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Taking "Pandemic" Seriously: Making the Black Death Global

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PANDEMIC DISEASE IN THE MEDIEVAL WORLD

RETHINKING THE BLACK DEATH

Edited by MONICA H. GREEN

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TAKING "PANDEMIC" SERIOUSLY: MAKING THE BLACK DEATH GLOBAL

MONICA H. GREEN

In 2009, when the most recent major monograph was published on life in an Italian city of the mid-fourteenth century, its author deferred judgment on whether the disease that struck Italy in 1348—"an infinite mortality the likes of which ha[ve] never been seen on earth"—was in fact plague as defined by modern science (Wray 2009: 1). The authors contributing to this, the inaugural issue of *The Medieval Globe*, no longer feel that such caution is necessary. Since 1998, several international teams of microbiologists have tested and contested the possibilities for establishing the presence of plague's causative organism, *Yersinia pestis*, in the physical remains of Europeans who died at various moments in premodern history when major epidemics were raging. The reason that there is scientific consensus now, when there was not before, is a function of two developments, both of them having to do with trajectories in genetics research in the past thirty years that have come together quite recently.

On the one hand, researchers have been exploring methods to capture and analyze "ancient" DNA (aDNA), by which they mean any genetic material from older remains. Because *Y. pestis* would be circulating throughout the bloodstream by the time it kills a person, and because the hard enamel of intact teeth could potentially preserve small amounts of blood found within the dental pulp, teeth became the focal point of attempts to retrieve *Y. pestis* from human remains. But the challenges of developing viable methods of extraction and analysis were significant. DNA, like

In addition to the individuals and institutions thanked in the Editor's Introduction to this volume, I would here like to acknowledge my debt to the participants at a conference held in Oxford, "Proto-Globalisation in the Indian Ocean World," in November 2013, and to Patrick Manning, Mark Horton, and Neil Kodesh for their guidance on incorporating Africa into this narrative. Funding for this project was provided by fellowships from the Institute for Advanced Study at Princeton (with funds from the National Endowment for the Humanities and the Willis F. Doney Membership Endowment) and the World History Center of the University of Pittsburgh (which has also subsidized open-access publication of this volume). Any views, findings, conclusions, or recommendations expressed in this publication do not necessarily reflect those of the National Endowment for the Humanities.

every other part of the body, begins to decay immediately after death, so degradation of the genetic material was the first of the challenges encountered by researchers. For example, the full genome of *Yersinia pestis* is about 5.6 million base pairs long. The fragments that researchers have had to deal with are rarely even fifty to seventy-five base pairs long. Add to this issue the problems of the material's possible contamination (which could occur when collecting it in the field, or in the lab, or at any point in between), and it is quite understandable, looking back on them now, why the "aDNA debates" of the late 1990s and 2000s were so intense. Already in 2004, however, another diagnostic mechanism—a protein assay that tested for an antigen produced uniquely by *Yersinia pestis*—was shown to be useful in determining the presence of *Y. pestis* not simply in modern diagnostics and epidemiological surveys, but also in historical samples. Meanwhile, studies reporting success in extracting *Y. pestis* aDNA kept appearing, each with more confidence, and by 2011 it was announced that the complete genome of *Y. pestis*, assembled from fourteenth-century remains, had been sequenced (Bos et al. 2011; Little 2011).

At the same time as this aDNA work was being pursued, highly sophisticated studies were being done of modern samples of *Y. pestis*, which is now a globally distributed pathogen. Scientific studies of *Y. pestis* have been going on since the late nineteenth century, but have increased in pace and intensity in recent years, both because *Y. pestis* is a useful model for studying pathogen virulence, and also because there are heightened concerns of bioterrorism and disease re-emergence (Ziegler 2014, in this issue). One main objective of phylogenetic studies of *Y. pestis*'s modern genome (which was first fully sequenced in 2001) has been to reconstruct the organism's evolutionary history. The principle is simple: by categorizing like genetic variations with like, the modern samples can be grouped into clusters, from which phylogenetic relationships can then be inferred. In other words, different modern strains are placed at different end points of a family tree, with inferred branches connecting back down to a common root. Doing this on the basis of long sections of the genome (or now, more commonly, using multiple samples of the whole genome itself) allows for analysis of the organism's phylogeny down to the level of individual base-pair changes (what are called single nucleotide polymorphisms, or SNPs).

The most critical development comes from the fact that both of these lines of research have now converged. Fusing the phylogenetic work and the aDNA work shows that the fourteenth-century genome does in fact fit onto a branch of the phylogenetic tree that had already been postulated (Cui et al. 2013; see **Plate 1 below**). In other words, the organism found in

historical human remains is not simply *Y. pestis*, but a kind of *Y. pestis* not too far distant in its genetic structure (and in its potential virulence) from the organism known in the world today. Is it possible that other pathogens were involved in causing the extreme mortality levels of the fourteenth century, in addition to *Y. pestis*? Yes, or at least we cannot rule out that possibility now. Is it possible that other strains of the organism might be found that complicate the narrative I have sketched above? Absolutely. Is it possible that science will keep doing what it always does, making new discoveries? We should hope so.[1] But the microbiological science, whose rigorous methods are becoming standardized and are proving equally fruitful in exploring the histories of other pathogens and organisms, is building up a mounting body of evidence.[2] This confirms that we know enough about the history of *Y. pestis* as a biological organism to structure research programs around certain shared assumptions about its character and behavior. This is not "fringe" science. It is leading-edge work and it demands the serious attention of historians and all others investigating plague in history.

But the skeptic will ask: So what if we can say definitively that people died of *Y. pestis* infections in disease outbreaks of the past? How does that change the work of historians or those working in other historicist disciplines? It is the purpose of this special issue of *The Medieval Globe* to argue that the new microbiology matters not simply because it solves the question "What was the disease?," but because in solving that question (as I believe it does) it opens up entirely new questions, ones we did not previously know we needed to ask. First and foremost, it grounds plague history in the eastern part of the Eurasian landmass. As will be explained in more detail below, study after study is narrowing in on the Tibetan Plateau and Qinghai as the likely place where *Yersinia pestis* diverged from the rela-

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1 I have omitted from this assessment a survey of climate science that may eventually implicate major geological or other events in the disturbed ecosystems that initiated the premodern pandemics. Although there is now compelling evidence for coincidental events (e.g., the dust-veil event of *c*. 535 as background to the Justinianic Plague, and a volcanic explosion *c*. 1257 as well as major climatic events in the 1340s that may have contributed to the Black Death), no evidence has yet been brought forth that would link such events directly to the biological evolution of *Y. pestis*. Such evidence may soon be forthcoming, however, since it is clear that alterations in the weather (like excessive rain) contribute to flea production and hence to the possibility of the spread of *Y. pestis*.

2 On continuing efforts to make aDNA research as methodologically rigorous as work on modern genetic materials, see Seifert et al. 2013, and papers presented at the 2013 aDNA conference at the Royal Society in London.

tively harmless soil pathogen *Y. pseudotuberculosis* and became one of the most highly lethal organisms in the world. Plague's history is now firmly on the map in a way it has never been before, and that (as well as other considerations) demands that we rethink almost everything that has been said to date about the disease. As historian Lester Little noted, referring to the significance of the Tübingen-McMaster study of 2011 that reconstructed the Black Death genome from a London cemetery (Bos et al. 2011):

> They are calling for work on the contexts of particular epidemics, including such factors as climate, presence and characteristics of vectors, interactions with concurrent diseases, living conditions, and means of communication and travel. If nothing else, this finding is an open invitation to historians, among others, to re-enter the fray. (Little 2011: 289–90)

That "fray" is already quite active, with various participants coming from different disciplinary backgrounds and, with some cause, bringing with them a wariness of differing approaches. At the conference "Human Evolution, Migration and History Revealed by Genetics, Immunity and Infection," which was held in London in 2011, the organizers (two biologists and a physician) expressed concern that relying on what was already known to historians offered too limiting a view of the history of pathogens and human evolution.

> [W]e have, often, an equally fragmentary account of the historical timeline to which we seek to tether the twists and turns of the biological story. The warning is that we should resist the temptation to link relationships casually through *cherry picking those milestones in human history that are best recorded*. While it is tempting to look for the correlates in evolutionary selection of the Black Death, the fall of the Roman Empire or the colonization of South America, how many equally or more dramatic bottlenecks have been imposed in the past millennia by pathogens, climate change or natural disasters for which we lack a good historical record (or any awareness at all)? (Altmann et al. 2012: 765–66, my emphasis).

Altmann and colleagues then go on to suggest that *Yersinia pestis* should be excepted from this caution on the grounds that it is of relatively recent origin and genetically monomorphic (not showing significant levels of genetic diversity). Yet from the historian's perspective—we who study humans and only secondarily the pathogen—plague's history is still stunningly lacunous. Yes, there is no question that a major plague outbreak happened between 1347 and 1353 in the Mediterranean basin and Western Europe. Whether we estimate its highest mortality rates at 30%, 40%, or 60% (as have variously been proposed), its effects were cata-

strophic. But while we may have good—in some cases, excellent—records to document human fatalities from plague in those areas, the new plague science tells us that there is much about this disease of which we still lack awareness, including most of the factors that caused it to become, for many centuries, a defining feature not only in the landscape of Western Europe, North Africa, and the Middle East, but also in Russia, India, and East and Central Africa: all the latter being places where it still remains in enzootic foci. As will be noted below, most of the new plague science is also relevant to studies of the First Plague Pandemic, the Justinianic Plague which raged, at least in the Mediterranean basin, from *c*. 541 to *c*. 750 (Little 2007; Mitchell 2014).

This essay, then, argues that it is no longer helpful for historians to take a posture of scientific agnosticism when it comes to the history of plague. I will focus here mainly on the Second Pandemic, sketching out two areas where we have yet to search fully for plague's historical effects: its range across different host species and its geographical extent outside of the Tibetan Plateau and Qinghai region. The revised zoology reminds us to take into account not only the incidental animal species that immediately spread *Y. pestis* to humans in epidemic outbreaks, but also the many other species involved in the long-distance spread of the disease. Taking the genetics science seriously also demands that we revise our geography of plague's history. This new geography is not limited to the Mediterranean and the lands surrounding it. It extends from the Tibetan highlands and Western China overland into Western Eurasia but also, I will suggest, southward, into the Indian Ocean basin and nearly all points connected to it.[3]

An Elephant of a Disease: Widening the Zoological Lens

The Indian tale of the six blind men and the elephant is a useful metaphor for thinking about plague. Believing that the elephant was like a fan (by touching only its ear) or like a tree trunk (by feeling only its thick legs), individual blind men could not perceive the huge and complex beast in its entirety. In thinking about plague, we too need to keep in play all the elements that allowed a single-celled, non-motile bacterium to become a (semi-)global pandemic. There is plenty of scientific literature to explain

3 On the possibility that plague spread east and southeast from the Tibetan-Qinghai Plateau in the medieval period, see Hymes 2014, in this issue. To date, I know of no evidence—genetic, skeletal, or documentary—that suggests that plague reached either Madagascar or Australia prior to the Third Pandemic radiation at the end of the nineteenth century.

the mechanisms connecting pathogen, natural hosts, accidental (or intermediate) hosts, environmental and climatic factors, and human elements. On some issues there is widespread consensus; on others, research is only beginning. Here, I wish to focus on the need for more historical study of the animal hosts that may be involved in plague transmission. We need to look at more than the elephant's tail.

The Tibetan-Qinghai Plateau, whose average elevation is several thousand meters above sea level, is as far removed in its climatic and ecological environment from most of the areas hardest hit by plague in the fourteenth century as it is in geographic distance. *Y. pestis* had to move across many animal species (arthropod vectors as well as mammalian hosts) as well as climatic zones in order to cause the massive human mortality that it did in the fourteenth century (and episodically thereafter). Tracing *Y. pestis* across many animal species will be a necessary part of retracing its geographical spread. That includes re-examining the role of one particular species, *Homo sapiens*, who is likely responsible for plague's most extensive dissemination. But we'll get to that species later.

As Michelle Ziegler explains (2014, in this issue), the trifold litany of plague modes usually cited in historical accounts—bubonic, pneumonic, and septicemic—needs to be broadened now to include the gastrointestinal. As Ziegler notes, these different presentations of plague are better thought of by their method of transmission: insect bite and abrasion or cutting (bubonic and septicemic), inhalation (pneumonic), and ingestion (gastrointestinal), respectively. These are not different diseases, of course. They are all caused by *Y. pestis*. They differ only in the path by which the organism enters the body and the speed with which it reaches the bloodstream, and, in clinical terms, by the symptoms induced when different immune responses are triggered, depending on what tissues the organism first encounters (Pechous et al. 2013). It is important to begin with this distinction, because while any or all of these modes of transmission may be operative in a given outbreak, each can have its own characteristic microenvironment.

Most historical work has focused on bubonic plague, and the rat-flea mode of transmission that was documented early in the Third Pandemic at the turn of the twentieth century. But rats are not the only carriers of plague: they are not even efficient ones in terms of the organism's evolutionary survival.[4] There are several hundred animal species that can be

4 A new line of research has raised the question of whether human lice might also be a factor in plague's spread (e.g, Ayyadurai et al. 2010). If so, this would radically increase the importance of studying practices of clothing exchange and resale,

infected with plague.[5] For studying human outbreaks, interest has focused on "commensals," those species that "share our tables," living around human settlements, eating our foodscraps. But the new geography of plague tells us they we need to look at much wider biological systems to understand how *Y. pestis* moved many thousands of miles across varying ecological environments to reach large human population settlements. Beyond commensal rodents, we need to look at wild rodents, lagomorphs (hares, rabbits, etc.), and ruminants (cattle, goats, sheep, camels, deer), as well as the carnivore species that prey on them.

For their 2013 study of historical variations in the mutation rates of *Y. pestis*, Cui and colleagues drew on 133 complete genome sequences of the organism, which had been collected from nineteen different mammalian genera infected with *Y. pestis*, as well as from a variety of fleas, ticks, and lice that prey on them (Cui et al. 2013, supplemental data: 10). Eleven of the mammalia were rodent species, one was a type of lagomorph, and then there were other various grazing animals (sheep, bharals) and carnivores (badgers, weasels, canids, and foxes). There were also humans. But the list of susceptible animals is far larger than that. A surveillance study in India carried out between 1989 and 2007 found that *Tatera indica cuvieri* (Hardwicke), the Indian gerbil, made up the highest percentage of plague-infected rodents (41.9%), followed by *Rattus rattus rufescens* Gray and *Rattus rattus wroughtoni* Hinton, and finally the lesser bandicoot rat or Indian "Mole-Rat" (Biswas et al. 2011). These several species have different relations to human populations, the rats being "peri-domestic," while the gerbils are wild. But all of them shared two species of flea, showing how transmission could occur across rodent populations and move from rural enzootic foci into proximity with humans.

Similar field surveillance of plague transmission in rodent communities has been going on for over a century and has shown repeatedly that multiple species are involved (e.g., Davis 1953). Especially important is research showing the importance of looking beyond rodents. Camels have been known to be plague carriers for over a century, but they seem to infect humans for the most part when sick animals are slaughtered and eaten, thus producing gastrointestinal plague. Such a case has been doc-

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and their implications for transmission of disease among humans, without the intervention of a flea vector. (See Veracx et al. 2012 for the connections between human head and body lice.)

5 Anisimov, Lindler, and Pier (2004) note that over two hundred species of wild rodents and over eighty different flea species are known to be involved in plague transmission.

umented as recently as 2009 (Federov 1960; Christie, Chen, and Elberg 1980; cf. Ziegler 2014, in this issue). How commonly camels were involved in plague transmission in the past has yet to be explored. But we can see the possible scenarios. Small rodents called jirds (in the genus *Meriones*) are known to live around the tents of nomads and have been documented as being infected but plague-resistant, most recently in Algeria. This raises the specter that even isolated nomadic herders or traders might have helped transmit plague across wide expanses not otherwise thought hospitable to *Y. pestis* transmission (Bitam et al. 2010; see also Varlık 2014, in this issue). Various species of *Meriones* are found from Northern Africa to Mongolia, in a variety of different climatic environments. Jirds can be commensal as well as sylvan, making possible plague transmission from enzootic foci to human communities. Since camels were domesticated as early as the fourteenth or fifteenth century BCE and served as the major beast of burden for overland trade in the Middle East from about the second century of the Common Era (Bulliet 1975), they should surely be factored into our calculus of how *Y. pestis* moved as far as it did.

I am not proposing a grand new monocausal thesis here. Few of these species, by themselves, make sense as plague-transmitters across the wide terrains of Afro-Eurasia,[6] and the climate science needed to help understand faunal dispersals (independent of human herding or trade) is only beginning to emerge. But it is important to stress that historical research, likely combining the efforts of historians working with documentary sources and bioarcheologists working with biological remains (and climatologists as well), will be necessary to assess whether some of these possibilities are more plausible than others. The phylogenies of commensal animals are now being studied as bioproxies for human histories: that is, tracking the histories of animals that have moved at great distances along with humans can serve to document human movements even when all other traces have vanished (Jones et al. 2013). But as the case of plague makes plain, the movements of such commensals are important in their own right, since these species can serve to recreate fairly homogenous microenvironments in many different parts of the world.[7] Introduce even a limited number of *Yersinia pestis*-bearing fleas into such populations, and you may well have the ingredients for epizootics that spill over into human populations.

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6 A conference on precisely this issue was held in Leipzig in 2010 (Franz, Riha, and Schubert 2010).

7 Anthropogenic replication of microenvironments has likewise been key to the global spread of other vector-borne diseases, like malaria and dengue fever. See Green (forthcoming) for an overview.

Reaching Africa: *Yersinia pestis* in Evolutionary Time and Place

As noted above, one of the key developments in genetics has been the construction of robust phylogenetic trees that postulate the evolutionary relationships among various strains of *Y. pestis*, as documented from aDNA and modern samples. Evolution, of course, is a historical phenomenon par excellence, involving that most essential object of the historian's quest: change over time. Earlier calculations of *Y. pestis*'s history, working from the assumption that there was a biomolecular clock that "ticked" at a regular rate, postulated that *Y. pestis* diverged from the most recent common ancestor it shares with *Yersinia pseudotuberculosis* anywhere from 1,056 years before the present (50% confidence limit) to 20,436 years (95% confidence limit) (Achtman et al. 1999).[8] The complete sequencing of the fourteenth-century genome—which at last put a time-date stamp on one point of the organism's premodern evolutionary tree—allowed a recalibration of that molecular clock. A new history of *Y. pestis* was proposed, which estimated its emergence as having taken place as recently as about 3335 years before the present, with 95% confidence intervals still within the range of recorded human history (4394 BCE to 510 CE) (Cui et al. 2013).

The calculation of time using molecular evidence is still a highly contested area of paleogenetics (Ho and Larson 2006; Larson 2013). The phrase "about 3335 years [ago]" has no calendrical authority, but is rather just a computational cipher. In fact, the notion of a fixed "molecular clock" is now seeming like a red herring: genetic change likely occurs for various reasons and at various rates, and is therefore historically variable.[9] Still, the phylogenetic tree of *Y. pestis* (as it has been refined over the course of the past decade and a half) does offer important chronological infor-

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8 These biomolecular clock rates were initially calibrated in the 1980s on the basis of changes observed under laboratory conditions in *Enterococcus coli* and *Staphylococcus enterica* type *typhimurium*. The lower limit cited here, 1056 years before the present, would, if true, obviously put *Y. pestis* out of the running as the causative agent of the Justinianic Plague. But the authors, assuming that the First Plague Pandemic was caused by *Y. pestis*, simply adjusted the clock back: "Justinian's plague was 1,500 years ago, and, therefore, *Y. pestis* is at least 1,500 years old" (Achtman et al. 1999: 14047). A revised calculation was offered in Achtman et al. 2004, and again in Cui et al. 2013, which I discuss below.

9 See, for example, Wagner et al. 2014, which offers a second fixed point in time for *Y. pestis*'s historical genome and suggests that "[w]hatever the cause of this rate variation, these data suggest that previous molecular-clock-derived estimates of the timescale of *Y. pestis* evolution, including the date of its divergence from *Y. pseudotuberculosis*, might be erroneous."

mation in that it suggests a *sequence* of chronological change: "this" happened before "that." And once we tie that relative chronological information to fixed geographic space, the real value of the new work in genetics for the historian becomes apparent: the fact that approximate place in time can be connected to specific place in space.

The question of plague's geographic origins has long troubled historians. Oddly, the lines of argument have gone in different directions: for the Justinianic Plague, the standard argument had been to assume an African origin;[10] for the Black Death, its genesis has been placed more or less vaguely in "the East." Given a confused historical record and the current distribution of *Y. pestis* across most of Asia and Africa (World Health Organization 2008, Neerinckx et al. 2008), it is little wonder the question has long been unresolved. In 1976, McNeill, who placed the origins of *Y. pestis* "at some perhaps geologically ancient time," chose to remain on the fence: "There appears to be no basis for deciding which of these two natural reservoirs [Central Africa or northeastern India, i.e., the Himalayas] is the oldest" (McNeill 1976: 139).[11] For McNeill, the obscure geographic origins of *Y. pestis* could be shrugged off so long as it was seen to exist "time out of mind." Even the emerging evolutionary narrative in the 1990s left the question ambiguous because a widely used classification system, dating from the 1950s, collapsed African and Asian strains into a single subspecies grouping, the biovar "Antiqua" (Giuyoule et al. 1994; Achtman et al. 1999). Extensive work has now been done on the genetic diversity of *Yersinia pestis* as it is currently found throughout the world, allowing the pathogen's evolutionary history to come more clearly into view. That work does not prove in any absolute sense where *Y. pestis* originated as an organism. But as a hypothesis, it accords with the emerging evolutionary and historical understanding of the organism.

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10 Horden 2005; Sarris 2007: 120–23; and Sallares 2007. McCormick (2007: 303–04), relying on Actman et al. 1999, defers judgment on the question. No written evidence has yet been gathered that can push plague's presence in sub-Saharan Africa back definitively before 1877; see Neerinckx et al. 2010.

11 For reasons that I will address in the last section of this essay, I consider all of this prior work "retrospective diagnosis" in the older sense, based exclusively on textual sources whose ability to properly document a specific microbiological pathogen is not robust. It is for this reason, too, that I will not engage here with the many maps that have been published in history textbooks and elsewhere, "plotting the course" of plague's spread with arrows and routes that have no foundation in documentary evidence, let alone material remains. On the many pitfalls in mapping as it relates to plague's histories, see Mengel 2011.

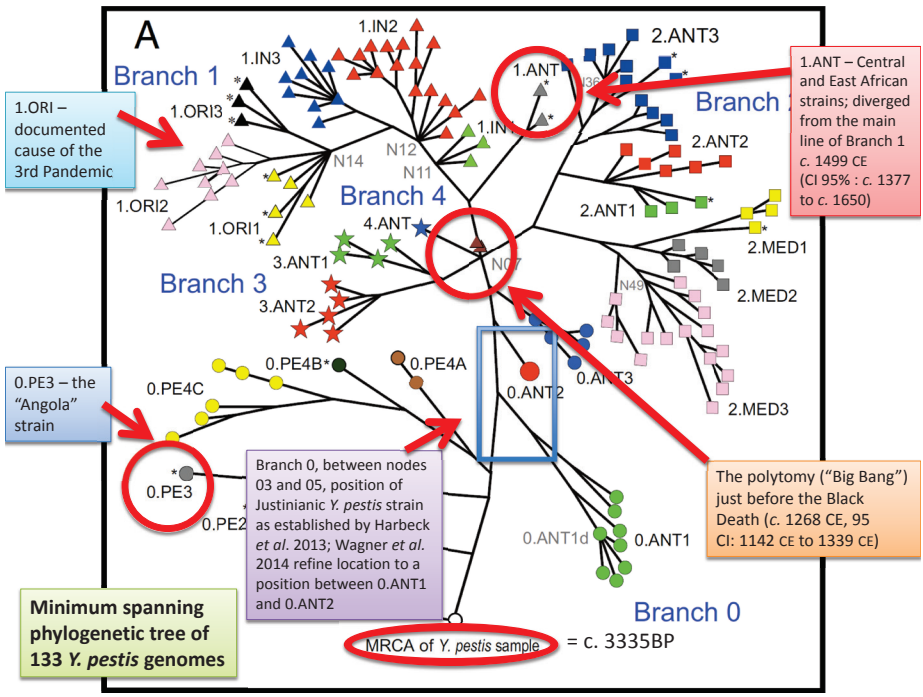

Plate 1. Phylogenetic Tree of *Yersinia Pestis*. *Adapted with permission from Cui et al. 2013*.

A basic principle of evolutionary theory is that the greatest subspecies diversification will be found in the area where the species has lived the longest. This is no constant, of course, since changed environmental conditions can drive any life form out of its original habitat. And strains go extinct, erasing from view certain lines of development. But as a rule of thumb, the association of geography with subspecies diversification generates valuable hypotheses. For *Y. pestis*, the current hypothesis—first proposed on genetics grounds in 2004 and elaborated further since then—is that the organism took its origin in the Tibetan-Qinghai Plateau, now a part of modern China.[12] If this is true, then all narratives of plague's history must be connected to that place of origin. And if it is true, it resolves definitively the Africa vs. Asia dilemma of plague's origin. This is one of the biggest game-changers in historical studies of plague since

12 The Chinese plague researcher Wu Lien-teh seems to have been the first to suggest, on epidemiological grounds, that *Yersinia pestis* had its origin within the boundaries of what we now call China (Wu 1924). The hypothesis was first advanced on genetic grounds by Achtman et al. 2004; see most recently Li et al. 2009, Morelli et al. 2010, Cui et al. 2013, and Yan et al. 2014.

the identification of the rat-flea nexus at the turn of the twentieth century. But the emerging evolutionary narrative has a second result, and that is to suggest that we should broaden our narratives of the Second Pandemic to include sub-Saharan African and, by implication, the Indian Ocean basin.

The phylogenetic tree shown in **Plate 1** comes from Cui and colleagues' 2013 study of historical variations in the mutation rate of *Y. pestis* and summarizes the entire history of the organism as it is understood from genetics. The historical significance of its five major branches (or lineages) and twenty-five subbranches (or phylogroups) is as follows.[13]

- Branch 0 (the main stem of tree) contains almost all pestoides isolates, which, with the exception of the oldest strain, 0.PE7, are found primarily in voles (groups 0.PE1, 0.PE2, 0.PE3, 0.PE4, 0.PE7).[14] It also includes the earlier strains of the ANT group (0.ANT1-3).
- Harbeck and colleagues (2013), working from human remains found in Bavaria, established that the strain that caused the Justinianic Plague seemed to lie on branch 0 between nodes 03 and 05. Wagner and colleagues (2014) have refined that conclusion to put the Justinianic strain specifically between nodes 04 and 05 (that is, between the current extant strains 0.ANT1 and 0.ANT2).
- Node 07 is the polytomy event ('Big Bang') that leads to branches 1-4. Cui et al. (2013) calculate a date for the polytomy at *c*. 1268 (95% CI: 1142, 1339).
- Branch 1 contains African Antiqua (1.ANT) and all of the Intermediate (1.IN) and Orientalis (1.ORI) strains. Strains on this branch caused both the Black Death and the Third Pandemic. 1.ANT is localized now in Central and East Africa.[15]

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13 The trifold categories of biovars—"Antiqua," "Medievalis," and "Orientalis"—formulated in the 1950s according to the ability of different strains to reduce nitrate and to ferment glycerol, have been shown to be misleading and are now otiose (Achtman et al. 2004); these phenotypical categories have no meaningful relationship with the strains likely to have caused the three plague pandemics. The labels "ANT," "MED," and "ORI" are, nevertheless, still found on laboratory samples, relics of that older classification system.

14 In 2012, Riehm et al. proposed an additional strain, 0.PE8 (MNG 2972) found in Mongolia, and suggested the possibility of yet another strain, 0.PE9 found in Tajikistan, Uzbekistan. The species from which the samples were collected were not reported. Neither strain is shown on the diagram of Cui et al. (2013).

15 Seifert et al. (2013: 5, table 2) refer to one sample of 1.ANT as "NCTC_570 Bombay 267." Assuming this is not simply a labeling error, it would be the only known case of

- Branch 2 contains all of Medievalis (2.MED) and yet another "Antiqua" strain (2.ANT). These strains are currently found throughout much of Eurasia, including India (cf. Kingston et al. 2009) and as far west as Turkey and Libya (Cabanel et al. 2013).
- Branches 3 and 4 (3.ANT and 4.ANT) were first announced in 2013 by Cui and colleagues; to date, these have been found only in China and Mongolia, in marmots and in the genus *Meriones*, which includes many kinds of jirds as well as gerbils.

Three features of this evolutionary tree are significant for plague's standard premodern history: that is, the narrative centered on plague's arrival in the Mediterranean and Western Europe. At the center of the tree, at node 07, is the polytomy ("Big Bang") dated by Cui and colleagues to *c*. 1268 CE, with a 95% confidence interval ranging from 1142 CE to 1339 CE.[16] This period of rapid diversification of *Y. pestis* (possibly caused by the organism moving into new climatic environments and new hosts) immediately preceded the Black Death, whose genome (taken from the London Black Death cemetery) lies near the root of Branch 1 (the maroon-colored triangles). Below the great polytomy, on Branch 0, lies the strain involved in the Justinianic Plague, which seems to fall between nodes 04 and 05 (Harbeck et al. 2013; Wagner et al. 2014).

The phylogenetic tree produced by Cui and colleagues was constructed from information drawn from the complete genomes of 133 samples of *Y. pestis*. Two of these (the two maroon triangles near the base of Branch 1) are medieval samples from the London Black Death cemetery. All the others (131) are samples collected between the late nineteenth and early twenty-first century; and all of these, save seventeen, come from areas now within the political boundaries of China and Mongolia. The outliers come from Russia (2), other areas within the former Soviet Union including Georgia (3), Africa (3), India (1), the United States of America (2), Myanmar (1), Madagascar (3), Nepal (1) and Iran/Kurdistan (1). Here I wish to focus on the three samples from Africa.

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the African strain 1.ANT being found outside of Africa. Amann (2007: 22) gives the provenance of NCTC_570 Bombay 267 as "G. Liston in 1920 / fatal bubonic plague, Bombay." W. Glen Liston (1872–1950) was a leading plague researcher in Bombay (Mumbai) and helped establish the role of the flea in transmitting plague from rats to humans. I have not been able to determine anything further about the provenance of this sample.

16 See Hymes (2014, in this issue) for the significance of this date.

Little genetics work has been done on sub-Saharan Africa on a scale comparable to what has been done in China and Mongolia, a lacuna in research that makes the following speculations tentative in the extreme.[17] Nevertheless, such *hints* as are now available from *Y. pestis* genetics work suggest that a premodern history of plague in sub-Saharan Africa might be worth exploring. The earliest of the three African samples is also the single most problematic part of the phylogenetic tree of *Y. pestis* as it is currently understood. This strain, called "Angola" (0.PE3), comes from a laboratory sample collected in or before 1984.[18] All provenance information has been lost, and it is not known whether it was collected from a human being, a rodent, or an arthropod vector. It is in every sense a conundrum, because no other samples from this region have yet entered into the genomics databases for *Y. pestis*. The "Angola" strain is distinctive in two respects. First, it is the most divergent of all the strains thus far studied. 548 SNPs separate it from the most recent common ancestor (MCRA) that *Yersinia pestis* shares with its closest related species, *Yersinia pseudotuberculosis*. All other strains, save two, of *Y. pestis* that were studied from complete genomes by Cui and colleagues have less than 300 SNPs that separate them from the MRCA (the mean is 248). "Angola" is the ultimate outlier. Second, "Angola" also lies very close to the base of the phylogenetic tree of *Y. pestis*. Just how old it is as a strain is unclear, but it clearly derives from some very ancient form of the organism. Only

17 Work coming out of the Institut Pasteur in Paris in the 1990s (e.g., Guiyoule et al. 1994) drew heavily on samples from Kenya and the (then Belgian) Congo. More recent genetics work, however, has focused mostly on Madagascar, where a radiation of one of the Third Pandemic strains predominates (Morelli et al. 2010). According to the World Health Organization, foci exist in the Democratic Republic of the Congo, Kenya, Lesotho, Libya, Mauritania, Mozambique, Namibia, Senegal, Tanzania, Uganda, and probably Egypt (World Health Organization 1999: 16 and 26–31). I return to the implications of this sampling and research bias at the end of the essay. See also Varlık 2014, in this issue.

18 Dos Santos Grácio and Grácio (2011: 1) claim (without offering citations) that the "first reference to the occurence [*sic*] of plague in Angola was made in 1921. . . . However, others believed plague was already there, and that a sylvatic cycle with a reservoir in wild rodents was already present in Austral Africa before the XV century European arrival." The complete genome of the Angola strain (NC_010159) was sequenced in 2010 and can be found online at <http://www.ncbi.nlm.nih.gov/nuccore/NC_010159.1> [accessed September 10, 2014]. Morelli et al. (2010) first broached the idea that the Angola strain might be an extant remnant of the strain that caused the Justinianic Plague. That notion has since been challenged by Harbeck et al. (2013) on the basis aDNA retrieved from a gravesite in Bavaria.

two phylogroups that have yet been sequenced are more basal, and these come from Eurasia.[19]

So, how long has "Angola" been in sub-Saharan Africa? There is simply no way to tell at this point. But here is where a multidisciplinary perspective would allow us to take the question seriously, even if we cannot yet answer it. We actually have a least one account from a Greek writer in the first century CE, Rufus of Ephesus, reporting on other writers who described a "pestilential fever" characterized by swollen glands in the neck, armpits, groin, and back of the legs. According to Rufus, this condition was reported in Libya, Egypt, and Syria—in other words, around the Mediterranean littoral—at least six centuries before the Justinianic Plague arrived at the port of Pelusium in 541 CE.[20] By itself, Rufus's report is insufficient for a clear determination of the presence of plague in North Africa around the beginning of the Common Era. And connecting the Mediterranean to sub-Saharan Africa is even more problematic for this period of time. But we can see why the historiographic tradition linking plague with Africa in ancient times cannot be dismissed out of hand.

The real surprise that comes from genetics, however, is the possibility that it might help us to reconstruct a medieval or early modern history of plague in sub-Saharan Africa. The other African phylogroup represented on Plate 1 is 1.ANT (gray triangles, top center), which breaks off from the main stem of Branch 1 just above (i.e., just after) the 1348 London genome. 1.ANT, also called "Africa Antiqua," is the group of strains of *Y. pestis* found now in East and Central Africa, from Kenya to Uganda and into the Democratic Republic of Congo. In 2010, Morelli and colleagues proposed an age for the origin of this branch somewhere between 628–6914 years ago. In 2013, Cui and colleagues recalibrated this biological clock, suggesting that the African clades had branched off from the main Black Death clade at a later time: *c*. 1499, with a 95% confidence interval of *c*. 1377 to *c*. 1650.

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19 These are 0.PE7, which was sampled for the study of Cui et al. (2013) from the Mongolian five-toed jerboa (*Allactaga sibirica*) and a human patient, both in the Chinese province of Qinghai; and 0.PE2, which was sampled from voles in an area of the Former Soviet Union and Georgia.

20 Rufus's original work no longer survives; rather, the excerpt describing buboes is reported by the fourth-century CE writer Oribasius, in his *Collectionum*. See Sallares (2007: 251) for the Greek text and, for an English translation of the passage, Simpson (1905: 4); see also Simpson (1905: 281) for an image of a popliteal (behind the knee) bubo. My thanks to Heinrich von Staden for confirming the reading of the Greek and advising me on the identification of the authorities Rufus cites, none of whom (*pace* Sallares) can be dated before the first century BCE.

Again, as stressed above, the chronology established by genetics is more valuable in suggesting *relative* position in time than absolute position. It establishes a historical sequence: X happened before Y. 1.ANT is, in terms of its place on the *Yersinia pestis* phylogenetic tree, one of the most closely related "descendants" of the strain that killed the individuals in London whose remains were studied in reconstructing the first complete sample of the Black Death genome. Or, one could say that it is a "distant cousin" of the London strain, rather than a direct descendant. This distinction is important, since the phylogenetic tree does not show us all the dead ends, the extinct sub-branches that may have moved into new geographic terrains or new hosts but then failed to establish mechanisms of long-term survival. The Black Death genome from London is an incredibly lucky find: that rare one-in-a-zillion fossil that captures a strain of the organism as it existed seven hundred years ago, before it hit a "dead end" in the human beings it killed in fourteenth-century London. The modern specimens of 1.ANT, in contrast, have had seven hundred more years to evolve. And like the "Angola" strain, they show that 1.ANT has undergone considerable unique evolution: on a differently calibrated tree, one where physical distance more closely approximates genetic difference, 1.ANT is the furthest spanning subbranch of lineage 1, representing up to 100 SNPs from the 07 polytomy and 300–305 SNPs from the most recent common ancestor of *Y. pestis* (Cui et al. 2013: 579 and supplementary data 8, fig. S3, A). Moreover, a study of this East and Central African strain done back in 1994 found a considerable level of local genetic diversity, suggesting that it had been established in the area for a considerable period of time (Guiyoule et al. 1994).

According to current biological theory, when populations diverge from a common ancestor, genetic differences between them can arise for several reasons. One of those reasons may be adaptations necessary for survival in a new host species. There are no marmots (one of the main host genera for *Y. pestis* in Eastern Eurasia) in Africa, nor, aside from commensal rodents, does there seem to be any major overlap in the known host species of *Y. pestis* on the Eurasia and African continents. In other words, adaptation to new host species may well account for the stark divergence of 1.ANT from other strains on Branch 1, none of which are currently known in Africa save for the modern 1.ORI strains.

So, to summarize, genetics currently tells us three things about 1.ANT. First, it does not derive directly from the "Angola" strain. Hence, whenever "Angola" did arrive in Africa, it did not give rise to 1.ANT. The history of those two strains, and specifically the history of their arrival in Africa, can thus be separated. Second, 1.ANT is older than the strains dis-

seminated worldwide (including to parts of sub-Saharan Africa) during the Third (modern) Pandemic, the 1.ORI strains. So the history of 1.ANT can be separated from the narratives of the Third Pandemic.[21] And third, 1.ANT, the strain of *Y. pestis* associated (so far as we currently know) only with Central and Eastern Africa, is most closely related to the Black Death strain now documented from the London Black Death cemetery.

So when did 1.ANT arrive on the African continent? And by what route? As will be discussed in the next section, the arrival of plague into the Mediterranean basin (and so into North Africa) in the ancient Greco-Roman period was almost certainly via the Red Sea, where trade would have connected the Mediterranean to the Indian Ocean. Whether plague moved further south into the African continent, beyond the Horn of Africa, during the ancient Greco-Roman period is as yet unclear. Clearly, trade was occurring across the Sahara and, up to a point, in the Nile Valley, but we do not yet have strong evidence either for the presence of commensal rodents or for developed urban communities that would have facilitated plague's spread south of the Sahara before the seventh century CE.

By the time of the later medieval plague, however, the situation in sub-Saharan Africa was different. Considerable trade occurred across the Sahara in exchanges of gold, slaves, salt, and probably also ivory (McDougall 1990; Guérin 2013). Mansa Musa's famous pilgrimage from Mali to Mecca in 1324 remains symbolic of those medieval trade networks, a trip whose timing should not go unnoted. For just a few decades later, we find abandonment of sites in sub-Saharan Africa that had previously been thriving metropolitan centers. The sudden late medieval abandonment of earthwork settlements at Jenne-Jeno and Akrokrowa, in what is present-day Ghana, has proved a puzzle to archeologists. In 1998, McIntosh mused on the possibility that plague may have been involved in Jenne-Jeno's abandonment (McIntosh 1998: 247–50), while Chouin and DeCorse (2010) were willing to speculate openly about it as the cause of widespread abandonment:

> Looking at world history during this period, it seems that only one event can possibly explain such a large-scale phenomenon: the occurrence of

21 The historical works of Myron Echenberg on plague in Senegal and South Africa (Echenberg 2001 and 2007) do not draw on the genetics works that was only then emerging. Nevertheless, subsequent genetic analysis suggests that the strains of plague that hit northern, northwestern, and southern Africa in the twentieth century were of the 1.ORI phylogroups, and hence distinctive of the Third Pandemic; see Bitam et al. 2010; Morelli et al. 2010. On the recent discovery of a MED strain (found on Branch 2 of the phylogenetic tree in Cui et al. 2013) in Algeria, see Cabanel et al. (2013).

> the Black Death or Great Plague. . . . In affecting societies living in forest areas of southern Ghana, the Plague would have had exactly the same effect as in other parts of the world: it would have destroyed a large portion of the population living in densely populated settlements, resulting in their abandonment—a well-documented phenomenon in many other contexts. (Chouin and DeCorse 2010: 143)

But in placing plague in sub-Saharan Africa, Chouin and DeCorse were overinterpreting the superficial and unsubstantiated claims of a non-academic book published nearly thirty years earlier (Cartwright and Biddiss 1972).[22] To date, there is neither bioarcheological research nor aDNA to tell us what happened to these societies which, it seems, left no written records. Hence, the question of plague's possible penetration across the Sahara to West Central Africa must remain for the moment an open question.

The situation for East Africa is different. Here, the likely point of entrance for the strain of *Yersinia pestis* that now dominates in Central and East Africa, 1.ANT, was the Indian Ocean coast, where from the seventh century on there is increasing evidence of a "a deeply networked trade and contact situation" (Boivin et al. 2013: 1; cf. Horton 2004). With two exceptions, Indian Ocean transit of *Y. pestis* to East Africa below the Horn has not previously been suggested for plague prior to the Third (modern) Pandemic.[23] Nevertheless, we have cause to raise the question of the timing of plague's arrival. For in Eastern Africa, we have a phenomenon similar to that in West Africa, at virtually the same time: at least four important trading centers suffered major decline. On the coast, Shanga (on the Lamu Archipelago off the coast of modern-day Kenya) and Tumbatu

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22 In fact, Cartwright and Biddiss never said anything about sub-Saharan Africa per se, referring only generically to "Africa" (1972: 32 and 51). Their narrative typically just summarizes the impact of the Black Death on Europe.

23 Horton (1996/2012) notes the coincidental timing of the Black Death and the decline of these towns, but implies that the latter is due to the general collapse of the international economy at that time. Horton and Middleton (2000: 82) offer a passing suggestion that plague may have indeed reached East Africa. They cite Dols (1977) in support of their claim that plague spread "throughout the Middle East and the Indian Ocean world," but Dols in fact claimed that transmission through the Indian Ocean was unlikely (Dols 1977: 43–44). The other claim of Indian Ocean transport was made by the microbiologist Mark Achtman, who proposed in 2010 that *Y. pestis* might have been brought to East Africa by the Chinese explorer Zheng He (1371–1433) (Morelli et al. 2010). Achtman has since retracted that suggestion (Cui et al. 2013), not because of its historical or epidemiological improbability, but rather because he recalibrated his biological clock, pushing the divergence of 1.ANT from the main branch to a later era and thus making Zheng He's role less likely.

(on Tumbatu Island) were abandoned, while Kilwa on the island of Kilwa Kisiwani (modern-day Tanzania) became derelict. Inland, Great Zimbabwe (near present day Masvingo, Zimbabwe), known for its monumental mortarless structures, was abandoned. The dating of Great Zimbabwe's abandonment has been intensely debated; carbon-14 dating performed in the early 1990s showed that Great Zimbabwe's growth had stopped by the mid-fourteenth century, and the site was abandoned definitively by the sixteenth, at the latest (Huffman and Vogel 1991; Pikirayi 2001: 150–51). While there was not complete devastation of the East African coast in this period (e.g., Wynne-Jones 2013), the pattern of coastal areas and an upland region (Great Zimbabwe is on a plateau, over a thousand meters high) being simultaneously affected bears the distinctive stamp of plague (Carmichael 2014, in this issue). Moreover, the conditions for plague's spread were there: intense human trade in various commodities across the Indian Ocean and the arrival of Asian commensal rodents in East Africa.

Reaching Tibet: A New Geography of Plague

> And the coral *which is brought from our homeland* is sold more in this land than any other kind.[24]

Marco Polo (d. 1324) was a latecomer to the vast trade networks that brought Mediterranean coral all the way to Kashmir (where he made this observation) and to Tibet, where he noted that coral brought "great delight" there, too.[25] Polo's *Description of the World,* read from the perspective of what we know now about the global spread of pathogens, opens a window onto the material connections of the premodern world. Coral was one of the few precious yet easily transportable substances that the Mediterranean world could trade eastward in exchange for so many kinds of materials and goods coming from East and South Asia (Sibon 2012). Tracing its trade, as well as that of other substances like Tibetan musk, Indian pepper, or East African ivory, helps us reconstruct human exchanges otherwise lost to his-

24 Chapter 48, on Kashmir ("Thesimur"): "Et le coral qui se porte de nos contrees se vent moult en ceste contree plus que en autre" (Polo [and Rustichello of Pisa 1298–99]/2003: 7, emphasis added). My thanks to Barbie Campbell Cole for bringing this statement to my attention, and to Markus Cruse for providing me with the citation.

25 Chapter 115, on Tibet ("Tebet"): "En ceste prouvince s'espant le corail et est moult chier, car il le meitent au col de leurs fames et de leur ydres par moult grant joie" (Polo [and Rustichello of Pisa 1298–99]/2003: 71).

tory. By themselves, these luxury goods were probably innocuous in terms of disease transmission.[26] But add in the furs that may have carried fleas and their excrement far beyond where their animal hosts would have taken them while living (Ducène 2005); the beasts of burden laden with these goods, with their own fleas and ticks; the shipholds and storage containers full of locally traded grain, and the rodents they attracted; and a medieval taste for luxury textiles that drove international trade (but also could transport fleas and their excrement), and we have a web of connections that make up Le Roy Ladurie's "microbial unification of the world" (Le Roy Ladurie 1973). We cannot, of course, be casual in our use of globalizing frameworks. Specifics in time and space matter hugely. Genetics has some contributions to make, archeology and history even more. But together, they suggest that a whole world region should be added to our narratives of the Black Death, a region that would connect the Tibetan Plateau and Qinghai to sub-Saharan Africa: the Indian Ocean basin.

It is nothing new to connect the Black Death narrative with trade, of course; it has long been suggested that plague moved to Western Eurasia and thence the Mediterranean and North Africa "via the Silk Road." That is simply a statement of its trajectory, however, rather than an actual documented mode of transmission, since it is likely that (whether carried by rodents or humans or other species) *Y. pestis* followed the topography of mountain passes, rivers, and valleys that traversed Central Asia and served as the network of trade connections that we aggregate under the rubric "Silk Road" (Beckwith 2009). Overland routes make most sense, of course, as trajectories for plague's arrival in the Black Sea region, whence it spread into the Mediterranean. But an important question needs to be asked: did it go in other directions as well? Arguments have been made by Buell (2012), Sussman (2011), and Anandavalli (2007) that plague (or at least major human outbreaks) cannot be documented in medieval China or India. Hymes's contribution to this issue challenges these claims with respect to the Gansu corridor and areas of China further east. Moreover, as science reminds us, there can be plague without "plague." Even in the absence of human outbreaks, *Y. pestis* can subsist and thrive in mammalian communities normally beneath the notice of humans. A new genetics of plague—especially the fact that the African 1.ANT strain demands explanation—suggests that southern routes of plague in the late medieval period merit further consideration.

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26 Or perhaps one should say "usually innocuous." A twelfth-century Latin account explains that musk (both real and fake) was often sold in the skin of the musk deer (Wölfel 1939: 79–80), so perhaps it posed the same problem as the transmission of furs.

First of all, it is likely that we have underestimated the role of a southern route, via the Indian Ocean, for the First Plague Pandemic itself. Contrary to assumptions made by historians of late antiquity, it would seem that plague manifested first at the Egyptian port city of Pelusium (in the same site as modern-day Tell el-Farama) not because it "came out of Africa" (i.e., Egypt), but because it came via traffic from the Red Sea (Little 2007).[27] The port of Clysma in the Red Sea has recently been proposed as the possible entrance gate of *Yersinia pestis* into the Mediterranean (Tsimias et al. 2009). This argument was based solely on considerations of ancient geography, but the need to plot a route for *Y. pestis* out of the Tibetan Highlands fits both the emerging genetics data and the newer research on trade in the Indian Ocean basin, which can now very easily account for an intensity of travel that (however slowly) might have moved plague out of its Central Asian home into the very different environments of the Indian Ocean and ultimately the Red Sea and the Mediterranean littorals.

Secondly, the field of Indian Ocean studies is one of the areas that has been transformed radically in the decades since the main lines of argument in plague historiography were laid down (Seland 2014; Beaujard 2005 and 2012). We have long known about the *Periplus of the Erythraean Sea*, a Greek report on Indian Ocean trade and navigation, written in the mid-first century CE by an Egyptian merchant. It notes, for example, the import of coral at the Skythian port of Barbarikon/Minnagar, which was at the mouth of the Indus, and the export of "Chinese pelts" (Seland 2010: 59). More recent discoveries include the existence on Socotra Island (off the coast of modern Yemen and Somalia) of a cave with ancient inscriptions. These have been determined to be by sailors who visited the island between the first century BCE and sixth century CE. Texts written in Indian Brahmi script, and South-Arabian, Ethiopian, Greek, Palmyrene, and Bactrian scripts and languages give evidence of the breadth of Indian Ocean trade up to and including the time of the Justinianic Plague (Strauch 2012). For the later Middle Ages, we have equally remarkable letters and other documents of Jewish traders from the Cairo Genizah that show an intense and thriving trade across the Indian Ocean (Goitein and Friedman 2008).

The Socotra and Genizah finds are unusual, however, as is the Greek *Periplus*. For the most part, we have neither identifiable individuals nor

27 The claim that rats carrying plague brought the disease from the Tanzanian coast up to the Red Sea in the sixth century was premature (Horden 2005: 153). Instead, as discussed below, the archeological evidence now suggests the arrival of rats in East Africa somewhat later.

any written records at all. However, as with Marco Polo's coral, tracking unique commodities in their travels allows us to glimpse how intense Indian Ocean traffic was. Take pepper and musk, for example. Black pepper was, throughout antiquity and the Middle Ages, produced primarily on the Malabar Coast of what is now southwest India. Pepper's history has long been told in terms of trade with the West, but it is apparent now that China was one of the main markets for pepper in the Middle Ages. In other words, pepper was going both directions from the Malabar Coast, connecting China with all points in the Indian Ocean (Prange 2011). Similar motives in the trade of musk connected Tibet with the Indian Ocean world and parts beyond. The Egyptian encyclopedist and historian al-Nuwayrī (677/1279–732/1332), for example, explained that Tibetan musk (deemed far superior to that that came from China or elsewhere) was brought down to the Arabian Sea via the Indus Valley, whence it was shipped to various other ports in the western Indian Ocean (Akasoy and Yoeli-Tlalim 2007: 221). In fact, a whole array of archeological evidence—pottery, beads, tableware, and so forth—is stark witness to the intensity and extent of trade, both short- and long-distance, throughout much of the Indian Ocean basin.

Where there is extensive human traffic across a geographical expanse, it stands to reason that we should also look for commensals that traveled with these traders: a scenario perfect for the establishment of *Y. pestis* in new terrain (and amply documented from modern studies of *Y. pestis*'s very rapid establishment in North and South America and Madagascar during the twentieth century). And in East Africa, such commensals are readily found. The Asian house shrew, *Suncus murinus*; the black rat (ship rat), *Rattus rattus*; and the house mouse, *Mus musculus*, are all documented as imports from Asia (Boivin et al. 2013). The bones of *Rattus rattus*, the black rat, have been found in archeological remains from Unguja Ukuu, Tumbatu (one of the abandoned sites mentioned above), and Shanga along the Swahili Coast of East Africa. *Rattus rattus* may have arrived there as early as the seventh century and seems to have been common in urban contexts in the fourteenth.[28]

Hence, we have two parts of the "elephant" of plague here: the mechanisms of trade across the Indian Ocean basin, and the presence of a "rodent infrastructure" in East Africa to support a vector-borne disease when and if it arrived. We currently have, however, no paleogenetic work from this part of Africa; and indeed, very little genetics work has been done at all on

28 Nina Mudida and Mark Horton, personal communication (February 10, 2013).

the modern *Y. pestis* organism as it currently exists throughout much of sub-Saharan Africa, and where it still causes the second largest number of human cases per year. Indeed, we do not even have clear evidence of a trail of human plague from the Tibetan highlands down to the Indian Ocean ports in the fourteenth or fifteenth centuries, a point used by Anandavalli (2007) and Sussman (2011), in their respective studies, to argue against the presence of plague in medieval India. Yet we should be reminded of the mantra of bioarcheologists: absence of evidence is not evidence of absence. In a 2006 report of the 1994 plague outbreak in Surat, plague is given a history in India that goes back three thousand years.

> The first known outbreak of plague occurred from 1500–600 BC as recorded in *Bhagvata Purana*. The plague was seen again in 1031 AD when the disease reached India from Central Asia following the invasion of Sultan Mahmoud. In 1403 AD, Sultan Ahmed's Army was supposed to have been destroyed by a plague epidemic in Malwa. (Dutt, Akhtar, and McVeigh 2006: 757–58)

No supporting documentation was offered. But other, less extreme claims of the antiquity of plague in India, in some cases pushing it back to the eleventh century, occur in plague literature of the nineteenth and twentieth centuries (e.g., Simpson 1905: 40).[29] For India, then, we are at the same stage of feeble retrospective diagnosis from a handful of opaque written sources as we were when studying the history of plague in Europe prior to 1998. The first "clinical" description that meets Anandavalli's criteria for plague is a 1689 report by a historian, Khafi Khan, writing in Arabic, who recounts how "the plague (*taun*) and pestilence (*waba*), which had been ravaging Dakhin (South India) for several years, had spread to Bijapur. He describes the visible marks of the plague: 'swellings as big as a grape or banana under the arms, behind the ears, and in the groin'" (Anandavalli 2007: 25).[30] For Sussman, the "first" clinical report is that of the Mughal emperor Jahangir (d. 1627), who reported in 1619 that

> At this time, again, it appeared from the reports of the loyal that the disease of the plague was prevalent in Agra, so that daily about 100 people,

29 The possibility of a hitherto unrecognized pandemic in the eleventh/twelfth century merits more examination. There is a story of "epidemics (*wabā'*) and the plague (*ṭā'ūn*)" in early eleventh-century Tunisia "which carried off the greater part of the population" (Talbi 1981: 221). Dols (1977: 33) reports an outbreak in the Hijaz, Yemen, and Egypt in the twelfth century.

30 For the latest assessment of the semantic weight carried by these Arabic terms, see Stearns 2011.

> more or less, were dying of it. Under the armpits, or in the groin, or below the throat, buboes formed, and they died. This is the third year that it has raged in the cold weather, and disappeared in the commencement of the hot season. (Jahangir, as quoted by Sussman 2011: 336)

Is it possible that it was only in the seventeenth century that *Yersinia pestis* first came down from the Tibetan highlands into India?[31] Sometimes absence of evidence really does signal absence of the phenomenon. But India's proximity to the Tibetan highlands, the well-documented networks of trade that connected much of south and southeast Asia throughout the ancient and medieval periods, and, now, the evidence of deep genetic links between the strains of *Y. pestis* found in East and Central Africa and those that came out of Central Eurasia in the fourteenth century: all of these factors raise new questions about India's history with plague.

Anandavalli and Sussman are right to be cautious of reading too much into the ambiguous evidence of the few written sources they had at their disposal. Plague is hardly the only disease that presents in epidemic form, and careful interpretation of such sources always demands the expertise of scholars skilled in the nuances of the original languages. But it has been the objective of this essay to argue that historical narratives can be crafted from other sources, too. Transmission of plague through India (whether via the Indus Valley or the Ganges) begins to look plausible if we take seriously the emerging narrative derived from genetics, and posit an arrival of plague in late medieval East Africa.[32] Indeed, the fourteenth and fifteenth centuries were periods of intense upheaval in many areas of Asia, and it may be worthwhile to consider the possibility that a major social disruptor such as plague contributed to that upheaval (Lieberman 2011; Hymes 2014, in this issue).

In the field of climate and environmental history, a search for consilience among different kinds of historical evidence is openly embraced

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31 Ansari (1994, also citing from Jahangir) says that the emperor first reported the disease not in 1619 but in 1615, noting that "the king wrote that it was the first time that the disease had occurred in India." Catanach (2001: 141) similarly identifies the seventeenth century as the time of the first outbreak of plague in India, when he notes an outbreak in 1631 among handloom weavers in Gujarat.

32 Granted, at this point we cannot rule out another possible narrative, unlikely though it may be given the distances involved and the fact that shipping of trade goods was not a factor: that plague reached East Africa via the Portuguese, who arrived in 1498. On the experience of plague in Portugal, see da Costa Roque 1979. The chronicles of Fernão Lopes, a fifteenth-century chronicler, are due to be published soon in English translation. My thanks to Iona McCleery for this information.

(McCormick 2011). A multidisciplinary approach to disease history, in contrast, might be more immediately served by focusing not just on points where the narratives of different disciplinary approaches converge (as, for example, in the case of the genetics, historical, and bioarcheological work on the London Black Death cemetery),[33] but also on areas where they seem to be in utter disagreement. As Altman and his colleagues have warned us, we should beware of "cherry picking those milestones in human history that are [already] best recorded." A study of *Y. pestis* strains in just three areas of India in 2009 showed three very different lineages (Kingston 2009), making it likely that plague has been imported there more than once. Much more research on the genetics of the modern African and Indian strains of plague, in addition to much more combing of historical archives and sampling of archeological evidence to look for extinct *Y. pestis* strains, will be necessary to confirm whether the suggestions I have made here about a late medieval impact of plague in India and sub-Saharan Africa are plausible. But we would have never seen this as a possible line of research had there not been a convergence of many different disciplines on questions of plague's histories in recent years.

Reclaiming Retrospective Diagnosis: Sources, Methods, and Goals for a Global History of Health

> If we want to understand what health and illness meant for past sufferers, we have to accept their labels, not impose ours. (McCleery 2013: 90)
>
> What was invisible to the [peoples of the past] need not remain invisible for us as well. (Stathakopoulos 2011: 95)

These two statements about what is called "retrospective diagnosis" capture the historian's dilemma: is our task to reconstruct the world as historical participants perceived it (what anthropologists would call an *emic* approach) and reject modern (biomedical) understandings of disease? Or, in a positivist mode, is our task to use the methods and categories of modern science to find out what "really" happened, as judged from an external frame of reference, that of modern science (an *etic* approach)? This question haunts the historian working with cultural remains in a way it would not haunt the microbiologist reconstructing aDNA fragments. For the historian, both of these perspectives are "real," since human experience and

33 See DeWitte (2014, in this issue) for a review of research on the London Black Death cemetery at East Smithfield.

the motives for human actions are major objects of our quest.[34] But among historians, too, there are different motives and different objectives.

"Plague," as Andrew Cunningham has most incisively pointed out, is a construct of modern biomedicine, built on a foundation of laboratory science, epidemiological studies, entomology, and zoology which together have contributed to our understanding that "plague" is a disease caused by an infectious microorganism (*Yersinia pestis*), transmitted by flea bites or other means, presenting certain characteristic clinical signs and affecting the human body through physiological processes known through countless clinical observations and laboratory studies of afflicted humans and animals both pre- and postmortem. Absent that laboratory, there is no "plague" in this sense (Cunningham 1992; cf. Arrizabalaga 2002 and Cunningham 2002). All we have are texts that describe various kinds of suffering, experiences that even those suffering might not have put into a single category of a namable disease. The "linguistic turn" that has affected most Anglophone historiographical traditions over the past thirty years has reinforced a sense that we can never fully break free of the conceptual categories of our historical texts and reconstruct a "real," unfiltered past.

I do not contest this interpretation; on the contrary, I accept it wholeheartedly in so far as it *applies to histories drawn from human cultural products* like written documents or works of art (cf. Green, Walker-Meikle, and Müller 2014, in this issue). But here is where the challenge, and the possibilities, of a multidisciplinary history face us. Microbiologists have broken through the nineteenth-century barrier of laboratory medicine. Nearly all modern laboratory samples of plague and other pathogens have been collected in just the past 150 years. But aDNA research reaches beyond that chronological limit, reconstructing and identifying old organisms in a way that microbiologists themselves now believe is possible (even if they still argue among themselves about best methods) and that connects plausibly with the narratives of pathogen evolutionary history created by genome-based phylogenetics. "Retrospective diagnosis," in other words, now has a completely new meaning, one based on assessment of a material substrate of the past rather than cultural products alone.[35]

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34 The bioarcheologist, who draws on both cultural and material remains, stands somewhere in between these poles, a point I address below.

35 I have foregrounded here the diagnostic possibilities of molecular microbiology, but in diseases other than plague (most notably, leprosy as caused by *Mycobacterium leprae*, discovered by Armauer Hansen in 1873), the older field of paleopathology also has methods to draw plausible "retrospective diagnoses." See Green 2012 and forthcoming for an overview.

So, to return to the skeptic's "So what?" question about the significance of the new plague science posed at the beginning of this essay. My response is: "Because it gives us something to think with." The broadened narrative of plague I have sketched out in this essay suggests the possible significance of animal species and human populations and areas of the world that have never before been part of Black Death narratives. And the other essays in this special issue push those boundaries of new thinking on the histories of plague even further. In a typical anti-retrospective diagnostic stance, historian Iona McCleery says that "If we want to understand what health and illness meant for past sufferers, we have to accept their labels, not impose ours." But what if those sufferers are populations of the destroyed African civilizations of Zimbabwe or possibly Jenne-Jeno and Akrokrowa? What "labels" have we to interpret when these vanished societies left no written records? As Anna Colet and her colleagues make clear in their study of the communal graves of Tàrrega (2014, in this issue), it is sheer chance that we now have both documentary records and physical evidence for the slaughter of the Jewish community of that town in 1348. For so many victims of the Black Death—both those who died of plague and those who died at others' hands—we have no testimony of their suffering other than their physical remains or other traces. And even those are rare, especially in parts of the world where archaeological traditions are still nascent (Campana et al. 2013).

Reconstructing the history of the Black Death can proceed from many motives, and methodologies will be chosen according to the objectives that various researchers wish to achieve as well as their training and resources. Some possible methods for exploring plague's histories have not even been discussed here, such as the use of historical linguistics, which has already proven a powerful ally to both archaeological and genetics work (e.g., de Luna, Fleisher, and McIntosh 2012). Plague is an "elephant" that demands the efforts of many blind men and women to assess its full, huge, and awful expanse. *Yersinia pestis* has established itself as a worldwide pathogen not because (like, say, measles or tuberculosis) it is continually circulating in human bodies. Rather, it exists today in every continent save Australia and Antarctica because it was carried far from its apparent site of origin in the Tibet-Qinghai Plateau by combinations of human and animal carriers who recreated microenvironments in which *Y. pestis* could thrive.[36] It was then able to establish foci in new areas, moving from com-

36 Plague did reach Australia during the Third Pandemic (if not before), but it did not establish permanent foci thanks to aggressive public health measures. See Curson and McCracken 1989.

mensal rodent populations (transported by humans) into new, rural species where the organism could find permanent hosts.

This essay presents the new face of the Black Death, a medieval pandemic that may have spared Oceania and North and South America only because those areas had not yet been brought tightly into the networks of Afroeurasian trade.[37] A global approach to the disease's history—thinking across time and space and our own methodological perspectives, taking in the full evolutionary history of the pathogen and the full human impact of the disease—will give us a mechanism to explain how plague reached those last corners of the world, what it did when it got there, and how its many stories compare to those of other global diseases.[38] A "global" approach will also facilitate greater understanding of a disease that still affects several thousand people every year and could threaten many more (Ziegler 2014, in this issue). Much of the genetics work described above has been based on modern samples collected in China, and on the efforts of Chinese, Russian, and North American scientists, who for many decades have prioritized plague research because it exists as an enzootic (and even bioterrorist) threat in their own countries. But very little comparable genetics work has yet been done in other areas where plague is enzootic: in India, Southeast Asia, South America, and most parts of Africa. Johanna T. Crane (2011), among others, has noted the effects of such an imbalance in molecular research infrastructure on the study of another global disease, HIV/AIDS. This examination of the Black Death suggests that framing pandemics both widely (in geography) and deeply (in time) may not simply broaden our historical knowledge, but help us reconceive the world we live in today.

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37 Though here, too, tracing commensal rodents shows medieval human movements. See Wilmshurst et al. 2008.

38 As this essay will have made plain, it will be necessary to gauge the hows and whys of plague's global history not in terms of human witnesses alone, but from other kinds of evidence. Moreover, all of the methods and perspectives surveyed here can be applied to other conundra in disease history, for example, the still mysterious case of the cocoliztli epidemic of 1544–50 in Central America (see Warinner et al. 2012). As outlined in Green 2012 and Green forthcoming, a global approach to the history of disease is not simply multidisciplinary but deliberately seeks out parallels and interactions between different diseases.

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Abstract This essay introduces the inaugural issue of *The Medieval Globe*, "Pandemic Disease in the Medieval World: Rethinking the Black Death". It suggests that the history of the pathogen *Yersinia pestis*, as it has now been reconstructed by molecular biology, allows for an expanded definition of the Second Plague Pandemic. Historiography of the Black Death has hitherto focused on a limited number of vector and host species, and on Western Europe and those parts of the Islamicate world touching the Mediterranean littoral. Biological considerations suggest the value of a broadened framework, one that encompasses an enlarged range of host species and draws on new archeological, genetic, and historical researches to look for the presence of plague in the premodern Indian Ocean basin and East Africa, areas where it has previously not been suspected.

Keywords Afroeurasia, Indian Ocean, zoonotics, retrospective diagnosis, Black Death, Justinianic Plague, global history of health.

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