was never more than a calculus of probabilities, of course, and the recent recalibration of *Y. pestis*’s overall evolutionary development will likely push this estimate back a few decades.⁷² The 1.ANT strains have undergone considerable evolutionary change since splitting from the main Branch 1B. Assuming the main reservoir of Branch 1B was somewhere between the northern Caucasus/Volga basin region (the documented locus of 1.ANT’s closest progenitor) and western China (where the next most closely related strains survive today), the ancestral strain of 1.ANT must have passed through the Middle East—the lands of the Ilkhanate and its successor states in Iran; the growing Ottoman empire in what is now Turkey and the Balkans; the final phases of the Mamluk sultanate in Egypt before its overthrow by the Ottomans in 1516–17; or the Safavid empire, which ruled 1501–1722 in territory that would include modern-day Iran and parts of Pakistan, Afghanistan, and Turkmenistan, and would thus connect Central Eurasia with the Indian Ocean and thence East Africa. All these polities suffered repeatedly from outbreaks of plague throughout the 15th to 18th centuries.⁷³ Again, we have no aDNA from any of these regions, at any date, so we can identify “plague” only by the consistent epidemiological profiles we find in written documentation from the Ottoman, Mamluk, and Safavid regimes. Significantly, no living trace of early Branch 1B strains has been found

between the Caucasus and Africa in modern times, even though plague is still present in many of these regions. The only *Y. pestis* strains to be recovered from the Middle East and genetically typed have been either Third Pandemic strains (1.ORI) or those from Branch 2 (mostly 2.MED).⁷⁴

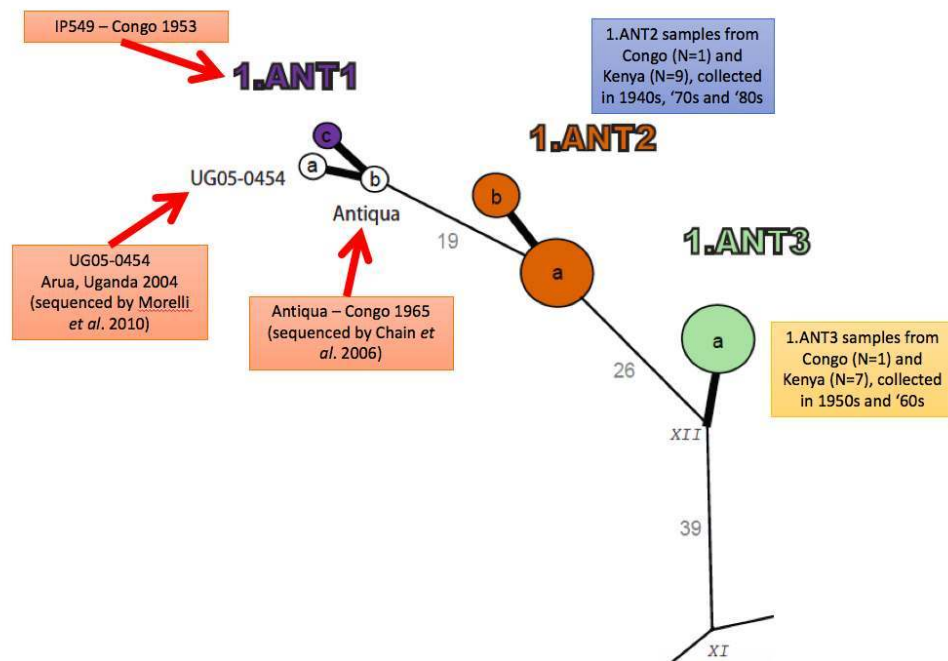
- 39 There is much about the original circumstances of 1.ANT's genesis and path across the Middle East that we cannot yet answer. That it needs to be separated from Africa's initial experiences with the Black Death in the 14th century seems clear, as does the fact that much research is needed to fill in the history of plague in Central Eurasia in the 15th and 16th centuries, when this new strain was likely evolving. For 1.ANT's history within Africa itself, however, there is a good deal that can be said both about how long it has likely been present there and the routes it has taken through the continent.

C. 'From time immemorial': How long has 1.ANT been in Africa?

- 40 In 2016, a study was published looking at 61 *Y. pestis* isolates collected from among 1,092 human plague cases from Vurra and Okoro counties within the Arua and Zombo districts, respectively, of western Uganda. These cases had occurred in Uganda between 2004 and 2012. Although researchers did not do whole genome sequencing, they were able to distinguish two SNP Group strains which differed as to location (including elevation) and likely in the vector-host ecology.⁷⁵ They showed that over the course of the study, plague moved into a new, more northerly region. Indeed, the fact that a Congo strain collected in 1965 and sequenced in 2006 (Antiqua) is ancestral to all the Ugandan SNPs might suggest that plague had only recently been imported into Uganda from the Democratic Republic of Congo.
- 41 Such studies of plague couched in the rhetorical framing of "emerging diseases" makes the disease seem as if it were new in the region, the result of recent events, such as the Ituri conflict going on over the border in the Democratic Republic of Congo. It would probably be better to say that it had newly re-arrived. Increasingly, it is understood that *Y. pestis*, and especially the Branch 1B strains, is an itinerant disease. In the United States, for example, where one of the 1.ORI strains arrived around 1900, the organism has yet to establish any permanent mammalian reservoir. Rather, it keeps cycling from rodent burrow to rodent burrow, moving across species of ground squirrels, prairie dogs, and various predators, killing whole populations at a time but surviving in enough nests or burrows to continue the enzootic cycle.⁷⁶ Local "burn-outs" are, therefore, likely part of plague's normal *modus vivendi*, so we should not assume that plague will always be visible in causing human outbreaks in any given area. We get a broader perspective on plague in East Africa when we look at data on the reported presence of plague in the Democratic Republic of Congo, Uganda, and Kenya collected from 1877 until 2009.⁷⁷ Here we see that all three countries have had massive numbers of human cases over the course of the 20th century, suggestive of widespread focalization of plague in silvatic rodent populations.⁷⁸ For example, in the ten-year period from 1910 to 1919, Uganda reported 31,305 deaths due to plague. That considerable epidemiological burden could, of course, be the result of recent introduction of the pathogen. India, for example, suffered millions of deaths due to plague in the early 20th century as part of the Third Pandemic. Yet in Africa, we can look back even earlier than the beginning of epidemiological data collected by colonial

governments. For this, we have genetic evidence; the written testimony of colonial authorities, visitors, and missionaries; and the testimony of Africans themselves.

Figure 3: Detail of the 1.ANT branch of a minimal spanning tree of SNPs for isolates of *Yersinia pestis*



Supplementary Fig. 7 from G. MORELLI *et al.*, 2010b; modified by M.H. Green and reproduced with permission.

- 42 In their 2013 phylogenetic tree for *Y. pestis* (fig. 1 above), Cui and colleagues placed only two isolates on the long, otherwise empty branch that led to 1.ANT. That was because at the time, and still to this day, these were the only samples of 1.ANT that had been fully sequenced. One, mentioned above, is called simply “Antiqua” and was sequenced in 2006 from a sample collected from a patient in the Congo in 1965. The other, “UG05-0454”, had been collected in Arua, Uganda, in 2004 and was sequenced for the first time for Morelli and colleagues’ 2010 study of *Y. pestis*’s global distribution. “Antiqua” is the more ancestral of the two, though both strains are very far distant from the main Branch 1 lineage, having acquired 79 SNPs that differentiate them from all other extant post-Black Death strains.
- 43 If we look at the corresponding part of the *Y. pestis* phylogeny from the 2010 study of Morelli and colleagues, which included data not simply from whole sequences but also from partially sequenced isolates (fig. 3), we see that the 1.ANT branch is fuller and shows much more divergence. 1.ANT3, which is represented by eight isolates—one from the Congo and seven from Kenya, all of them collected in the 1950s and ‘60s, is the most ancestral sub-branch of the lineage, separated from the main Branch 1 stem by a minimum of 39 SNPs. Connecting 1.ANT3 back to the polytomy via its common ancestor with the Bolgar City genome (10 SNPs before the point where the 1.ANT strains branch off), the London 6330 genome (1 SNP before Bolgar City), and the Black Death genome (2 SNPs before London 6330, and 2 after the polytomy), we can count a total of 55 SNPs. From the polytomy to 1.ANT1b, the “youngest” and furthest documented node on the 1.ANT branch, is a total of 104 SNPs. In other words, about half of the divergence that

has occurred within the 1.ANT branch—which has likely occurred over the past 300 years or more—is still documented in the surviving strains.

- 44 Did all that diversity happen within Africa itself? Only multiple, well-dated aDNA samples could prove that definitively. It is possible that this diversity could have already accrued in another reservoir and been imported into East Africa wholesale. But because some of the collection dates go back as far as the 1940s (IP547 and IP548, both samples of 1.ANT2a) and, by 1953 at the latest, comprises isolates from the furthest node, 1.ANT1c (IP549), we know that all of the documented diversity already existed in at least Kenya and Congo by the middle of the 20th century (see Table 1.) All these isolates were obtained from human patients, meaning we cannot be sure what role recent relocation may have played in their infection. Nevertheless, it is interesting that all three groups (1.ANT3, 1.ANT2, and 1.ANT1) are found in both Kenya, on the coast, and in Congo (now, Democratic Republic of Congo), about 1,000 km or more away. All Congolese cases have come from the eastern portion of the country, pointing to the plague foci in those elevated regions near the headwaters of the Uele River and near Lake Albert. Studies from the 1920s and 1930s suggest that it was just becoming a new problem in the region then, though that may be an artifact of increased colonial scrutiny by foreign doctors.⁷⁹ Ituri province remains one of the most heavily infested plague regions in the world and was the site of the two worst recent outbreaks in the Democratic Republic of Congo (in Zobia in 2005 and Bolebole in 2006).⁸⁰

Table 1. East African *Yersinia pestis* isolates used for 2010 study of plague

Group	Node	Strain	Original	Biovar	Country	Year
1.ANT1	a	UG05-0454	[Arua, Uganda, from a 10-year-old female]	Antiqua	Uganda	2004
1.ANT1	b	Antiqua	[human]	Antiqua	Congo	1965
1.ANT1	c	IP549	Logo 1/53	Antiqua	Congo	1953
1.ANT2	a	IP544	Muimi	Antiqua	Kenya	<1984
1.ANT2	a	IP545	David Matata	Antiqua	Kenya	<1979
1.ANT2	a	IP547	Dar Es Salem	Antiqua	Kenya	1948
1.ANT2	a	IP548	343	Antiqua	Congo	1948
1.ANT2	a	IP550	Edmond	Antiqua	Kenya	<1981
1.ANT2	a	IP554	Rosaline	Antiqua	Kenya	<1983
1.ANT2	a	IP677	Beatrice	Antiqua	Kenya	1981
1.ANT2	b	IP551	Tanzania	Antiqua	Kenya	<1981
1.ANT2	b	IP552	Margaret	Antiqua	Kenya	<1982
1.ANT2	b	IP553	Kyule	Antiqua	Kenya	<1981

1.ANT3	a	IP536	129	Antiqua	Kenya	1955
1.ANT3	a	IP537	147	Antiqua	Kenya	1952
1.ANT3	a	IP538	162	Antiqua	Kenya	1952
1.ANT3	a	IP539	164	Antiqua	Kenya	1952
1.ANT3	a	IP540	169	Antiqua	Kenya	1952
1.ANT3	a	IP542	144	Antiqua	Kenya	1952
1.ANT3	a	IP543	Lita	Antiqua	Congo	1953
1.ANT3	a	IP566	129M	Antiqua	Kenya	1964

Information drawn from G. MORELLI *et al.*, 2010b, Supplementary Table 3; source of 1.ANT1a and 1.ANT1b provided from G. MORELLI *et al.*, 2010a, p. 1144, and P.S.G. CHAIN *et al.*, 2006, p. 4454, respectively.

- 45 Genetic diversity does not, of course, necessarily prove age in a given habitat. As we saw earlier, a real-time study of an outbreak in Madagascar documented the production of 13 new SNPs in a ten-year period. Nevertheless, the *Y. pestis* phylogeny overall shows the effect of purifying selection. Most mutations get eliminated from the lineage. Only those that have successfully reproduced in long-term reservoirs survive. Out of the whole *Y. pestis* phylogeny, few other post-polytomy strains have accumulated as many sustained mutations as have the 1.ANT strains. When we add the genetic diversity of 1.ANT to the evidence for deep infestation in an area that is very minimally urbanized, distant from any seaports that might have recently introduced the disease, the question is raised whether this is not indicative of deeper historical roots of plague in the region.
- 46 The presence of plague in the region was noted in the accounts of numerous 19th-century and early 20th-century European colonialists. The English explorer Richard Burton reported that Arab informants told him of the presence of plague (*ta'un*) in Karagwe (now northwest Tanzania), upon their arrival there in the 1830s or '40s.⁸¹ The German bacteriologist Robert Koch (1843–1910) commented on this deeper history of plague in the region when he visited the region now constituting Zimbabwe in the 1890s. Having been reassigned there from India (where what we now call the Third Plague Pandemic had just broken out), Koch had good reason to reflect on the question of whether plague infestations were new or not.⁸² As Sussman notes (quoting Koch himself), although the disease had been present in Zimbabwe for thirty to forty years, it “existed in other parts of Uganda since time immemorial [*seit undenklichen Zeiten*]”.⁸³ While Sussman is right that Koch had no evidence for a *continuous* presence of plague in this part of Africa since ancient times, in fact there is near unanimity among early missionaries and other colonial functionaries that plague was, upon their arrival in the 1870s and later, already widespread and embedded in the landscape.⁸⁴
- 47 One of the reasons Koch assumed a long presence of plague in East Africa is probably because he, or his European interlocutors, took seriously the stories about plague they heard from Africans themselves. Cuthbert Christy, a British medical officer stationed in

Uganda who had been sent there as part of the British Sleeping Sickness Commission and who knew Koch's work well, similarly reports in 1903 that plague had been endemic in the district for many years, and acknowledges that it was established in the region long before British occupation of the region began.⁸⁵ Three decades later, according to a summary by R.E. Barrett, a British medical officer in Uganda, Sir Edward Norton reported that

early mission settlements at Rubaga and Buddu in Buganda were evacuated on account of the ravages of the disease, whilst the Nilotic branch of the Kavirondo tribe now established on the eastern shore of Lake Victoria are stated by him to have experienced epidemics of the disease not only after their arrival, but before and during their migration southward from the Nile basin.⁸⁶

- 48 J. Isgaer Roberts, a British entomologist writing from Kenya in 1935, likewise confirmed the unanimity of opinion among colonial administrators and missionaries as to plague's long presence in the region. Summing up, he cited one of the leading colonial plague researchers of the day, W.J. Simpson, who wrote in 1914 that

the evidence as to plague having existed in Uganda for many years before 1894 when the country became a British Protectorate is such as to establish the fact beyond dispute. Still, there can be no doubt that plague has been endemic in the western province of Uganda for many years and, if tradition is correct, it has been there for ages.⁸⁷

- 49 In other words, separate testimonies of British and German observers in the late 19th and early 20th century, *talking to local inhabitants who recounted their own experiences with a disease causing fevers, buboes, and mass, swift mortality*, reported plague's presence on the western, northern, and eastern shores of Lake Victoria. They were unanimous in their opinion that its presence in the region was of long duration, even if its manifestation in particular areas was variable.

- 50 These colonial accounts do more than reflect the impressions of colonial administrators and missionaries, however, who were all newcomers to the region. Indirectly, they also capture the experiences of East Africans themselves. In 1903, British physician Cuthbert Christy recounted an expedition he made seeking to confirm the presence of sleeping sickness, only to find instead widespread presence of, and knowledge about, *kaumpuli*.⁸⁸ (In other sources, this will be spelled *kawumpuli*, the form I have adopted here, and *kapumpuli*. Christy also reports the term "*tubunga*".) Christy found, in his visits to the Ssesse Islands in Lake Victoria, Bikira, Mbarara, Kifumbero, and Fort Bukoba (the last in what was then German East Africa and what is now Tanzania), that *kawumpuli* was rampant. At Mbarara, to the west of Lake Victoria, for example, Christy received testimony about *kawumpuli* from a missionary:

Father Gorgu informed me also that *kaumpuli* was well known to the natives and dreaded by them long before the missionaries came to Bikira, eight years previously.

- 51 At Fort Bukoba, a German physician's account made clear that infestation was perennial:

Dr. Feldmann said, too, that it was generally believed at Fort Bukoba that the plague was introduced each year into German East Africa from Budu because of its always occurring in the former place about June, when the rains had ceased, and when the natives were able to cross the Kagera River and journey southwards.

- 52 Christy's conclusion is important:

And there can be no doubt, however extraordinary it may seem to those who have studied the disease in India and elsewhere, that there exists at Bikira, in Budu, a

province of the Uganda Protectorate, to the west of the Victoria Nyanza, a focus of bubonic plague, which annually gives rise to small, but very virulent epidemics, and that from this focus the disease is carried at certain times of the year into German East Africa. It is also probable that the Egyptian epidemics have had their origin in this endemic centre in Uganda.⁸⁹

- 53 Christy went on immediately to suggest an outbreak in Nairobi that had happened the previous year had been imported from India. That may well have been possible, and there is no reason there could not have been a mixing of plague strains in East Africa with those radiating from the Third Pandemic out of Hong Kong, which by the time Christy was writing was well underway.⁹⁰ As was noted earlier, to all observers of plague prior to the 1950s, “plague was plague”; there was as yet no mechanism to differentiate strains. Nevertheless, Christy concludes that “*Kaumpuli* seems, then, to have been known to the natives of Uganda for ages”.⁹¹
- 54 The fact that there was a specific and widespread name for the disease is itself evidence for its longevity in the region. A study of diaries written by recently Christianized Baganda in the 1870s and 1880s, as well as Europeans’ accounts, produces a number of references to *kawumpuli*, confirming that it was widely understood that plague was endemic to Buganda. Not simply was *kawumpuli* the name of a disease; Kawumpuli was the name of the son of the historical *kabaka* (king) Kayemba (c.1670–c.1700), who was born without arms and legs, and who came to function as a protector against plague, helping sufferers to reintegrate to society by distributing goods gathered from those who had succumbed.⁹² Historian Neil Kodesh has suggested that the historical figures who became the objects of shrines in Buganda, known as *lubaale*, should be seen as “national spirits”, figures whose stories enlarged to become protectors of a “nation” of Buganda, providing both social and biological health and so uniting the Baganda people into a larger political entity. Archaeologist Scheherazade Amin looked in detail at the material evidence for the Baganda *lubaale*; although the majority of *lubaale* shrines were located on the islands of Lake Nyanza (Lake Victoria), she identifies the Kawumpuli shrine in the far north of Buganda lands, in Bulemeezi County.⁹³ This is, of course, very significant in light of the narrative from genetics I have reconstructed here, since it likely would have been from the north that 1.ANT1 entered the lands of the Baganda surrounding Lake Nyanza (Lake Victoria). Whatever interpretations must be made to sort through the several versions of the story of Kawumpuli (that he was really the son of Kanakulya de Buyembe, not Kayemba; that “he” was really a goddess, etc.), the story itself shows that plague had been impressing on the culture of the Baganda since at least the 17th century.
- 55 Christy credits Koch with plague’s formal, biomedical identification in East Africa and provides his own translation of Koch’s report on the disease from 1897. In that case (already summarized by Sussman), Koch, then in Dar-es-Salam, sent a proxy, Dr. Zupitza, into Bukoba (Tanzania) to gather clinical samples of an outbreak on-going in Kisiba. From Zupitza’s reports, Koch, too, gathered that the disease already had a local name, *ruburunga*. From the laboratory slides Zupitza prepared both from plague victims and from rats found at the site, Koch confirmed that he “cannot discover the slightest difference between the plague bacteria from Kisiba and those from Bombay”.⁹⁴ It would not be until the advent of strain typing some decades later (and, more recently, genotyping), that it would be possible to reveal that plague’s history in Africa was infinitely more complicated than either Koch or Christy imagined.

D. Whence did 1.ANT arrive?

- 56 Such, then, are the chronological parameters of 1.ANT's genesis and entry into East Africa. The environmental or anthropogenic circumstances that facilitated plague's movement into the region remain unclear. The genetics of *Y. pestis* cannot yet rule out any of several possible routes that the progenitor of 1.ANT might have taken to reach equatorial East Africa. Did it cross the Sahara from the west, passing into Congo? Did it instead arrive from the east, via the Indian Ocean littoral? Did it travel south from Egypt through the Nile Valley, eventually reaching the Great Lakes via the White Nile?⁹⁵ Or did it come more indirectly via Ethiopia, passing through the highland regions there?
- 57 One scenario of 1.ANT's introduction into East Africa that can be ruled out now is importation from China itself. In 2010, microbiologist Achtman suggested that the Chinese explorer Zheng He (1371–1433 or 1435) may have been involved in the transmission of plague from Asia to East Africa, since his voyages extended to the East African coast. Achtman retracted this hypothesis in 2013, not because of its epidemiological implausibility (the idea that Zheng He's massive fleet could have transmitted plague all the way to Africa, but not succumbed to outbreaks itself), but because the sequencing of the East Smithfield Black Death genomes in 2011 had forced a recalibration of the dates at which the strain likely separated from the main Branch 1 lineage.⁹⁶ The late medieval or early modern eastern Chinese coast is an even less plausible source of Africa's 1.ANT strain than India, not because China did not witness plague in this period (it almost certainly did),⁹⁷ nor even because there was insufficient maritime trade coming out of China and connecting with the Indian Ocean in this period (an exceptional amount of trade had been going on for centuries),⁹⁸ but simply because, as already noted (section II.A above) we have no reason to believe that Branch 1 strains reached that far east at this early period.
- 58 Another possible route for 1.ANT into equatorial East Africa might have been across the Sahara. Here, the Chad Basin, and the caravan route, nearly 2,500 km long, was linked via Kavar and Fezzan to Tripoli.⁹⁹ Continued circulation of plague in the Mediterranean is well attested in the late Mamluk period and into the Ottoman era, so the suggestion is not inherently implausible. Moreover, transmission of plague across open desert landscapes is well attested, especially when camels (which are very susceptible to plague) are involved. Especially after the Ottoman conquest of Mamluk Egypt in 1517, there would have been ample opportunity for the continued circulation of plague from the Caucasus area to North Africa.¹⁰⁰
- 59 As for an Indian Ocean route, although I have already ruled out, on the basis of genetic evidence, the idea that Africa's 1.ANT strain was imported from India, there are other scenarios that might involve introduction of plague from the east coast. As is becoming more and more apparent from archaeological researches, the Swahili Coast was a region of intense biological and commercial exchange with the Indian Ocean basin from at least the 7th century on.¹⁰¹ A plague-permissible rodent infrastructure, including the commensal black rat (*Rattus rattus*), was present on islands off the Swahili Coast from at least the 7th or 8th century CE, though this species, implicated in all three plague pandemics, cannot be documented in the hinterlands until the 15th century.¹⁰² Moreover, both the Red Sea and the Persian Gulf connect East Africa to the ecologies of the Middle East. It has been noted that for some communities on the Indian Ocean

littoral, there is a pattern of abandonment or decline in East African coastal trading zones in the 14th or 15th century just as in West Africa. Although it has been speculated that plague may have played a role in these apparent declines, it has also been suggested that, being reliant on international trade, these mercantile economies were simply suffering secondary effects of the Black Death's effects elsewhere.¹⁰³ Even if plague did strike the Indian Ocean coast in the 14th or 15th century, genetics argues against this being the founding event for 1.ANT's genesis, since that strain, as explained above, must have a common history that persisted in Central Eurasia until it broke free of the rest of Branch 1B near the end of the 15th century.

- 60 A more likely scenario for 1.ANT's introduction into East Africa—one that would fit the demands of the genetics narrative—would be one that ties it to the Persian Gulf and the Red Sea, which in the 16th and 17th centuries involved several of the global and semi-global superpowers of the day, including the Ottoman empire, the Safavid empire, and the Portuguese. In the 16th century, for example, there was considerable wrangling between the Ottomans, the Portuguese, and the Mughal empire for control of the Red Sea, the crucial link between the Mediterranean and Indian Ocean spheres of trade. Although less well documented, those maritime interventions extended at times down the length of the East African coast, creating a potential disease trajectory that linked to the Black Sea, which the Ottomans controlled exclusively by this point.¹⁰⁴ The possible role of this exceptionally active trading zone in the dissemination of plague into Africa will need to be the subject of another study.¹⁰⁵ But it will be useful here to assess a particular claim that has entered plague history, if only to see how it might be differently interpreted.
- 61 Just as the famous siege of Caffa has played an outsized role in narratives of the Black Death's entry into Western Eurasia in the mid-14th century, so another famous siege, this time in the 17th century, has been said to have introduced plague into a new ecological niche. In 1696–97, soldiers from Oman attacked the Portuguese stronghold of Fort Jesus in Mombasa (modern-day Kenya), part of the Portuguese buildup of defensive forts along the Swahili Coast at the end of the 16th century to halt Ottoman ambitions in the region.¹⁰⁶ According to a standard account, following the commencement of the siege in November 1696, two Portuguese ships arrived from Goa, in India, to provision the fort. Their hasty departure in January 1697 coincides with the beginning of a “swelling sickness” (*inchações*) that was killing three to four men each day. By August, the fort was completely depopulated of Portuguese. Although it has been implied that the Omani introduced the plague, no new arrivals from Oman are reported in this period. One commentator implies that it was the Portuguese themselves who brought the disease.¹⁰⁷
- 62 Leaving aside the question of the unusual name “swelling sickness”, epidemiologically the rate of mortality reported for the fort is plague-like. Were the siege really the point of first entry of 1.ANT into East Africa, however, it would put plague's arrival in East Africa too late to fit plausibly with Baganda narratives about Kawumpuli. If the disease came from Oman, that would fit a Middle Eastern scenario of transmission currently demanded by the genetics narrative, though if it was brought by the Portuguese themselves, then that would likely have been from Goa, India, where, as noted above, we do not yet have evidence of Branch 1 strains.
- 63 There is, however, another interesting detail in this account, which suggests that the disease was not newly imported into East Africa across the Indian Ocean at all, but was

already present inland and was now reaching the coast. It is reported that one of the Portuguese commanders, who departed precipitously in January 1697, had been ordered to make contact with a local group, the Musungulos.¹⁰⁸ The Musungulos (or Mossegulos) or “Africans of the coast behind Mombasa”, were part of the Mijikenda, the Nine Tribes (Digo, Giriama, Chonyi, Kambe, Ribe, Jibana, Kauma, Rabai, Duruma) who had migrated out of an area in present-day Somalia into Kenya, sometime in the late 16th century.¹⁰⁹ In what has been called a “symbiosis”, the Nyali Coast upland was a topographically complex region occupied by multiple groups, including hunter-gatherers and farmers, engaging in different modes of subsistence.¹¹⁰ Trade then connected the upland region with the coast. An ecologically complex disease like plague, which thrives at high elevations yet flourishes as well in lower-elevation commensal rodent communities where it causes epizootics (and consequently, epidemics), might readily have moved through an area such as this. Indeed, this is precisely the scenario of resurgence of plague that is now being envisaged for early modern Europe, with plague persisting at higher elevations and periodically erupting into outbreaks in cities below.¹¹¹ In short, the story of the siege of Mombasa (which we know of only through European sources) may be a red herring: telling the story only through the perspective of the Europeans (the fatalities within the fort) and remaining blind to what might have been a larger epidemiological catastrophe.

- 64 Another possible avenue for the progenitor of the 1.ANT strain into equatorial East Africa would have been the Nile. The White Nile’s headwaters reach all the way to the Great Lakes, whereas the Blue Nile extends into Ethiopia. For the First Plague Pandemic, there is little specific evidence for plague’s effects in Egypt anywhere other than in the region of the Delta, and what there is suggests that, demographically, Egypt was readily able to bounce back from its losses.¹¹² In the Second Pandemic, in contrast, plague’s effects on Egypt were extreme. As already noted, there is ample evidence for plague’s repeated and severe attacks on Cairo, founded in 969 CE and situated at the point where the Nile River begins to branch into the Delta. After its first arrival in 1348, plague came in repeated waves, producing a total of forty outbreaks by the time the Ottomans claimed authority over Egypt in 1517.¹¹³ Plague’s effects seem to have been even more severe as it progressed into Upper Egypt.¹¹⁴ The town of Asyut, about half-way between Cairo and the First Cataract, and an important link in the trade routes that carried goods and slaves westward into Sudan, was nearly bereft of all inhabitants by the end of 1348.¹¹⁵ The system of water-level reporting on the Nile that had been in place since Pharaonic times effectively ceased in the years thereafter, with long-term consequences for the economic stability of Egypt, which lost its prized control over the unique irrigation system of the river and Delta. If the disruption of river traffic was as severe as the Arabic documents suggest, the cessation of economic activity may have effectively created a buffer, beyond which plague did not pass. The Christian kingdom of Nubia or Makuria (Arab. *Muqurra*; Nub. *Dotawo*), stretching from the First to the Fifth Cataract, collapsed sometime around 1360, the capital of Dongola being abandoned and a makeshift new capital being established further downriver at Daw (Gebel Adda); although recent archaeological finds show some continuing occupation of the area, that Nubia suffered political and economic disruption in the second half of the 14th century is clear.¹¹⁶ Egypt continued to have a particularly severe history with plague throughout the early modern period, with seasonal outbreaks seemingly beginning in Upper Egypt and moving northward. Such extreme devastation, which drastically reduced the economic activity that so effectively moved plague over long distances,

may itself be an argument against further expansion of plague infestation up the Nile and into the White Nile and thence to the Great Lakes. This is a topic on which bioarchaeological and palaeogenetic work could contribute importantly.

- 65 The final possibility is that the lineage of plague now found in Uganda and Kenya and the Democratic Republic of Congo arrived from their mutual northern neighbor, Ethiopia. None of the 20th-century historical or epidemiological accounts of plague in Africa include Ethiopia, save for a nod to its possible role in the Justinianic Plague, which was already noted above.¹¹⁷ Nevertheless, an exercise in ecological niche modeling, published in 2008, suggested Ethiopia's continuing suitability as a plague environment.¹¹⁸ Marie-Laure Derat has brought forward suggestive evidence for plague's effects on Ethiopia during and immediately after the Black Death. As Derat notes, the history of Ethiopia's late medieval and early modern experience with plague is in its infancy, and the specific routes and circumstances that might have brought plague to Ethiopia have yet to be investigated.¹¹⁹ The lack of any aDNA from the region means, for example, that there is as yet no way to know whether three reported outbreaks of plague in the 15th century—in 1403, 1430, and then between 1454 and 1468—reflect new introductions of plague into Ethiopia, or resurgences of strains that had focalized there after the Black Death. In their timing, at least, all of these accord approximately with outbreaks recorded not simply for Egypt, but for southern Russia as well.¹²⁰ Moreover, the importation of the western Christian plague saints, Roch and Sebastian, further suggests a society trying to adapt to a new disease regime.¹²¹ A further indication of a major environmental disruption are two major migrations out of Ethiopia in the early 16th century: the Funj, a population of cattle herders, who move to the northwest, down the Blue Nile into the Gezira, the region near the confluence of the Blue and White Niles in the Sudan; and what Kropáček has called “a chain reaction of population movements everywhere in the neighbourhood, including perhaps even the southward push of Nilotes from the upper Nile to the equatorial lakes”.¹²²
- 66 In sum, we cannot yet specify the route of 1.ANT into the Great Lakes region because we still know so little about the experiences of plague in surrounding areas. The overall narrative of 1.ANT is currently known only from its two endpoints—its separation from the main Branch 1B *Y. pestis* lineage sometime in the 15th or 16th century, and its presence in East Africa in the 20th and 21st centuries. Should any aDNA be retrieved from any of the regions where 1.ANT is or was found, it will contribute importantly to filling in the stages of the organism's migrations and evolution in East Africa. But so will further interrogation of the cultural record of the Buganda and other peoples of the Great Lakes region, and investigation of cultural histories of plague in regions further to the north.

IV. Conclusion

- 67 As the other contributions to this special cluster suggest, there is reason to think that plague may have been more widespread in late medieval Africa, in the belt just below the Sahara as well as in Ethiopia, than previously contemplated in historical studies or currently postulated in genetics.¹²³ For equatorial East Africa, I have suggested here that the arrival of the 1.ANT lineage of *Y. pestis*, even if it was not “medieval”, was likely much earlier than the 19th-century introduction postulated by Sussman. I have also suggested that there are currently no grounds to connect the strain in East Africa with

India, nor to view its importation as being due to the arrival of European colonialism—neither in the 19th century (as postulated by Sussman) nor even in the late 17th century, as supposed by earlier stories of the outbreak at Fort Jesus. The genetic histories of *Y. pestis* in India and in East Africa, respectively, contradict outright the former suggestion, while the Baganda's own cultural narratives suggest a history of plague in the interior Great Lakes region that goes back at least to the mid-17th century. Whether or not there were also earlier penetrations of plague along the Indian Ocean littoral, as suggested by archaeological evidence, cannot yet be proven or disproven; at the very least, they are not contraindicated by what is currently known of the faunal infrastructure and permissive climate events, nor by the well-documented trade networks along the coast.

- 68 Despite these continuing questions, it has now been demonstrated that East Africa's early modern, and modern, history of plague is part of the broader history of the Branch 1 lineage of *Y. pestis*, which has traversed all five inhabited continents at one point or another in the past 800 years. 1.ANT, the earliest extant sub-branch to diverge from the main Branch 1 lineage, persists to this day in Kenya, Uganda, the eastern territories of the Democratic Republic of Congo, and Zambia because it found rodent hosts capable of keeping the chains of transmission active from year to year, generation to generation. Further genomic analysis may document that strains still found in Tanzania and other neighboring countries where plague remains a persistent enzootic also belong to the 1.ANT lineage, in which case their analysis will contribute to the interpretations offered here. As an enzootic, plague may well have been stirred up in new ways by colonialist activities in the modern period: cotton production and the railways in Uganda, for example, have both been suggested as factors in the heightened outbreaks of the late 19th and early 20th century.¹²⁴ But this study has suggested that the genetics, linguistics, and even the testimony of 19th- and 20th-century colonialists document plague's much older, much deeper penetration of this complex landscape. If, as I suggest, East Africa's history with plague goes back considerably longer than the 150-year period posited by Sussman, then there is a great deal of work here for Africanist historians to assess the ecological and epidemiological challenges of life in East Africa in the early modern period. Indeed, the apparent pervasiveness of plague in these regions for the past several centuries may be an important link in histories of migration, cultural development, and health-seeking practices that historians of East Africa have already noted.¹²⁵
- 69 Methodologically, the present study makes an argument not only for the importance of interdisciplinarity, and its increasingly articulated goal of consilience, but also for taking seriously the tasks of global history. Consilience (meaning literally “jumping together”) has been variously defined as a kind of intense multidisciplinary, especially linking the sciences and humanities.¹²⁶ The present study has shown the need to bring genetic, linguistic, and environmental-historical questions to bear on explaining the presence of 1.ANT in equatorial East Africa. More than that, however, this study has been framed from the perspective of global history, one of whose precepts is to address questions from multiple scales. The present study has little to add to debates about the exact causal mechanisms that moved plague to equatorial East Africa (such as the animal hosts or ectoparasite vectors involved in plague transmission), or any climatic factors that may have been involved in fulminating episodes of plague. It has shown, however, that insights about bacteria in England and Russia can have implications for

societies half a world away. As has been demonstrated with respect to the Indian Ocean world, the genetic histories of globally distributed pathogens can be used to outline experiences with infectious diseases even in those areas where aDNA has not yet been recovered.¹²⁷

- 70 Finally, solving the particular question of the origins of the 1.ANT lineage contributes not only to historical questions. Populations in East Africa have likely been exposed to this organism for generations, and, in addition to Madagascar, they still bear the burden of more than 95% of human plague cases in the world every year.¹²⁸ Yet the East African and Malagasy strains of plague, although both in the general Branch 1 lineage, are very different organisms, with over 100 SNPs separating them, suggesting that both clinical and epidemiological research is needed to better understand the differing experiences of plague on the mainland and on Madagascar. Understanding the African history of plague may well be the beginning of efforts to tame this killer once and for all.

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NOTES

1. Overviews of developments in global health history in general, and plague research in particular, can be found in M.H. GREEN, 2017; M.H. GREEN, 2018a. Although there was an older belief that *Y. pestis* took its origin in Africa (e.g., R. DEVIGNAT, 1953, 1954), that line of thinking was challenged by geneticists in 2004 (see M.H. GREEN, 2014b) and has been repeatedly disproven by the findings of Bronze Age plague in northern Eurasia.
2. The beginning date of the Justinianic Plague, in the sense of the date of divergence of the specific strains of *Y. pestis* involved in the pandemic, is currently being recalibrated. Transmission out of the long-term reservoir of *Y. pestis* in central Eurasia likely occurred several centuries before the actual human outbreaks reported in the Mediterranean in 541.
3. K.I. BOS *et al.*, 2011; K.I. BOS *et al.*, 2016; M.A. SPYROU *et al.*, 2016; A. NAMOUCHI *et al.*, 2018; M.A. SPYROU *et al.*, 2018b.
4. Available surveys of the history in Africa of the disease we now understand to be plague are valuable for documenting where the disease is or has been present in the past century and a half. None, however, save Spinage (who was writing too early to capture the most recent developments in plague aDNA), have factored in the new narratives from genetics analyzed here. See, for example, J.A. MITCHELL, 1927; J. SCHWETZ *et al.*, 1929; E.N. THORNTON, 1930; R.E. BARRETT, 1933; J.I. ROBERTS, 1935; G.H.E. HOPKINS, 1949; D.H.S. DAVIS, 1953; R. DEVIGNAT, 1953; R. DEVIGNAT, 1954; S.B. NEERINCKX *et al.*, 2008; S. NEERINCKX *et al.*, 2010; C.A. SPINAGE, 2011; G.M. LA RUE, 2015; W.M. LOTFY, 2015; M.A. MALEK *et al.*, 2016; S.S. NYIRENDA *et al.*, 2016.
5. G. MORELLI *et al.*, 2010a and 2010b. For a full listing of samples used for that study, many of which were only partially sequenced, see their Supplemental Table 3, available at <http://wrap.warwick.ac.uk/81946/>.
6. N. CABANEL *et al.*, 2013.
7. For example, plague is never mentioned in N. KODESH, 2008; N. KODESH, 2010; or R. MARCHANT *et al.*, 2018.
8. M.H. GREEN, 2014b.
9. G.D. SUSSMAN, 2016, p. 353.
10. On the concept of consilience, see the Conclusion.
11. For the larger context of infectious disease history in Eurasia, see M.H. GREEN, 2018b.
12. S. RASMUSSEN *et al.*, 2015; A. ANDRADES VALTUEÑA *et al.*, 2017; M.A. SPYROU *et al.*, 2018a.
13. G.A. EROSHENKO *et al.*, 2017; V.V. KUTYREV *et al.*, 2018; M.H. GREEN 2018c.
14. The best summary of the important developments of the 1990s up through 2010 is L.K. LITTLE, 2011.
15. J. PARKHILL *et al.*, 2001.
16. G. MORELLI *et al.*, 2010a and 2010b.
17. K.I. BOS *et al.*, 2011. On the problem with the dating of one of these samples, see below.
18. Y. CUI *et al.*, 2013.
19. In M.H. GREEN 2018c, I argue that a consilient approach to genetics, epidemiological surveillance data, and historical documentary records supports the most recent estimated date for the polytomy: around the end of the 12th or the beginning of the 13th century, proposed by M. SPYROU *et al.*, 2018a. Y. CUI *et al.*, 2013, had previously suggested a date ca. 1268 (with a 95% confidence interval of 1142 to 1339).
20. L.K. LITTLE, 2011, was just going to press when the results of K.I. BOS *et al.*, 2011, were announced. An early articulation of what a molecular historical epidemiology would look like was M. MCCORMICK, 2007.
21. For a summary of this new type of epidemiology, see the literature cited in M. WOROBAY, 2017.
22. The future potential of this work is summarized in N.F. ALIKHAN *et al.*, 2018.

23. From the 1950s to the early 2000s, René Devignat's laboratory-derived classificatory system was used. G. SUSSMAN, 2016, p. 335-36, gives a useful summary of this classification system, which was widely adopted in scientific reporting on plague for decades. See also Z. QI *et al.*, 2016. In the present essay, I use only the modern sub-branch (or "phylo-group") designations as standardized in Y. CUI *et al.*, 2013. For aDNA strains, I use the city designations and, where further differentiation is required, the archeological sample number.
24. There may have been more. The strain known as "Angola" (0.PE3 on fig. 1), which is currently known from a single isolate that has no clear provenance history, is basal to the Justinianic Plague strain. See M.H. GREEN, 2014b, p. 40.
25. By "modern" here, I mean isolates collected using laboratory methods for preservation and analysis. These will all postdate the laboratory revolution in microbiology that started in the late 19th century.
26. In what follows, I am drawing for geolocation information and dates of collection on the supplemental data in G. MORELLI *et al.*, 2010b, Supplemental Table 3, available at <http://wrap.warwick.ac.uk/81946/>, which used partial genome analyses to type isolates as well as whole genome sequences; and Y. CUI *et al.* 2013, Dataset S1, available at <http://www.pnas.org/content/110/2/577/suppl/DCSupplemental>, which is based exclusively on whole sequences. Genome sequences published subsequently are cited individually.
27. M.H. GREEN, 2018c.
28. Y. CUI *et al.*, 2013, sequenced sample MGJZ12 from Mongolia. Other isolates of 4.ANT have been retrieved from southern Siberia, as reported by V.V. KUTYREV *et al.*, 2018.
29. The host species of 3.ANT strains are reported in Y. CUI *et al.*, 2013. L.M. KUKLEVA *et al.*, 2015, document additional isolates of 3.ANT2 in Mongolia but provide no information on hosts.
30. T.F. HAJDAROV [KHAYDAROV], 2015, 2016, 2017a, 2017b; and U. SCHAMILOGLU, 2017.
31. I introduced the labels for Branches 1A (documented thus far only from aDNA samples in Western Europe) and 1B (found in the London 6330 and Bolgar City aDNA, and all living strains of Branch 1) in M.H. GREEN and B. SCHMID, 2016. For additional post-Black Death genomes in Europe, see now M.A. SPYROU *et al.*, 2018b. For a diagram showing all five post-polytomy lineages, see M.A. SPYROU and K.I. BOS, 2017.
32. Both the London 6330 genome and that from Bolgar City have one unique polymorphism, not yet found anywhere else on the main Branch 1B lineage. On the Bergen op Zoom genome, see A. NAMOUCHI *et al.* 2018.
33. Y. CUI *et al.*, 2013; Y. ZHANG *et al.*, 2018; X. XU *et al.*, 2018.
34. P. WANG *et al.*, 2016, report the presence of a strain similar to 1.IN2, and possibly ancestral to later Branch 1 strains, in Yulong province; this would be the first sign of Branch 1's movement toward Yunnan. My thanks to Yujun CUI (correspondence, 10 December 2016), for information on the phylogenetic positioning of the WANG *et al.*, 2016, isolate. See also P. WANG *et al.*, 2018.
35. G. MORELLI *et al.*, 2010a, reported one isolate of 1.ORI3k that was collected in 1950 in Elizabethville in the Congo (sample #IP678 = item 190). Leaving aside Madagascar, whose heavy plague infestation is solely the result of post-1898 introductions, this was the only sample of Third Pandemic strains reported from East Africa.
36. G. MORELLI *et al.*, 2010a; Y. CUI *et al.*, 2013; B.K. KURMANOV *et al.*, 2016; N.Y. NOSOV *et al.*, 2016; X. XU *et al.*, 2018; Y. ZHANG *et al.*, 2018.
37. J.J. KINGSTON *et al.*, 2009; N. CABANEL *et al.*, 2013.
38. A recently discovered strain in the Central Caucasus Mountains, 2.MED0 (sample C-627), is ancestral to all other 2.MED strains; see G.N. ODINOKOV *et al.*, 2013; N.Y. NOSOV *et al.*, 2016; and V.V. KUTYREV *et al.*, 2018. The next most ancestral strains of 2.MED, 2.MED3, are found over 3000 km to the east (Y. CUI *et al.*, 2013, Supplemental Data). On the Mongol postal roads, see H. SHIM, 2014.
39. M.L. GUPTA and A. SHARMA, 2007; J.J. KINGSTON *et al.*, 2009; K.N. MAHALE *et al.*, 2014.

40. On the larger trading networks that linked early modern India and Central Asia, see, for example, E.A. ALPERS, 1976; R. MATTHEE, 1994; S. SUBRAHMANYAM, 1995; R. MATTHEE, 1999; S.C. LEVI, 2002; E. MONAHAN, 2016.
41. JAHANGIR, 1909-1914, p. 330, 442.
42. M.H., GREEN, L. JONES, forthcoming.
43. In fact, SNPs occur quite commonly. A study of 34 *Y. pestis* isolates collected in the American West between 1980 and 2006 counted 42 SNPs in a targeted comparison (J.L. LOWELL *et al.*, 2015). A study of an active plague outbreak in Madagascar between 1991 and 1999, based on a lineage with 39 isolates, identified 13 new SNPs (A.J. VOGLER *et al.*, 2013; for their study, vogler *et al.* used the multiplier 1.7×10^{-10} mutations per nucleotide per generation to estimate how many SNPs would naturally be generated in the population).
44. M. ACHTMAN, 2012.
45. M. ACHTMAN, 2016 and 2017.
46. Y. CUI *et al.*, 2013, sequenced sample MGJZ12. On the other isolate of 4.ANT, from southern Siberia, see B.K. KURMANOV *et al.*, 2016.
47. The 1.ANT strains to be discussed here accumulated about 100 SNPs in the past 500 years. The lineage seeded in Europe by the Black Death in 1347-48, Branch 1A, accumulated 88 SNPs by the time it burned out in the 18th century.
48. Y. CUI *et al.*, 2013, p. 577: “we identified 2,326 SNPs from 133 genomes of *Y. pestis* strains that were isolated in China and elsewhere. [...] All but 28 of these SNPs represented mutations that happened only once within the genealogy, and they were distributed essentially at random among individual genes. Only seven genes contained a significant excess of nonsynonymous [function-changing] SNP”. With the discovery of the Bronze Age strains of *Y. pestis* in 2015, the number of identified SNPs in the *Y. pestis* phylogeny has grown, but it still remains in the lower four digits. On SNPs as signals of historical change, see M.H. GREEN, B. SCHMID, 2016.
49. See the literature cited in footnote 4 above.
50. M.H. GREEN, 2014b, for summary, to which can be added R.M. GONZALES, 2009: chap. 2, paragraphs 78-79, and chap. 6, paragraph 39. See now G. CHOUIN, 2018, for a historiographical overview of the question.
51. L.K. LITTLE, 2007; K. HARPER, 2017; T.P. NEWFIELD, 2018b (2016).
52. K. HARPER, 2017; T.P. NEWFIELD, 2018a.
53. M.H. GREEN, 2018b; M.H. GREEN, 2018c; M. KELLER *et al.*, 2018.
54. M. FELDMAN *et al.*, 2016; G.A. EROSHENKO *et al.*, 2017; M.A. SPYROU *et al.*, 2018a. My thanks to Mark Achtman and Zhemin Zhou for assistance assessing data on the newly discovered 0.ANT5 strain in V.V. KUTYREV *et al.*, 2018, via their new platform, *EnteroBase* (N.F. ALIKHAN *et al.*, 2018).
55. M. FELDMAN *et al.*, 2016; M. KELLER *et al.*, 2018. No aDNA has thus far been retrieved from the Justinianic Plague grave sites in North Africa, though mass grave sites possibly connected with the Justinianic Plague have been identified in Tunisia and Egypt. See M. MCCORMICK, 2016, items 11 and 17.
56. M.A. SPYROU *et al.*, 2018a.
57. M.H. GREEN, 2018c.
58. C. TSIAMIS *et al.*, 2014, summarize the evidence for trade between the Red Sea and Pelusium. See also K. HARPER, 2017, who assumes an Indian Ocean route. As noted above, earlier authors (particularly Devignat) believed Africa to be the original home of plague.
59. W. SCHEIDEL, 2001.
60. G. HATKE, 2013, section 3.3.

61. P. MAYERSON, 1993. Cf. P. SARRIS, 2007, p. 121-122; M. MCCORMICK, 2007; M.H. GREEN, 2014b, p. 47; M.H. GREEN, 2015. See also M.G. MORONY, 2007, p. 63, for a Syriac account identifying Ethiopia as the origin of the first outbreak of the Justinianic Plague.
62. Y. GEBRE SELASSIE, 2011.
63. M.H. GREEN, 2014a, p. 18.
64. On Tunisia, see M. TALBI, 1981, p. 221. On Egypt, see the multiple documents indexed for “plague” in the CAIRO GENIZAH, 2018. On Mesopotamia, see M.G. MORONY, 2007, p. 68.
65. M.H. GREEN, 2018b and 2018c.
66. S. BORSCH, T. SABRAA, 2017.
67. G. CHOUIN, 2018; M.-L. DERAT, 2018; D.E. GALLAGHER, S.A. DUEPPEN, 2018.
68. To the evidence collected in M.H. GREEN, 2014b, now add: J.E.G. SUTTON, 1993; J.E.G. SUTTON, 1997; R.M. GONZALES, 2009; S. WYNNE-JONES, A. LAVIOLETTE, 2017, p. 220, 223, 288, 623; G. CHOUIN, 2018.
69. J.E.G. SUTTON, 1993 and 1997.
70. On the environmental landscape of equatorial East Africa for the past 6,000 years, see R. MARCHANT *et al.*, 2018. Genetic studies of *Y. pestis* have been most extensive for Kenya, Uganda, and the Democratic Republic of Congo, which will thus form the focus of the present analysis. For Zambia, see S.S. NYIRENDA *et al.*, 2018; and also B.M. HANG'OMBE *et al.*, 2012; S.S. NYIRENDA *et al.*, 2017. Although plague is still documented in Tanzania, with genetic analysis now being used in its assessment, no studies have yet reported the genotype of the strains found there; see B.S. KILONZO, R.S. MTOI, 1983; M.H. ZIWA *et al.*, 2013; P. HIERONIMO *et al.*, 2014; M. HUBEAU *et al.*, 2014; and R.H. MAKUNDI, 2015. For Rwanda and Burundi, both established as independent polities in 1962, I have found no specialized literature on plague.
71. Y. CUI *et al.*, 2013, p. 580, Table 1, and Supplementary Information, Appendix, Fig. S8.
72. M.A. SPYROU *et al.*, 2018a, p. 8, Supplementary Figure 6, and Supplementary Table 11, propose several new estimated dates for key points in *Y. pestis* emergence, but do not offer an estimate for 1.ANT's emergence. Divergence dates proposed for other points along the extant Branch 1B lineage provide *termini* sometime between 1309 CE (641 BP), the estimated divergence date between Branches 1A and 1B, which was a prelude to the *pestis secunda* of the 1360s, and ca. 1599 CE (351 BP), the point at which the later extant strains of Branch 1B, the 1.IN strains, began to diverge from each other. (For the placement of the 1.IN strains on the *Y. pestis* phylogeny, see fig. 2.) Confidence intervals were not provided by M.A. SPYROU *et al.*, 2018.
73. There is as yet no study of the experience of plague throughout all these areas, though for the early Ottoman empire there is now the superb work of N. VARLIK, 2015. For the Safavid empire, see also R. MATTHEE, 1994 and 1999.
74. Again, I refer to the supplementary data accompanying G. MORELLI *et al.*, 2010a. For historical sources on plague in Iran, see A.H. SHAHRAKI *et al.*, 2016; M. ASSMAR *et al.*, 2018.
75. L.B. RESPICIO-KINGRY *et al.*, 2016.
76. J.L. LOWELL *et al.*, 2015; M. KOSOY *et al.*, 2017.
77. S. NEERINCKX *et al.*, 2010.
78. As in most other areas of the world where plague has established reservoirs, the documented rodent hosts extend beyond the commonly cited black rat (*Rattus rattus*). A survey of the East African rodents involved in plague can be found in, for example, D.H.S. DAVIS, 1953.
79. J. SCHWETZ, 1929; J. SCHWETZ *et al.*, 1929; R. Devignat, I. Vincke, 1937.
80. E. BERTHERAT *et al.*, 2011.
81. J. KOPONEN, 1988, p. 657, 659, citing R. BURTON, 1859, p. 367.
82. R. KOCH, 1912.
83. G. SUSSMAN, 2016, p. 333.
84. See, for example, J.I. ROBERTS, 1935.

85. C. CHRISTY, 1903.
86. R.E. BARRETT, 1933, p. 161. I have not yet been able to consult a copy of E.N. THORNTON, *A report on an investigation into plague in the Protectorate of Uganda*, 1930.
87. J.I. ROBERTS, 1935, p. 213.
88. C. CHRISTY, 1903.
89. C. CHRISTY, 1903, p. 1266.
90. For the single piece of genetic evidence of Third Pandemic strains reaching East Africa, see footnote 35 above.
91. Similarly, we find testimony of an indigenous name for the disease in Uganda. As S. ORACH-ORACH, 2003, reports, “The first local name used for the disease and is still popularly known now is ‘Zukpa’, a Lendu word for plague. This word became popular *because in the old days almost all epidemics used to start among the Lendu tribe in Congo* (now the Democratic Republic of Congo)” (emphasis added).
92. J. ROSCOE, 1911, p. 309; H. MÉDARD, 2005. See also C. PENNACINI, 2009. “Baganda” is the name of the people, “Buganda” the name of the country.
93. N. KODESH, 2010, mentions Kawumpuli only in passing (p. 177), never discussing his particular significance. S. AMIN, 2015; cf. J. ROSCOE, 1911, p. 309.
94. C. CHRISTY, 1903, p. 1267. For important analysis of this episode of colonial science, see P. MALLOY, 2014.
95. A map suggesting a Nile entry for 1.ANT was included, without explanation, in D.M. WAGNER *et al.*, 2014.
96. G. MORELLI *et al.* 2010; Y. CUI *et al.* 2013.
97. R. HYMES, 2014.
98. B. ZHAO, 2015.
99. A. HAOUR, 2011. M. DOLS, 1974, p. 372, claims that “From the sixteenth century we know that plague epidemics have customarily spread from east Africa through the Sudan by the caravan traffic to Egypt and North Africa”. But he cites no source. My thanks to Suzanne Blier, who suggested possible transmission from oasis to oasis along this route in our discussions at William and Mary in 2016.
100. Transmission across long, open expanses of desert must also be inferred if, as suggested by D.E. GALLAGHER and S.A. DUEPPEN, 2018, plague also reached West Africa in this period.
101. S. WYNNE-JONES and A. LAVIOLETTE, 2017, offer the most recent survey of the scholarship.
102. M.E. PRENDERGAST *et al.*, 2017.
103. M.H. GREEN, 2014b, p. 44-45. To the examples given there, there might now be added the apparent late medieval population shifts at Gede (M. PAWLOWICZ, 2017), northern Madagascar (C. RADIMILAHY, 2017), and Tanzania (R.M. GONZALES, 2009).
104. On developments of the mid- and later 16th century that radically increased the amount of spice trade moving through the Indian Ocean, the Persian Gulf, and the Red Sea, see G. CASALE, 2006 and 2007.
105. N. VARLIK, 2015, addresses the circulation of plague within the Ottoman empire, but it will be necessary to add palaeogenetic evidence to track plague’s path *through* it.
106. J.I. ROBERTS, 1935, p. 206; C.A. SPINAGE, 2011, p. 1343. Cf. C.R. BOXER, C. DE AZEVEDO, 1960; J.S. KIRKMAN, 1974.
107. H. SASSOON, 1982, p. 104.
108. H. SASSOON, 1982, p. 103.
109. A.J. H. PRINS, 1952; J. KIRKMAN, 1983.
110. C. SHIPTON *et al.*, 2013.
111. A. CARMICHAEL, 2014.

112. S. MITCHELL, 2014, p. 488-489; K. HARPER, 2017, p. 266-268. W. SCHEIDEL, 2001, does not think Egypt was much affected at all by plague in Antiquity.
113. S. BORSCH, T. SABRAA, 2016 and 2017.
114. M.W. DOLS, 1977, p. 60, 161, 165, notes progression of plague into Upper Egypt as early as 1349, but never suggests further movement southward. In addition to the evidence collected for Upper Egypt by Dols for 1406, 1416, 1430, 1438, 1476-77, 1492, and 1507, B. SHOSHAN, 1981, finds additional instances for plague's presence there in 1358, 1420, 1422, 1428, and 1436. 1460 is identified as a year when "all of Egypt" was affected. For the effects of plague on Egypt generally, see S. BORSCH, T. SABRAA, 2016; G.M. LA RUE, 2015 and G.F. PETRIE, R.E. TODD, 1924.
115. S. BORSCH, T. SABRAA, 2017.
116. My thanks to Stuart Borsch for sharing with me his preliminary observations on the disruption of the system of Nile monitoring. Although the late medieval collapse of Nubia is well known (e.g., G. RUFFINI, 2012; W. GODLEWSKI, 2013), to my knowledge only L. KROPÁČEK, 1984, p. 402, has suggested that plague may have been a factor, which he mentions as a force driving Bedouin groups out of Egypt southward into Nubia. The on-going analysis of recent archaeological finds from the area may contribute to this question.
117. J.A. MITCHELL, 1927; J. SCHWETZ *et al.*, 1929; J.I. ROBERTS, 1935; D.H.S. DAVIS, 1953; R. DEVIGNAT, 1953; R. DEVIGNAT, 1954; S. NEERINCKX *et al.*, 2010; C.A. SPINAGE, 2011; G.M. LA RUE, 2015; W.M. LOTFY, 2015; M.A. MALEK *et al.*, 2016; S.S. NYIRENDA *et al.*, 2016. R.E. BARRETT, 1933, cites at second hand the opinion that "the original home of [*Xenopsylla cheopis*] [a flea widely implicated in plague transmission] is the valley of the Nile in the East African sub-division of the Ethiopian Plateau", but offers no further elaboration on the geographic association.
118. S.B. NEERINCKX *et al.*, 2008.
119. M.-L. DERAT, 2018. The evidence for plague's arrival in Ethiopia in the latter half of the 13th century accords with an emerging understanding that the 14th-century Black Death had a 13th-century antecedent; cf. M.H. GREEN, 2018c.
120. T.F. HAJDAROV [KHAYDAROV], 2016. T.F. HAJDAROV [KHAYDAROV], 2017a, lists the following dates for outbreaks in Russia: 1351/52, 1360, 1363, 1388, 1409, 1414, 1410, 1417, 1419-1430, 1442, 1465-1467, 1478, 1486/87, 1506, 1521, 1523, 1543, 1551, 1561/62, 1566, 1584-1598.
121. M.-L. DERAT, 2018.
122. L. KROPÁČEK, 1984, p. 418-19; cf. C. EHRET, 1984.
123. D.E. GALLAGHER, S.A. DUEPPEN, 2018; M.-L. DERAT, 2018. See also G. CHOUIN, C.R. DECORSE, 2010; G. CHOUIN, 2013.
124. R.E. BARRETT, 1933; J.D. FORRESTER *et al.*, 2017.
125. R.M. GONZALES, 2009; N. KODESH, 2010.
126. M. MCCORMICK, 2011; M.H. GREEN, 2014b, p. 50-51; T.P. NEWFIELD, I. LABUHN, 2017; J. HALDON *et al.*, 2018.
127. M.H. GREEN, L. JONES, forthcoming.
128. E. BERTHERAT *et al.*, 2011; E. BERTHERAT, 2016.

ABSTRACTS

Did the Black Death, the famous, devastating plague pandemic that struck the Mediterranean and Western Europe in the mid-14th century and seeded new strains of the pathogen, *Yersinia pestis*, in new locales, also reach Sub-Saharan Africa? That it reached the Islamic North African littoral has never been in question: there is ample testimony from Arabic documentary sources for its devastating effects from Egypt to the Maghreb. But did it stop there? Archaeologists now have reason to believe that there was widespread abandonment of urban communities in West Africa in the 14th or 15th centuries. Focusing on the other side of the continent, the present essay argues that previously unutilized evidence—a “living archive” of genetic data combined with a new reading of linguistic and documentary evidence from the 14th through 20th centuries—suggests that plague did have a significant impact on pre-modern East Africa. The strains of *Y. pestis* currently found in East and Central Africa derive from one of the lineages involved in late medieval outbreaks in Central Eurasia. This post-Black Death strain, it is argued here, entered the continent most likely in the late 15th or early 16th century. This coincides with hitherto unexplained population migrations and other cultural signs of reactions to a deadly emerging disease. Although no aDNA (historical genetic material) for *Y. pestis* has yet been recovered from late medieval or early modern Africa, the present essay suggests that evidence for *Y. pestis*’s evolution and transcontinental movements, even those outside of Africa, can help archaeologists and historians draw Africa more centrally into the narratives of global history.

A-t-elle atteint l’Afrique subsaharienne, la peste noire, la fameuse et dévastatrice pandémie qui frappa le pourtour Méditerranéen et l’Europe occidentale au milieu du XIV^e siècle, et permit l’émergence de nouvelles souches du pathogène *Yersinia pestis* dans de nouveaux lieux ? Il ne fait aucun doute qu’elle a touché le littoral nord-africain : il existe nombre de témoignages dans les sources arabes sur ses effets dévastateurs, de l’Égypte au Maghreb. Mais s’est-elle arrêtée là ? Les archéologues ont désormais des raisons de croire que l’Afrique de l’Ouest fut le théâtre d’un abandon à grande échelle des nombreuses communautés urbaines au cours des XIV^e et XV^e siècles. Cet article, ancré lui en Afrique de l’Est, avance l’idée que des sources restées jusque-là inutilisées – une « archive vivante » constituée de données génétiques combinées à une relecture innovante de sources linguistiques et documentaires produites entre le XIV^e et le XX^e – suggèrent que la peste eut un impact significatif sur l’Afrique de l’Est avant le XX^e siècle. L’analyse génétique des souches de *Yersinia pestis* trouvées en Afrique de l’Est et en Afrique centrale montre qu’elles dérivent d’un lignage impliqué dans des flambées de peste en Eurasie centrale à la fin du Moyen Âge. Cet article suggère que ce lignage, plus tardif que celui qui causa la peste noire, pénétra le continent africain à la fin du XV^e ou au début du XVI^e siècle. Cette entrée coïnciderait avec des migrations de populations jusque-là inexpliquées, et avec d’autres signaux culturels qui seraient autant de réactions à l’émergence de la maladie responsable d’une forte mortalité. Bien qu’aucun échantillon d’ADN ancien (matériel génétique historique) datant du Moyen Âge tardif ou de la période moderne n’ait encore été retrouvé en Afrique, cet article suggère que les données concernant l’évolution et les mouvements transcontinentaux de *Yersinia pestis*, y compris ceux documentés en dehors de l’Afrique, peuvent aider les archéologues et historiens à donner au continent une place plus centrale au cœur de l’histoire globale.

INDEX

Keywords: *Yersinia pestis*, genetics, historical epidemiology, global history, Second Plague Pandemic, Uganda, Kenya, Democratic Republic of Congo, Zambia, India, Eurasia, Mongol Empire

Geographical index: Ouganda, Kenya, République Démocratique du Congo, Zambie, Inde, Eurasie, Empire mongol

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