Opinion Page

On Learning How to Teach the Black Death

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The Gifts of the Magi

Throughout my career, I have considered myself a specialist in the history of medieval European medicine. I trained in the History of Science program at Princeton University, taking my doctorate in 1985, and then I did a postdoc at the University of North Carolina at Chapel Hill with the renowned expert on medicine in medieval Spain and France, Michael R. McVaugh. But until 2012, I never taught a course on the Black Death, the plague pandemic that struck the Mediterranean and Europe in the late 1340s, and which is considered the largest single disease mortality event in human history. How was it that I could avoid such a massive elephant in the room of my discipline?

The answer was simple: I had no interest in teaching a topic that seemed bedeviled, on the one hand, by endless, insoluble debates about its cause and, on the other, by a rich but seemingly static body of primary sources that were endlessly trotted out as “set pieces” in the Black Death story: Gabriele de Mussis’s (non-eyewitness) account of Mongols of the Golden Horde throwing disease-ridden bodies over
the walls of Caffa; Boccaccio’s depictions of hysteria at the beginning of the Decameron; caricatures of the helplessness (or downright incompetence) of learned European physicians in the face of this unknown disease. I didn’t do the history of infectious diseases in my own work (my research for many years was on various aspects of the intellectual and social history of European medicine, a field that still commands much of my attention), so it all seemed too much trouble to try to make any pedagogical intervention aside from giving rudimentary summaries of the pandemic in my other lecture courses.

What turned my reluctance about teaching the Black Death around was the slow realization, over the course of several years, that microbiologists’ at-first-tentative attempts to intervene in debates about the Black Death’s cause had finally struck gold. Historians and demographers had certainly been right, starting in the 1970s, to ask the question of what caused this sudden mortality of millions of people in the middle of the 14th century. The problem was that they were digging themselves into a hole. Working in the period after the shift to a basic germ theory of infectious disease—the idea that infectious diseases are caused by microscopic foreign agents that come into our bodies—they shifted historiography of the Black Death to focus on a debate about microbes. But our historical evidence (chronicles, tax records, wills, medical treatises, sermons, etc.) was never going to solve this question for one fundamental reason: nobody prior to the modern period saw microbes. Historians, in other words, were never going to be able to answer the question they had posed.

What has happened in the past two decades could not have been predicted, and so has been all the more extraordinary to watch. Microbiologists took up the gauntlet that the historians threw down, not simply solving the question of what caused the Black Death (spoiler: it was indeed Yersinia pestis, as had been suspected since the 19th century), but also laying the foundations for what is now a minor subdiscipline within the larger field of genomic microbiology: the study of ancient pathogens and the evolution of infectious disease organisms. I was not involved in any of these researches, and only began to hear about them around 2005.

Curious to know whether or not I should incorporate this new work into my teach-
ing, I persuaded a colleague to join me in applying to run a U.S. National Endowment for the Humanities (NEH) Summer Seminar that would allow us to explore this issue (and other new historiographical trends in medieval medical history) along with other teachers in the field. It ended up taking three years of submissions before the NEH was finally persuaded that humanists could engage profitably with the sciences and ask the major “So what?” questions of how disciplines might work in unison (or at least in parallel) to address historical questions of major consequence. During the first seminar, in 2009, I was still skeptical that a scientific approach (beyond paleopathology, which is a different method\(^1\)) had anything meaningful to offer historians. By the 2012 iteration of the Seminar, I was convinced that paleogenetics was a method of real import, and finally dared to launch an undergraduate course on the Black Death. By 2014, in putting together a collection of published essays, I had finally figured out an answer to the “So what?” question.

The answer is that the “new genetics” of plague offers the historian three important gifts, all of which allow us now to begin to do historical epidemiology in a way that mirrors what modern epidemiologists can and must do in tracking disease spread. The first gift—and the one for which the paleogeneticists received international praise—was in decisively determining the pathogen involved in the Black Death. One particular study in 2011 got the most attention, but the process of developing the methods and protocols for aDNA (“ancient DNA”) work had developed over a number of years, and involved the work of a number of labs. *Yersinia pestis* (referred to in earlier science and historiography as *Pasteurella pestis*) was confirmed as the causative organism.

The second gift was that, in successfully sequencing the bacterium’s whole genome from samples taken from a well-documented Black Death burial site in London

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\(^1\)Paleopathology, which largely relies on ocular examination of lesions in the skeleton and teeth of human remains, had established, at least from the 1950s, that there were regular criteria that could be used to determine the presence of leprosy infections, one of the other main infectious diseases of medieval Eurasia. The Seminar focused on that work as well. Since 2013, paleogenetics has also succeeded in reconstructing the genome of historic samples of *Mycobacterium leprae*, the causative organism of one form of leprosy. See Benjak *et al.* 2018 and Honap *et al.* 2018 for the most recent work on *M. leprae*’s evolutionary history.
(this was the signal achievement of the 2011 study), researchers were able to say with confidence how—and importantly, how little—the Black Death genome differed from *Y. pestis* as it is documented in the world today. Over the course of the last 700 years, *Y. pestis* hasn’t changed that much. Only a few dozen single nucleotide polymorphisms (snps) separate the 14th-century organism from strains that persist in the world today. This is significant for historians because it was no longer permissible to hypothesize that the huge mortality of the 14th century was caused because the strain involved was significantly more virulent than strains that caused the Third (modern) Pandemic, or that have been sequenced in modern labs today. Causes for the almost inconceivable medieval mortality would need to be sought elsewhere. This finding also meant that modern laboratory and field studies of *Y. pestis* could be used analogically to investigate historical aspects of the disease for which we were unlikely to find written evidence, e.g., the role of specific flea vectors or mammalian hosts or ambient conditions that might affect plague outbreaks. Since plague is considered a Class A pathogen in terms of its bioterrorism potential (the classification refers to pathogens that can be easily transmitted and have the potential to cause high mortality), *Y. pestis* has and continues to elicit a good deal of laboratory research. In other words, this is a disease we can actually study in great detail.

The third gift is a result of what made the adna work possible, that is, extensive study of the genomes of modern *Y. pestis* strains, which have allowed the organism’s evolutionary history to be revealed. Every new *Y. pestis* genome that is reconstructed from historical remains can be fitted into a larger narrative of the organism’s history that has been constructed from modern samples, thus increasing the robustness of the narrative and fine-tuning our understanding of the organism’s (and hence, the disease’s) history. For example, in 2016, a genome was sequenced from remains buried within one of the basilicas of the Spanish port town of Barcelona. Although the carbon-dating of the remains produced only a rough chronological estimate of “1300-1420,” we know from documentary accounts that plague arrived in Barcelona by May of 1348. Hence, it is not in the least surprising to find that the genome from Barcelona matches, down to the last distinctive snp, the genome sequenced from the Black Death cemetery in London, a burial ground created in
late 1348 or very early 1349, and closed in 1350 when the epidemic had passed. Not every new genome sequenced fits the tidy narratives that geneticists would like to propose, but collectively each one tells us part of a unified story of plague, encompassing plague everywhere from Spain to China to Arizona (where I live), at every point from the Bronze Age (whence we now have our earliest complete sequences) up to the outbreak of plague in Madagascar in 2017.

As a result of these three “gifts” that molecular microbiologists have given historians, there are, I would argue, four new “truths” about the Black Death that should now be taught as basic elements of our narratives, whether in middle school when teaching about the larger trajectories of pre-modern history, in college survey classes that cover pre-modern Afro-Eurasia or global history, or in specialized courses either about the history of plague or the history of infectious diseases generally. I lay these new truths out below, followed by some observations about where the next “disruptions” of our common understanding are likely to take place. I conclude with some brief suggestions about how to organize teaching notes on the Black Death for those who have to cover it quickly in the context of other survey courses.

**Truth #1: Genetics Has Given Us a Unified Evolutionary History of Plague Throughout the World**

As the above account will have made clear, the “new genetics” is what has made teaching the Black Death possible for me. But, more than that, it has made a whole new mode of thinking about the disease possible, one that connects plague in any part of the world, at any time, with plague everywhere else in the world. No longer do we have to wonder how long plague has been present in the Americas. (Answer: since about 1900.) No longer do we have to wonder how the strains of plague in East Africa relate to those in South Africa. (Answer: they belong to completely different radiations of plague.) This story rivals any of the great commodity stories that we have seen in global history of late (like those of cotton, silver, or sugar), in that it involves a single organism, one that is still so like the ancestor it shares...
with another, comparatively harmless pathogen, as to be considered “clonal.” Even before the whole genome of Yersinia pestis was sequenced in 2001, microbiologists had begun to infer the rough outlines of the bacterium’s genetic history. Once a few additional sequences were available, it became possible to compare whole genomes with partial sequences collected from around the world. In 2004, it was argued that the concentration of evolutionarily ancestral strains of Y. pestis in western China suggested that that might be the organism’s ancestral home. By 2010, a whole global narrative had been constructed of Y. pestis’s dissemination.

Separately, in another field of the expanding genetics universe, other microbiologists were refining techniques to collect, reconstruct, and identify fragments of genetic material from the remains of victims of known plague outbreaks. This is the adna (“ancient DNA”) I briefly recounted above. Although Y. pestis often enters its mammalian host through the bite of an ectoparasite (usually fleas, but also ticks and possibly lice), and thereby enters into the lymph system (causing the distinctive buboes of bubonic plague in lymph nodes of the groin, armpits, or neck), by the time it kills its host it will have passed into the bloodstream and therefore move throughout the entire body. The idea occurred to early researchers that remnants of a blood-borne pathogen might remain in the teeth of victims: while blood comes into the teeth to feed the soft inner pulp (and so brings any blood-borne organisms with it), the hard external enamel offers a sterile casing that might preserve genetic material for considerable periods of time.

After much trial and error (nicely recounted in an essay by Lester Little in 2011), the field finally yielded results proving the presence of Y. pestis in historical remains from pre-modern Europe. Initially, those reconstructed samples were only partial genomes: enough to confirm that Y. pestis was present and to begin to distinguish strain from strain, but not enough to fully characterize it genetically. In 2011, however, came the breakthrough study that announced a complete sequencing of Y. pestis from a well-dated Black Death cemetery in London. This study, produced by research groups working at Tübingen in Germany and McMaster University in Canada, was published with great hubbub in the popular press, including an editorial–not simply a Science-page write-up, but an editorial–in the New York
In 2013, another international team of researchers took the adna genomes sequenced by the Tübingen/McMaster group and plotted them onto a revised phylogenetic tree, which drew on a total of 133 whole genome sequences (most of them newly sequenced for this project). Every genomics study on *Yersinia pestis* that has been published since then, whether it is reporting on modern samples, such as in Uganda or Madagascar or Kyrgyzstan, or on adna, such as studies on the newly discovered Bronze Age plague strains in 2015 and 2017, the Justinianic Plague in 6th-century Bavaria, or sequences from medieval Barcelona and Bolgar City, or early modern Germany, or 18th-century France, has produced results that can fitted into the overall phylogenetic tree first proposed, in rough outline, in 2004, and given its present 5-branch structure in a study by Cui et al. in 2013.

Below (fig. 1) is my own marked-up version of the Cui et al. 2013 tree, which shows the major plague events of history. And there, at the center of it all, is the Black Death and its immediate aftermath, the *pestis secunda* (1359-1363), as documented from the two genomes (the maroon triangles) sequenced in 2011 from London.² (The Justinianic Plague, indicated by the overlain blue box, and Bronze Age genomes, not shown at all, were sequenced after the study by Cui and colleagues was published.) Right before the Black Death, at a date estimated to have fallen at some point between 1142 and 1339, *Y. pestis* diverged into four new branches. That event—which technically is called a “polytomy” (manifold divergence) but was dubbed more evocatively, by Cui and colleagues, the “Big Bang”–created modern strains of *Y. pestis* that persist to this day. We can say now, for example, that the Black Death strain that reached western Europe in 1347-48 was indeed an early progenitor for what would, 600 years later, become the source of the Third Pandemic in Hong Kong. In calling these genetics findings a “gift” to historians, there-

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²The 2011 study by Bos et al., actually had an error. A sample (labelled 6330) that they identified as coming from the East Smithfield Black Death Cemetery, a burial ground which could be precisely dated to 1348/49-1350, in fact came from a later burial site, associated with the second major wave of plague, the *pestis secunda* (1359-63, though starting in London, it seems, in 1361). See Green and Schmid 2016 for more details about the significance of this finding for *Y. pestis* history; for an example of the *pestis secunda*’s differing demographic impact in England vis-à-vis the earlier Black Death, see DeWitte and Kowaleski 2017.
Therefore, I do mean precisely that: because of methods they brilliantly developed to ask questions in biology, the geneticists were able to create a coherent biological story of plague that no amount of scouring through textual sources was ever going to give text-reliant historians. And they have shown, decisively, that the most important chapter of plague’s history falls in the Middle Ages.

Truth #2: The Black Death Was Likely Even More Devastating Than We Have Ever Imagined

We will never have reliable estimates on the mortality levels caused by plague in the 14th century, for the simple reason that no mechanisms of systematically recording deaths existed at that time. Various kinds of proxy evidence are being investigated.
(see next section), and it continues to be important to economic history and social history to wrestle with the severity of impact. Here, however, my emphasis is not on our (in)ability to count, but on our inability to even imagine the extent of devastation in some places. On this point, we need to reckon with two different issues: (1) the possible underestimation of the extent of mortality within those areas we have always included in our geography of the Black Death; and (2) the underestimation of the geographic extent of plague's spread in the late medieval and early modern world that had previously been beyond our ken. I will return to the first issue in the next section. On the latter one, we don't have even rough estimates of mortality levels, since we have barely perceived that there were mortality events we should be looking for.

Look again at the phylogenetic tree above. In the upper left quadrant is Branch 1, one of the four new distinct *Y. pestis* lineages created by the “Big Bang.” Since Cui *et al.* 2013 was published, geneticists at Munich on the one hand, and at Jena and Tübingen, on the other, have established that a distinct new branch of *Y. pestis* caused plague outbreaks in Europe from the 14th to 18th centuries. Of the two 14th-century genomes shown on the tree (the two maroon triangles near the center), the earlier strain spawned “offspring” in Europe documented at Manching-Pichl (14th century) and Brandenburg (17th century), Ellwangen (15th-16th century), and Marseille (18th century). I now refer to this lineage as **Branch 1A**. If we can assume that all plague epidemics, save one, in western Europe between 1347 and 1722 (the end of the last Marseille plague) were caused by this strain—which seems now to be utterly extinct—then we can begin to assess how much plague devastation is nowhere indicated by the living strains that now make up the biological narrative we have for *Y. pestis*. To put it another way, if we didn't have our written record testifying to the millions of plague deaths in outbreaks in countless European communities between the 14th and 18th centuries, we would have no trace that this devastation had happened, simply because, in the end, the organism burned itself out, exhausting its supply of hosts.

If we turn to areas without written records, therefore, such as sub-Saharan Africa, a collection of essays, edited by Gérard Chouin, exploring the question of plague's possible presence in sub-Saharan West Africa, and in East Africa, in the late medieval and early modern
or areas where the kinds of sources we have used to reconstruct mortality levels in, say, England or the Low Countries, such as virtually unbroken series of tax rolls and other financial or legal records, don't exist, such as Egypt and other areas of the Middle East, how would we know whether or not comparable mortality events had occurred? Now look once again at the phylogenetic tree. Those branches (and subbranches) of plague that have survived in Branch 1B (all the rest of Branch 1 shown on the tree) likely did so primarily by subsisting through sequences of rodent hosts, probably wild ones. Yet we know that, at some point, some of those strains must have passed through human populations. Humans and their technologies of long-distance transportation, it seems, are the chief reason Y. pestis has been able to achieve the transcontinental (and intercontinental) spread it has enjoyed in its last 5000 years of existence. Thus, for every living strain of plague, we need to ask how many minor extinct strains burned out in the bodies of human victims.

Now look one last time at the phylogenetic tree. Branches 3 and 4, creations of the same late medieval polytomy as Branch 1, have only been documented in a handful of samples in western China, Mongolia, and southern Siberia. We do not know at the moment whether their historical impact may have extended beyond that narrow region. But look at Branch 2 (which is made up of two twin subbranches, 2.MED and 2.ANT). It has been every bit as prolific as Branch 1 in creating surviving strains. Although its geographic footprint is not so wide as Branch 1’s—it has never been documented in the Americas, for example, and only on the very northern edge of Africa—its reach across the breadth of eastern and central Eurasia is astounding. We have no adna yet to tell us the backstory of Branch 2. Whether it will end up explaining the many epidemics reported in 14th-century China, and later, remains to be determined; at the moment, we have no descriptive accounts of sufficient detail to postulate plague as the cause. But if the story of the extinct Branch 1A is any guide, the phylogenetic evidence of Branch 2’s prolifera-

world, is forthcoming from the online journal Afriques.


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tion everywhere from Mongolia to India, from China’s far eastern province of Jilin to as far west as Libya, will likely tell us a story of extraordinary human suffering.

Truth #3: Sometimes Silence is the Only Evidence We Will Find

Everybody dies, but it is actually rare in human history for many thousands, or even millions, of people to die all within a short period of time. Dealing with sudden and widespread death was one of the unique challenges in plague epidemics, something we see both in the Justinianic Plague in the 6th century, and in acute episodes of the Second Plague Pandemic. Mass graves are being increasingly investigated as unique witnesses of demographic catastrophe. Mass burials due to epidemic mortality must, of course, be carefully differentiated from battle sites and a few other situations (like volcanic eruptions or tsunamis) that might cause mass death. But that differentiation is usually easy to do, leaving archaeologists with the possibility of assessing various aspects of plague’s impact, such as the prior health status of the individuals killed by the disease or the relative health of individuals who survived the epidemic. We also, chillingly, are now able to document the reality of the assaults on Jewish communities that started with plague’s first arrival on the European mainland in the spring of 1348.

Sometimes, however, there are no remains at all. It is a commonplace of plague narratives, both in the Islamicate world and Christian Europe, to measure the level of catastrophe simply by saying that there weren’t enough people left living to bury the dead. Silence is being recognized as a feature unto itself now, an indication of catastrophic depopulation. Evidence of silence may seem an oxymoron, but in fact the edges of such absence are often visible. Bureaucratic records, for example, that would ordinarily, in their very monotony, record the cycles of living and dying, might simply cease, because the clerks who would normally prepare them have died, or the courts that adjudicated them have stopped functioning. Archaeologists

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5In her superb monograph, Plague and Empire in the Early Modern Mediterranean World: The Ottoman Experience, 1347-1600 (Cambridge: Cambridge University Press, 2015), Nükhet Varlık documents that the Ottoman Empire went from a pattern of infrequent outbreaks in the 14th and 15th centuries, to a pattern of almost annual outbreaks in the 16th century. Whether or not that shift was accompanied by a change in the Y. pestis strains involved cannot yet be determined. Branch 1B does have some points of overlap in its geography with Branch 2; both, for example, are now found in Qinghai and Yunnan Provinces in China.
are particularly adept at determining the diminution of human activity or even its total disappearance. Agricultural lands become overgrown and go “wild” again, something that can now be studied via pollen deposits; architectural remains can show a cessation of basic maintenance to allow habitation, accompanied by the absence of any new building; villages can “disappear” into the forest. In perhaps the most innovative technique yet devised, archaeologist Carenza Lewis has used crowd-sourced archaeology to assess the changes in human habitation of different areas of eastern England in the period after the Black Death. Ceramic pottery is an excellent record of human habitation because it’s very commonly used by most social classes; it often is produced according to the fashion of the times, which makes it roughly datable; it usually becomes useless when it’s broken because (unlike, say, metals which can be melted down) it cannot readily be recycled; and it tends to last a very long time once abandoned. Lewis had the idea to do lots of small test digs to see what areas seemed to continue to produce signs of activity, vs. areas which, although previously active, were later “quiet.” Her results were stunning.

Another kind of silence is iconographic silence. We, who live now in an imagesaturated world, accustomed to over a century and a half of photographic witnessing of events, and over 600 years of print media, find it hard to grasp the fact that only a handful of images depict plague-related scenes in the first half century of Europe’s new encounter with the disease. And none of them depict diseased bodies. The first surviving images of the distinctive buboes of bubonic plague don’t appear until the 15th century, at least half a century after the disease struck Europe. In other parts of the world, we have no images at all until centuries later. The modern proliferation in both scholarly publications and on the Internet of misconstrued images masquerading as “plague” has recently been revealed for the error it is. As with the pottery shards and missing bureaucratic records, sometimes we have to accept that what we’re looking for simply isn’t there.
Truth #4: The Black Death Never Ended

The mid-fourteenth century “pestilence of mortality” (as many Latin documents termed the Black Death) or “the universal plague” (as chroniclers writing in Arabic called it) did eventually pass. The crises of mass deaths abated, cemeteries were closed, and property (once courts began to function again) was divided up among the survivors. As noted, plague is not a human disease and cannot be sustained very long via human-to-human transmission. But the organism, *Yersinia pestis*, persisted. Textbooks will often list a series of outbreaks that followed the 1346-53 pandemics, and there is excellent monographic work on particularly large plague outbreaks in the early modern era, such as at Seville in the 16th century, or Italy and England in the 17th.\(^6\) There is more explicit discussion now of the Second Plague Pandemic, that correctly sees the Black Death not as an isolated phenomenon but the beginning of a long shared trauma, tied closely to the period of the Little Ice Age and an age where many more diseases besides plague alone afflicted the populations of both Old World and New. But we have reckoned too little with what these subsequent, lesser “pandemic” outbreaks actually meant as disease phenomena. The process of focalization has now become a research question in its own right, and it is central to figuring out how the long sequence of plague outbreaks that characterized much of the Old World between the later 14th and 19th centuries became established.

Between 80 to 90% of plague strains identified in the modern world took their origin after the great late medieval polytomy. Looked at another way, all those modern strains are evidence of how many locales proved continually hospitable to plague after the Black Death. Plague foci (plural of the Latin, *focus*) have been well studied in areas where plague persists in the modern world, such as Russia, China, and, since the early 1900s, much of the American West. Such areas may be fairly small, but as long as *Y. pestis* can keep moving from flea to animal host to flea to host, it will persist.\(^7\) Virtually all of these reservoirs would have had the potential to cause

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\(^6\) The essay reviews of Alfani and Murphy 2017, and Varlık 2017 provide excellent surveys of this literature.

\(^7\) Other possible stages in *Y. pestis* persistence have been postulated, such as a possible telluric (soil) phase or absorption by amoebas. These have not yet been proven, however, to be normal
new human outbreaks, provided that environmental circumstances facilitated the organism's propagation and, crucially, its transportation to areas of human habitation. Currently, we have no reasonable idea how many times that process may have occurred. Since many strains may have proliferated for a brief time and then burned out, we must assume that plague's late medieval and early modern footprint was much more extensive than we can yet document. The long history of plague in the Ottoman Empire has now been written, and we have good accounts for parts of the Low Countries, Russia, and the Baltic Region. Nothing comparable yet exists for the similar stories that must yet be written for North Africa, India, and China. Collectively, however, the new genetics has taught us that what is now regularly called the Second Plague Pandemic needs to be seen as part of the Black Death story, a sequel that, in reality, extends to the present day.8

Going Forward: The Black Death Must Be Taught as a Developing Field

I am currently writing a textbook on the Black Death. As the present account will have indicated, my objective is to create a new narrative of the Black Death as an epidemiological phenomenon: not just a single event but the beginning of a long "disease regime" that characterized not only Europe, but also the Ottoman Empire, and much of eastern Eurasia for many centuries. The question has been raised, from archaeological data, whether the Black Death also struck sub-Saharan West Africa, whereas genetics makes it clear that in East Africa, the plague regime initiated by the Black Death and extending, apparently, the entire length of the Nile all the way to the Great Lakes, persists to the present day. And it was because the particularly adept Branch 1B lineage migrated out of its late medieval focus (southern Russia? the western Caucasus?) and reached Yunnan Province (in the 17th century?) that a new proliferation of plague became possible at the end of the 19th century. Reaching the major shipping port of Hong Kong and exploiting now the elements of the Y. pestis life cycle.

8I have not, in this account, given attention to the questions that have long animated most Black Death historiography, to wit, the economic impact of the massive late medieval mortality. The work of Guido Alfani has been particularly important in connecting the late medieval and early modern narratives in demography and economics; Alfani 2017 provides a very good survey of the literature. On larger global impacts of Europe's new disease regime, see Belich 2016.
new, faster technology of the steamship, plague was now carried to Madagascar, India (again), South Africa, Australia, and the Americas, as well as revisiting its old haunts in Europe and the Mediterranean.

This evolutionary story has now shown a robustness in incorporating new findings that suggests it will endure. Our phylogenetic tree of \textit{Y. pestis} will get “bushier” with every new \textit{adna} sequence or \textit{new subbranch} found in hitherto unsampled locations. But its general structure will likely hold. However, while that global, evolutionary narrative of plague’s history is likely to remain intact, many particular elements of the story will undoubtedly change in the coming years as new research in a variety of fields is explored. A wise teaching program, therefore, would be to develop command over a few key issues as modules, which can be individually updated from time to time without having to revise the entire unit. (See the teaching plans below.) Here are some topics that are most likely to see major revision in the immediate future.

\textit{The Black Death Map.} The greatest need in the field right now is cartographic. Verbal reports of plague have been the foundation on which maps of the Black Death have been drawn for decades. Those \textit{demand scrutiny} themselves, of course, but they will continue to have their utility. Additionally, maps that show where \textit{Yersinia pestis} has been recovered from human remains—either from biochemical assays, partial genomic studies, or whole genome sequencing—bring a cold reality. (Instructors will need to assess the appropriateness of using images of mass graves in class, according to the maturity of the students and community norms.)

However, with the transition from partial genome studies to whole genome studies, as we have seen, we are in the position to \textit{track} specific strains of plague across space and time. Currently, we have no adequate maps to capture the basic geography of the late medieval polytomy and the initial spread of plague in the 13th and 14th centuries. The biggest absence is anything reflecting current understandings of the Black Death’s devastation or plague’s creation of new foci across Eurasia and into Africa. (An important exception is the maps created for a 2015 study of \textit{plague in the Ottoman world}.) Currently available maps can be used in the classroom, but only if they are immediately critiqued as not capturing the narrative as we presently
understand it. For example, the map below (fig. 2), included in an open-access world history curriculum for high school, tries to capture William McNeill’s notion that plague spread all the way from southeastern China to the Crimea in the space of just over a decade in the 1330s and 1340s. Our current understanding of \textit{Y. pestis} genetics and 14th-century history would make such a transmission scenario impossible. The strains involved in the two areas would likely have had no direct relationship, other than both being the result of centrifugal spread out of a central Eurasian focus; pan-Eurasian transmission does not accord with any narrative accounts we have either of plague’s spread or of commercial activities in the period. Similarly, maps showing plague striking western Europe in “waves” have been shown to be misleading for a variety of reasons, and not simply because the underlying research has, in some cases, failed to recognize the extent of plague’s effects on such areas as Bohemia and the Low Countries. A new Black Death map, conceived on a GIS basis and allowing for dynamic overlays plotting the “routes” of different strains of \textit{Y. pestis}, would be a boon in the classroom.

\textbf{The Mechanism of Spread.} Related to the issue of the timing and routes of plague’s spread in the 13th and 14th centuries (and thereafter) is the perennial conundrum of the mechanism by which plague spread. The idea of “rats on the march” is, of course, ridiculous but the fact of the matter is that we have no alternatives that can plausibly explain plague’s exceptionally efficient transmission in pandemic circumstances. Was it the grain trade? Was it the textile trade? Was it lice? Have we underestimated how much pneumonic (direct person-to-person) transmission may have played a role? Given how much variation there is in plague transmission historically (maritime vs. overland, summer but sometimes winter, high elevations and low), it is unlikely that any single mechanism will explain everything. But efforts to address this critical question will continue since, as noted above, genetics has already ruled out any significant difference in the causal organism that could explain the extraordinarily high mortality, and extraordinary geographical reach, of the 14th-century pandemic.

\textbf{The Role of the Mongols.} Now that genetics has tied together the genesis of Branches 1-4 of the late medieval \textit{Y. pestis} phylogeny, we cannot help but see the larger con-
Figure 2: A map of the Black Death's alleged routes across Eurasia, drawn to reflect the theories of William McNeill from 1979; distributed for an open-access high school World History teaching platform, *History For Us All*, and accessed 02/07/2018. This accords not at all with current genetic understandings of *Y. pestis*’s evolutionary development in the 13th and 14th centuries.
text of what unites much of Eurasia together in this period. And that, of course, is the fact that the largest pandemic in human history occurred at exactly the same time as the largest land empire in human history. Yet the Black Death has figured hardly at all in Mongol historiography (which has actually been a thriving field in recent years), despite the obvious coincidence in the timing of the polytomy and the rise (and fall) of the Mongol Empire between 1205 and ca. 1368. The new genetics, moreover, has now established the importance of the obvious geographical overlaps between where the strains leading up to the polytomy are currently found (far western China and Kyrgyzstan) and where the key areas of Mongol activity were. There remains, however, the absence of testimony in Mongol sources to plague, either as the cause of individual deaths or as the cause of major epidemics. But that may be a function of two factors: first, the basic character of plague, which is not a human disease, meaning that unless there are human outbreaks on-going, it is unlikely to elicit notice in written records; and second, conventions in medical thought and writing, which don’t necessarily emphasize superficial symptom description in diagnostics or explanation. Fortunately, there is new work being done that casts a fresh eye on written sources from areas under Mongol rule and it is beginning to suggest that, as noted above, the apparent silence of our sources has been covering over events of significant magnitude. Multidisciplinary teams will be needed both to sift through the extraordinarily diverse records of the Mongol Empire (which are found in over two dozen different languages) and to reconstruct a bioarchaeological record of a disease that managed to traverse 1000s of kilometers of open steppe and desert with nary a trace other than microbial descendants that inhabit that terrain today.

A One-Week/One-Day Curriculum for Teaching the Black Death

Most instructors will not be able to devote a whole semester to plague as a phenomenon in global history. The following curriculum allows incorporation of a mini-narrative of plague as a historical force, with the explicit intent of raising questions for the student about how to think about humankind’s relationship with infectious disease and why mass mortality needs to be assessed for both its demo-
graphic and social effects. The climate issues have been minimized, not because the coincidence of plague pandemics with major cooling episodes is not well documented now (that is, the onset of the Late Antique Little Ice Age, including major volcanic activity in 536 and 541, with respect to the Justinianic Plague, and the “Great Transition” preceding the early modern Little Ice Age, with respect to the Black Death and the Second Plague Pandemic), but only because the causal links with plague’s biology have yet to be convincingly explained.9

Some instructors, of course, need to compress their coverage of the Black Death into a single day, or less. For them, I’ve indicated the main take-home messages that any student of history should now understand about this epidemiological catastrophe. There are a number of syllabi publicly available which have extensive bibliography. My syllabus and extended reading list for my own Black Death course are already available online, and can be used to find both primary and secondary sources. (For K-12 teachers, the Black Death section of this teaching guide is very handy.) Additionally, I teach a course called “Global History of Health” that weaves the narratives of plague in with the origin, dissemination, and globalization stories of seven other “paradigmatic diseases,” that is, infectious diseases whose characteristics of zoonotic or environmental origin, proliferation, and containment can serve as models for understanding humans’ relations to microorganisms throughout history. An essay situating plague’s history within the larger context of disease and climate in medieval Eurasian history can be used to address comparative elements of plague’s Eurasian history alongside those of malaria, leprosy, and smallpox.

I haven’t made suggestions for specific readings. Because plague studies has become a dynamic new field of study, not only for paleogenomicists, but also for archaeologists (who study mass burial sites and indications of health before and after the Black Death) and traditional document-based historians, any list provided here would need almost immediate supplementation.10 Rather, I have suggested major

9I address these issues in an extended review of Bruce M.S. Campbell, The Great Transition: Climate, Disease and Society in the Late-Medieval World (Cambridge: Cambridge University Press, 2016), in Inference: International Review of Science (forthcoming).

10Keeping up with plague science is a task unto itself. For students who are biologically inclined,
themes and questions that, as I have explained above, should now be part of our basic approach to thinking about plague historically. In linking occasionally to specific studies, I do so because they are good illustrations of my point, not because I am necessarily recommending them for classroom use.

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<tr>
<th>Topic/theme</th>
<th>One Week (2 class periods)</th>
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<tbody>
<tr>
<td>Precedents (did this ever happen before?)</td>
<td>- Bronze Age plague&lt;br&gt;- Justinianic Plague</td>
<td>[skip]</td>
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<tr>
<td>The biology of plague</td>
<td>Epidemiological significance of bubonic vs. pneumonic presentations</td>
<td>Provide a basic CDC/WHO level summary</td>
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<tr>
<td>The geography of plague</td>
<td>Plague as a disease of the Eurasian steppe for the past 5000 years; <em>Y. pestis</em>’s ability to focalize in other environments</td>
<td>Introduce, but immediately critique, traditional textbook map</td>
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<tr>
<td>- The <em>Y. pestis</em> polytomy (the late medieval “Big Bang”)&lt;br&gt;- Instigating factors of spread</td>
<td>- Mongol Empire&lt;br&gt;- Changing climate&lt;br&gt;- Was there an eastern counterpart to the Black Death in wester Eurasia? (You're holding the door open here for any new research on China.)</td>
<td>Explain that mechanisms of dispersal in 13th/14th century are still unclear</td>
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This new overview of plague science will be useful: Rufui Yang and A. Anisimov, eds., *Yersinia pestis: Retrospective and Perspective*, Advances in Experimental Medicine and Biology 918 (Berlin: Springer, 2016). The free biomedical literature bibliographical database, PubMed, captures work published in multiple languages.
### The Western Pandemic

- **What happened at Caffa?**
- **Spread into the Mediterranean**

#### First strikes:
- Constantinople
- Aleppo
- Alexandria
- Almeria
- Sicily
- Florence
- London
- Norway

#### Narrative accounts can be analyzed to stress:
- Contemporaries’ understandings of the geography of plague's spread maritime spread
- The question of the disease's contagiousness (is it spread from one person to another?)

#### Traditional primary sources work very well to show mounting anticipation and fear

#### Secondary strikes:
- Iceland
- West Africa
- East African coast

For an abbreviated lecture, these more distant sites of plague activity can only be listed in passing. Nevertheless, it is important to note that research keeps expanding the terrain that plague is likely to have reached in the late medieval period. For Global History courses, these peripheral sites might become the focus.

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<tr>
<td>Panic</td>
<td>The topic of civil chaos can be approached from a variety of angles, and there is no shortage of primary sources (at least for Europe) to address this question. The question of scapegoating demands great care. Sources are available in English for the worst outrages against Jewish communities in Savoy and the Rhineland. The excavation of a burial ground in Spain serves as powerful testimony to the attacks. A new monograph provides more contextualization of the long-term consequences for Europe's Jewish communities.</td>
<td>Boccaccio still works well for this, though incorporation of an account from at least one other geographic area would offer useful contrast. Ibn al-Wardi, who witnessed plague in Aleppo (and died from it in 1349), is evocative.</td>
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<td>Silence</td>
<td>This is a field of disaster history that has not been well conceptualized yet. The work of Carenza Lewis, mentioned above, would be an excellent starting point, not least because its concepts are so straightforward and understandable to students, without requiring any background in science or archaeology.</td>
<td>An example from Lewis’s research on eastern England will suffice to demonstrate what is meant by abandonment. The misuse of the leprosy images example works very well to teach the problem of using improperly sourced materials.</td>
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<tr>
<td>Reestablishing Order</td>
<td>A few examples will suffice to show the reestablishment of daily routines of social interaction. However, the idea that the Black Death was &quot;good&quot; for societies, in that it raised the standard of living for survivors, needs to be tempered. Those studies are based on single regions. Other areas were permanently crippled by the disaster and never recovered their economic standing.</td>
<td>ditto</td>
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<td>The <em>pestis secunda</em>, 1359-63</td>
<td>Caused by a new strain of <em>Y. pestis</em>; this has been documented in both London and Bergen op Zoom (NL). This likely reflects a new introduction of plague into the Mediterranean basin from the Black Sea, though unlike the Black Death strain, this seems to have burned out in Europe, even though it would go on to spawn the rest of Branch 1B.</td>
<td>[skip, other than to indicate that plague did return by 1360 and thereafter, suppressing demographic recovery]</td>
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Topic/theme | One Week (2 class periods) | One Day (1 hour period)
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Focalization
• The normalization of plague in the context of the Little Ice Age
• The normalization of plague control protocols (including quarantine and the *cordon sanitaire*)

Establishing the existence of historical plague foci is still a major task. It has only been in the past 4 years that the possibility that Europe may have harbored its own foci (rather than reimporting plague repeatedly) has been suggested with plausible evidence (both documentary and genetic). Comparable work has not yet even begun for Africa or central and eastern Eurasia. Nevertheless, both genetics evidence and the documentary record make clear the survival of plague in diverse environments in the early modern period. Future studies will almost certainly underscore the role of the Little Ice Age in facilitating this perduring threat.

Although a few public health (*PH*) measures were implemented right at the time of the Black Death (usually drawing on pre-plague *PH* law), most of what we think of as specific plague legislation developed only after multiple experiences with the disease. It is important now to supplement the well-known European cases with data on plague control from the Ottoman Empire.

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<td>Early Modern epidemics</td>
<td>Any one of these outbreaks would serve to show how the persistence of rural foci (possibly in highland areas) posed a perpetual threat to neighboring human settlements. These were societies now inured to plague—they knew its signs, its symptoms. But they still had little power to stop an outbreak once it was underway.</td>
<td>The challenge here is to convey that these well known urban outbreaks were symptoms of a plague regime that had established itself across much of Eurasia and North Africa. Some background on the Little Ice Age is necessary. These outbreaks were neither unique nor inexplicable.</td>
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<td>• Ottoman Empire</td>
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<td>• Seville, 1582 and 1599-1600</td>
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<td>• northern India, 1615</td>
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<td>• Italy, 1630</td>
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<td>• Seville, 1647–1652</td>
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<td>• London, 1665</td>
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<td>• East Africa</td>
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<td>• Marseille, 1720-22</td>
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<td>Plague's modern history</td>
<td>Examples of plague cases in the 21st century can be readily found on Google. It is important, however, to stress that in some cases, especially Madagascar, continuing problems with plague aren't holdovers from pre-modern times, but rather the result of quite modern globalization. Cases showing panic that plague can still elicit are important for conveying the cultural memory that many societies still have. Clips from Soderbergh's <em>Contagion</em> (2011) are useful.</td>
<td>Plague was never eradicated. In Europe, the reasons for its disappearance are unclear, though environmental stress may have been a factor, as were almost certainly the control measures that obstructed human-facilitated plague movement and, thus, refocalization. Elsewhere (such as Australia, which never saw focalization of the disease), modern control of rodents, use of antibiotics, and field surveillance systems are what brought plague under control.</td>
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Acknowledgements: As explained above, the NEH Summer Seminars in London in 2009 and 2012 were critical to my ability to explore, out of the limelight of publication, the mounting questions I had about how (if at all) the findings from microbiology and what we now call paleogenetics were to be incorporated into the work we do as historians of the medieval past. But that work was also sustained by a wonderful international community of scholars on MEMED-L, the listserv for medieval medicine I started in 2008. Now grown to over 800 subscribers, it has been a forum in which I could think out loud about how to assess the significance of each new piece of plague research that has come out in the past decade. Kudos, too, to Michelle Ziegler, a biologist by training but a historian at heart, whose blog *Contagions* regularly offers a thoughtful take on plague research, from the perspective of someone who actually knows the biology. Her small list, the Plague Working
Group, was a vital resource and sounding board for several years. Finally, the Institute for Advanced Study (Princeton) offered me the opportunity to engage in some “unobstructed pursuit of useless knowledge” during 2013-14, when I edited the inaugural volume of *The Medieval Globe: Pandemic Disease in the Medieval World: Rethinking the Black Death*, whose open access publication was underwritten by the World History Center at the University of Pittsburgh, through the kind graces of Patrick Manning. This experience allowed me to turn my private queries of the “So what?” question of genetics’ contributions to History into a map of potential lines for future inquiry.