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PREFACE
THE BLACK DEATH AND EBOLA:
ON THE VALUE OF COMPARISON
MONICA H. GREEN

It was as if the voice of existence in the world had called out for oblivion and restriction, and the world had responded to its call.

Ibn Khaldun, d. 1406

The essays that make up Pandemic Disease in the Medieval World: Rethinking the Black Death grew out of dialogue first begun in 2009. The volume itself began to come together in the spring of 2013 and was sent to press a year later. Little did we know that by the time the volume would appear in November 2014, the world’s relationship to epidemic infectious disease would have taken such an abrupt turn. At the time of this writing, the 2014 West African Ebola epidemic has already caused over twenty-one thousand cases and taken over eight thousand lives.¹ Declared a Public Health Emergency of International Concern on August 8, 2014 by the World Health Organization (WHO), the Ebola outbreak prompted a United Nations (UN) Security Council meeting several weeks later. At that session on September 18, the resulting UN resolution called the outbreak a “threat to international peace and security,” garnering the highest level of support in the history of the organization. The West African Ebola outbreak has disrupted the economies and trade relations of countries in West Africa and beyond. It has decimated the already strained healthcare personnel of the three most affected countries. It has left hundreds of children orphaned and disrupted the education and welfare of many thousands more. For the families whose loved ones make up those statistics, no relativizing of numbers can offer solace. From the perspective of the devastated countries of Guinea, Sierra Leone, and Liberia, the Tunisian historian Ibn Khaldun’s bleak assessment of the world after the Black Death could just as well have been written today.

¹ I will refer to this as the 2014 outbreak, even though evidence suggests it began in late 2013 and, at the time of writing, gives every sign of continuing well into 2015.
Plague is not Ebola Virus Disease. Many of the easy parallels that were drawn in the popular press in mid-2014 between the unfolding Ebola outbreak and the medieval Black Death were exaggerated or careless. In several respects, Ebola is better compared to other diseases. The current Ebola epidemic has brought back memories of the 2002–2003 SARS epidemic, whose intercontinental spread was also abetted by airline travel; or the ongoing HIV/AIDS pandemic, which has been surrounded by intense debates about drug development and ethical provision of treatment. In its mode of transmission via human bodily fluids, Ebola is similar to cholera, though this virus is fragile in the extreme compared to the hardy bacterium, which thrives in open brackish water. In its current form—a virus passing directly from one human to another—Ebola more closely resembles smallpox, which made its zoonotic leap into humans several thousand years ago. Nevertheless, with comparable case fatality rates and rapid progress from infection to death, plague and Ebola elicit a terror few other infectious diseases have produced. With this re-issue of our essays in book form, it will be good to set the Black Death—or really, the Second Plague Pandemic in all its immensity—into a larger context. And in that task, comparisons with the current Ebola outbreak are indeed useful, both for better understanding our collective past and for anticipating our common future.

If a Tree Falls? Invisible Pandemics

In August through October of 2014, dire predictions of Ebola’s exponential growth were being made on the basis of mathematical modeling. One particularly alarming model, released 23 September, predicted that the epidemic might reach 1.4 million cases by January 2015, in Sierra Leone and Liberia alone (Meltzer et al. 2014). Similarly, predictions of exported cases—fueled by the documented transfer of the disease to Nigeria late in July, followed by a similar case that reached the United States in September—raised fears of outbreaks not simply elsewhere in Africa, but in large population centers in Asia and Latin America, which might not have the public health infrastructure to identify and contain new cases. The West African Ebola epidemic has not, as of this writing, become a pandemic. But it certainly had the potential to become one. And that is the more important point.

All pandemics start as local outbreaks. In the case of the West African Ebola epidemic, it is now understood to have started as early as December 2013 in a rural area of Guinea (Baize et al. 2014; Saéz et al. 2014/2015). It was not until March 2014 that the outbreak was officially acknowledged,
and then only because the alarum had been raised by local doctors and the international medical charity, Médecins Sans Frontières (Doctors Without Borders), which already had a presence in Guinea to treat HIV/AIDS and malaria. In the early, “silent” phase of transmission—when deaths were mounting but no diagnosis had been made to link them to Ebola—the disease was able to establish itself in three countries.

A major objective of the present volume is to make the radical claim that the Second Plague Pandemic likely had “silent” phases, too. As awful as we have always thought the Black Death to be, the essays in this volume argue that it was likely even worse, because its full geographical or chronological extent had never before been conceptualized. As Robert Hymes recounts in his essay here, many historians of China have long doubted whether plague was even present in medieval China. That debate is sure to continue, but it now has to take seriously the implications of the phylogenetics of *Yersinia pestis*, which point strongly not simply to *Y. pestis*’s origins in regions that now fall within China’s western borders, but also to a sudden evolutionary divergence of the organism around the thirteenth century. Just as historians of late antiquity have only recently realized how profound a role plague may have played in weakening the Roman Empire (see particularly Mitchell 2014), so with the Black Death we now have reason to suspect that it, too, had periods not simply when it was moving through wild animal communities with little or no human involvement, but also periods when it produced human outbreaks that we have failed to recognize as indications of epidemic disease. As we fully acknowledge here, to date we have no explanation for how plague might have moved across central Eurasia in the thirteenth and early fourteenth centuries.² And its postulated presence in sub-Saharan Africa still demands full investigation.³ But as both Ann Carmichael and Nükhet Varlık show in this volume, careful gathering and sifting of local historical data can produce evidence suggestive of plague’s stealthy focalization in new ecological niches.

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² Despite the rapid growth of Mongol studies in the past two decades (Biran 2013), the few scholars who have engaged with the question of plague simply repeat findings from McNeill 1976 or draw on western sources; an exception is Schamiloglu (1993). Aside from Buell (2012) (who denies that there were any significant outbreaks of human plague in Mongol China) and Hymes (in this volume, who asserts that there were several, pushing their presence back to the thirteenth century), there has as yet been no engagement with the genetics research on *Y. pestis* by historians of Central Asia.

³ After these essays originally went to press, I discovered that Gérard Chouin, now at the College of William and Mary, has taken his earlier suggestions about the possibility of a Black Death impact in late medieval West Africa and expanded them into a full research project. See Chouin 2013.
Relatively speaking, the 2014 Ebola outbreak did not long escape notice. But what if something like the Ebola outbreak had started a hundred years earlier, before cell phones and satellites and the Internet? We don’t know how old Ebola Virus Disease is, and have no way of knowing right now whether it has caused any large-scale human outbreaks prior to its official biomedical discovery in 1976. We do not have to imagine, however, the possibility of silent epidemics, or even pandemics. We already know, from the reconstructed history of the early decades of HIV/AIDS, how a local disease can become a pandemic—invisibly. HIV/AIDS did not begin in 1981, when the first study by the Centers for Disease Control described five cases of unexplained *Pneumocystis carinii* pneumonia in Los Angeles. Rather, it began in Cameroon around 1920 and spread soon thereafter to Leopoldville in the Belgian Congo (now Kinshasa in the Democratic Republic of the Congo) and beyond (Faria et al. 2014). By the time the first published clinical descriptions appeared in 1981, the virus had already spread to all five inhabited continents and at least a million people were already infected.

**Animal and Human Origins**

Both plague and the 2014 Ebola outbreak reflect our recent understanding that many infectious diseases are of zoonotic origin. In some cases, those viruses or microbial pathogens have been long accustomed to their animal hosts and cause only minimal disease. Once transferred into humans, however, these viruses or microbes become merciless killers. Ebola and plague are zoonotics in different senses, of course. Like HIV/AIDS, the current West African Ebola outbreak seems to have arisen from one animal-to-human transfer of the virus, and then took off in a series of continued human-to-human transfers because of shared practices of care-taking in the home or preparations for burial (Gire et al. 2014). Plague, in contrast, can only maintain short bursts of human-to-human transfer and then only under two conditions: either in the form of septicemic plague, when certain medical interventions—such as lancing of buboes (see the

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4 Morse and colleagues (2012) estimate that roughly 80% of viruses, 50% of bacteria, 40% of fungi, 70% of protozoa, and 95% of helminths that infect human beings are of zoonotic origin.

5 I am referring here, of course, only to HIV-1, Group M, which has been responsible for the global pandemic and which, according to current understanding, passed to humans via a single transmission from a *Pan troglodytes troglodytes* chimpanzee. On the Ebola virus, see Baize et al. 2014 and Gire et al. 2014.
image on p. 230 of this volume)—can transmit the disease from patient to caretaker; or, as a more common mode, in the form of pneumonic plague, when the plague bacterium is transferred in aerosolized form from a person with infiltrated lungs to others not yet infected. Needing crowded (and often cold) conditions, pneumonic plague outbreaks burn out quickly. However, if we think of plague not as a single organism but as an ecological package—the rodent-arthropod-bacterium nexus—then situations that bring that package into close, commensal relationships with human populations can create sustained human disease at the population level. For both diseases, therefore, the potential to tap into human networks of communication is what allows them to turn from small outbreaks into widely disseminated diseases.

A study that appeared in September 2014 suggested that Ebola was likely already present in the animal populations of at least twenty-two African countries (Pigott et al. 2014). Although not yet definitively proven as the main transmitters of the Ebola virus, bats are the likeliest suspect (Saéz et al. 2014/2015); as flying animals, their ability to transmit the disease across wide regions is distinctive. That Ebola was already a disease “underfoot” across wide swaths of Central Africa was, of course, already known, a fact underscored by the second outbreak in the Democratic Republic of the Congo in 2014, which caused sixty-six cases, including forty-nine deaths (WHO 2014). However, unnoticed by Pigott and colleagues, and by all other accounts of the Ebola outbreak in West Africa in the summer and fall of 2014, was the fact that studies going back to the late 1970s already documented that individuals in Liberia and Sierra Leone carried antibodies to Ebola in their blood. It would seem, therefore, that Ebola was already widely disseminated in animal populations in sub-Saharan Africa (including West Africa) prior to 2013, and it is possible for similar outbreaks to occur so long as animals infected with the virus are consumed for food.6

Even before extensive genetics studies were done of the Ebola virus circulating in West Africa, it was noted that the 2014 disease outbreak

6 Van der Waals et al. (1986) and Knobloch et al. (1982), neither of which were cited by Schoepp et al. (2014), which found nearly comparable levels of seroprevalence of Ebola antibodies. Cf. Becquart et al. (2010) for a similar study of Gabon based on samples taken in 2006–08. Importantly, scientists have noted for more than a decade that major die-offs were occurring from Ebola in primate populations in West Central Africa; see Muyembe-Tamfum et al. 2012. Saéz et al. (2014/2015), however, found no evidence of such die-offs in primate or other non-bat populations in the area of Guinea where the 2014 outbreak is thought to have begun.
was different from all previous ones, moving faster and into larger populations. But this is not because the *virus* was significantly different from those strains that had already caused several dozen outbreaks across Central Africa since 1976; rather, the 2014 outbreak was different because *Africa* was different. In seeking to understand what sparked the initial zoonotic transfer, questions have been raised as to whether new agricultural practices or other interventions have disrupted ecosystems in West Africa, making certain kinds of human-animal contact more likely (Wallace et al. 2014). More significantly, it has been noted that Africa in 2014 was different from Africa in 1976 in that there was greater socio-economic diversity and new kinds of interconnectivity (Osterholm 2014). Whereas earlier outbreaks had occurred mostly in isolated rural regions, where there were few cars or other connections to major population centers, the 2014 outbreak was characterized by its quick linkage to large urban areas, which in turn facilitated further spread both near and far.

Plague, too, is now “underfoot” in many regions of the world. How it came to be so was not (so far as we know) because of transmission by bats, but because the micro-environments which *Yersinia pestis* demands—the rodent-arthropod-bacterium nexus—managed to hook into human networks supporting the material exchange of goods (grain, textiles, furs). That has happened at least three times in history, and (to judge from the surviving genetic evidence) likely more.7 To what extent the three known plague pandemics—the Justinianic (so-called because it seemed to begin in 541 CE, during the reign of the Roman emperor Justinian), the Second Pandemic (commencing with the Black Death), and the Third Pandemic (commencing in the mid-nineteenth century)—were all magnified in their intensity by changing climate conditions has not yet been pinned down, though evidence is mounting for a major climatic element in the first two cataclysms (Sarris 2012; Campbell 2013; Brooke 2014). But for both the Justinianic Plague and the Black Death, the climate element did not *cause* the pandemics. Rather, it seems to have served as an *amplifier* of conditions that were already in the making. Even if the dust-veil event of 536 CE, which was followed by several years of cold weather and darkened skies, can be called the precipitator of the plague epidemics that broke out around the Mediterranean, that still does not explain how *Yersinia pestis* had already managed to wend its way from the Qinghai-Tibet Plateau into nearby territories like the kingdom of Aksum (encompassing parts of

7 See below, pp. 40–41, on the so-called “Angola” strain of *Yersinia pestis*, and also pp. 14, 39, 43n21, 199n12, 200n16, and 208n30, on the still unexplained dissemination of the so-called MEDIEVALIS strain across much of Eurasia.
modern Yemen, Ethiopia, and neighboring states at the mouth of the Red Sea), which was one of the main trading hubs linking Indian Ocean trade with that of the Mediterranean basin in the fifth and sixth centuries (Gebre Selassie 2011; Seland 2014). Even if the extreme weather that Campbell (2013) has now documented for the 1340s precipitated the sudden explosion of plague into new European and Mediterranean environments, and (if we can extrapolate) facilitated the focalization and persistence of plague in the European Alps as the chill of the Little Ice Age took hold, as Carmichael suggests in this volume, Hymes (also this volume) suggests we still must look at the extreme warfare, more than one hundred years earlier in an entirely different part of the Eurasian continent, to see what forces nudged Yersinia pestis out of its marmot burrows. And as Harrison (2012) has documented for plague and several other epidemic diseases that terrorized the world in the past five hundred years, human networks of trade have continued to play their role.

Whether climate change has played a role in the two dozen Ebola outbreaks known to have occurred since 1976 is not at all clear. Nevertheless, in assessing the pandemic potential of both diseases, we have to look to common human practices that facilitate disease transmission and the ways that local practices can connect to global networks. Seemingly innocent practices of human provision—eating bats or chimpanzees and hunting marmots, whether for food or for furs—have created conditions ripe for disease transfer. But those individual circumstances of disease transfer only develop pandemic potential when they are linked to wider networks of human exchange.

Comparing Crises

The time is not yet ripe for a historical post-mortem on all we have seen in terms of political posturing, economic collapse, or human bravery and compassion in the past few months of the 2014 West African Ebola epidemic. Nor was Pandemic Disease in the Medieval World: Rethinking the Black Death conceived specifically for comparative purposes, since it is not a general summary of the later medieval plague pandemic. The reader will find here no definitive estimates of total mortality or rehearsals of social catastrophe narrated by Boccaccio and his contemporaries. We do not offer an account of the history of quarantine nor, indeed, do we have much to say about the history of medical remedies for plague. Rather, we present this as an opportunity to think through, in a new way, what it really means for infectious disease to emerge or re-emerge—to move quickly and widely through human populations which are never (whatever we like to think to the contrary) wholly divorced from their ecological landscapes.
There is an important trend now in historical studies: not only to move analysis beyond the limits of the nation-state and even the human organism, but also to move it into deeper time scales. Although some diseases (like malaria and various kinds of parasites) have likely been with humans for many millennia (e.g., Webb forthcoming), increasing congregations of human populations into urban settlements have amplified the impact of infectious diseases in a shorter timescale, perhaps some 4,000 to 6,000 years, in both the Old World and the New. No definitive shift separates us from that history of increasing urbanization and the networking of human communities through long-distance trade. Granted, the revolution in scientific understanding of infectious diseases brought about by the great late nineteenth-century revelations of germ theory did put us on a whole new footing with respect to understanding infectious disease. But only one infectious disease has since been thoroughly eradicated (smallpox). Malaria and tuberculosis are still high on the list of the world’s killers; plague, as we have noted, is embedded in the landscapes of four continents, still regularly producing outbreaks in Madagascar, Peru, Vietnam, Central Africa, and China; cholera still rears its head in any number of places, most recently in Haiti and Ghana. And for all of these diseases, antibiotic resistance is weakening the arsenal of defenses we have. Ebola’s “small” numbers or (currently) limited geography should not, therefore, in any way be dismissed. Rather, we should think of this as a real-time lesson in how local diseases become global.

Recent comparisons of Ebola and plague have often been accompanied by an implicit shudder of horror: “How medieval.” Yes, the practice of quarantine—so discredited when it was invoked by the Liberian government in August 2014 in its own capital of Monrovia, and implicitly by many other nations as they closed their borders to travellers from West Africa and cut off air transport—is medieval in its origins, even if practices of segregation and exclusion of suspected infectious persons are much older (Grmek 1980). Yes, the sight of bodies being piled in streets calls to mind the chilling stories of the Black Death’s devastation. When Centers for Disease Control director Thomas Frieden described the situation in West Africa in October 2014 as “something out of Dante,” he can be forgiven for apparently eliding the description of hell in the *Inferno* (which was written more than a quarter century before the arrival of the Black Death in Europe) with the hellish scenes Dante’s Italian kindred would describe several decades later. But this derogatory deployment of

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8 Sun et al. 2014: “Frieden had seen plenty of death over the years, but this was far worse than he expected, a plague on a medieval scale. ‘A scene out of Dante,’ he called it.”
“the medieval” does not get us very far. Universities are “medieval,” too, as are public health regulations, autopsies, and the licensing of medical practitioners. The presence of a highly sophisticated biomedical infrastructure in the later twentieth century did not stop the HIV/AIDS pandemic from exploding, and it has not (yet) stopped the current Ebola outbreak. The journal that gave birth to these essays, *The Medieval Globe*, was founded to ask what it means for the Middle Ages to stop being “Other” and to become knowable. With climate change, among other factors, pushing the homeostasis of our own world out of balance, the past—which is the only knowable guide we have to the future—merits revisiting.

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Bibliography


Abstract The 2014 West African Ebola epidemic shocked a world that had grown insensitive to the threat of major epidemic infectious disease outbreaks. Looking for analogies, popular media that year often invoked the Black Death as a comparison. In this preface to the book edition of Pandemic Disease in the Medieval World: Rethinking the Black Death, it is argued that while most of the extreme comparisons of the Black Death and Ebola were unhelpful, the largest pandemic in human history can be used comparatively to think through certain frameworks of global health and emerging disease analysis. All pandemics start as local outbreaks. Studying the processes by which local outbreaks (often invisible in their early phases) grow into disease events of major historical import focuses our attention on the contingent historical circumstances that can move microorganisms through vast human networks of contact and exchange.

Keywords pandemics, global health, 2014 West African Ebola epidemic, Second Plague Pandemic, HIV/AIDS, zoonoses