History Compass 11/10 (2013): 808–820, 10.1111/hic3.12081

Should We Teach That the Cause of the Black Death Was Bubonic Plague?

Phyllis Pobst*
Department of History, Arkansas State University

Abstract

For most of the 20th century, historians accepted bubonic plague, *Yersinia pestis*, as the cause of the 14th-century Black Death, although a wealth of primary sources seems to conflict with that diagnosis. Scientists focused on *Y. pestis* have been less certain than historians, arguing over their findings and how these might apply to the 14th century. Historians who believe the diagnosis was premature point to the speed with which the disease travelled, and killed; its symptoms, and its extraordinary morbidity and mortality rates; the shortage of evidence for rats or fleas; and the seasons and climates in which the disease thrived; and they question whether every inconvenient bit of medieval evidence can be covered by later hypothetical mutations. Yet, guided by textbooks, and by popular media that distort the work of scientists, teachers often omit medieval evidence that doesn't fit *Yersinia*. Uncertainty over the cause of the Black Death offers historians a chance to show how historical knowledge is generated, by realistic use of primary sources in dialogue with other scholarly disciplines.

My students are a blood-thirsty lot. They relish horrors and disasters, the disgusting and the violent. So, I imagine I have their full attention when I lecture about the "Great Mortality"[1] of the 14th century – until the questions come. "Why didn't they just kill all the rats and end it?" And, "didn't the poor suffer more from the plague because they had more fleas?" Then, I repeat what I have already said: that primary sources for the 14th century Black Death show no evidence of rats or fleas. None. To students, this is a mystery; for teaching historians it is a pedagogical problem, because there is a discontinuity between the primary sources and "common knowledge."

The Black Death can either be an excellent case study for historians, with which we teach our students the primacy of the primary sources, and show them how to handle these sources with respect and an understanding of their limits, or a stumbling-block. If we leave the subject to scientists who have little interest in our sources, and less understanding of them, we teach our students that history is a "soft" discipline: mere story-telling for those who couldn't make it in the "hard" sciences. Does historical analysis produce knowledge, or not? How textbooks and teachers handle the Black Death may reveal more to our students than we realize.

From teaching about the Black Death to undergraduates and grad students, and from the research required to keep ahead of them, I have come to believe that we historians should be more circumspect about accepting modern diagnoses of that pandemic. We have run after every new scientific pronouncement on the subject, often without knowing enough about the sciences in question to judge the completeness, soundness or relevance of the material. In fact, most teaching historians don't read the scientific evidence itself; they read articles that distort it in the popular press. My purpose here is not to recommend another diagnosis than *Yersinia pestis*, but to urge that we remain skeptics until there is more agreement among scientists and sources than we have seen so far. Primary sources don't match any modern

disease, and grave-robbing scientists don't agree among themselves. We should not teach what is still unproven as if it were a certainty.

What caused the Black Death? Fourteenth-century Europeans made it clear that the epidemic disease of the 1340s was *new to them*. Many writers sketched it in detail for their successors, often before dying of it themselves. From the 1340s on, as the disease returned to European cities at short intervals, generations of physicians wrote careful descriptions of the symptoms and prognoses of their patients.[2] There was no decade when the disease arrived without being seen by many who had seen it before and recognized it; so we have a strong chain-of-custody, as criminalists call it, establishing that this same disease existed from the 1340s into the 18th century in Europe. Then, it seemed to disappear,[3] at least from Europe.

Historians should always take the primary sources seriously and teach our students to do the same, but in this particular case, we have sometimes failed. The diagnosis of the 14th-century pandemic as bubonic plague gained a foothold in academia because early 20th-century historians trusted scientists more than they trusted their own evidence; *real* science seemed to be answering the secrets of the universe, and when medieval evidence could not be forced into the bubonic mold, it was ignored.[4]

What Everybody Knows

Students believe they see rats and fleas that are not in the sources because textbooks identify the Black Death as bubonic plague. The connection is confidently stated in many college-level textbooks[5] and thousands of web sites, often crediting Philip Ziegler for this knowledge.[6] Ziegler is a retired English diplomat whose appealing account of the 14th-century pandemic has been the touchstone for non-specialists since 1969. Not an historian, he based it on the best scholarship available in English, and his lively survey pushed most other accounts off the shelf.

Before Ziegler, the most important work in English on the subject was *The Great Pestilence* (1893) by the magisterial F. A. Gasquet,[7] who adopted the bubonic plague diagnosis when it was brand new. In the second edition,[8] Gasquet noted that, thanks to health authorities in India, much more was known about "the great epidemic of the fourteenth century, now commonly known as 'The Black Death,'...."[9] Gasquet had considerable influence, and other historians also quickly embraced this idea as fact.[10]

Ziegler's overview of the state of research in 1969 is the overwhelming favourite of the English-reading public. It accepts that modern science firmly identifies *Pasteurella pestis* (*Yersinia pestis*) as the Black Death.[11] Countless books, encyclopedia articles and web pages repeat his views.[12] The monographs that have appeared recently have been unable to bump Ziegler from his throne.[13] So for college students, this is common knowledge.

A Disagreement of Scholars

A number of respected scholars of the 14th century have challenged the identification of the Black Death as bubonic plague.[14] Since the 1980s, medievalists have looked again at their primary sources and asked whether they confirm – or even allow – a diagnosis of *Yersinia pestis*. They have not been able to agree among themselves.[15]

Scientists working on the subject also have disagreements with one another. Since the 1990s, improved techniques for isolating microbial DNA and other useful material have drawn teams of scientists into the hunt. Not surprisingly, they have confidence in laboratory tests for answering medical questions, even over many centuries.[16] But what one team of scientists asserts, another team often rejects: the heated dialogue over *Y. pestis* in tooth pulp is an obvious example.[17] In 1998, a group of molecular biologists in Marseilles claimed to have

found septicemic *Y. pestis* in six teeth from Black Death cemeteries in Provence.[18] In 2000, a team with some of the same members announced that it had found *Y. pestis* in the teeth of three skeletons from a medieval site in Montpellier.[19] A pair of scholars in the same field responded with an open letter in *Science*, sharply scolding journals that published studies "that do not observe the necessary controls."[20] In September 2003, the claim of the Marseilles team was challenged again by the report of two teams collaborating on the original site, and sites in Denmark and England,[21] who concluded that

> the absence of *Y. pestis*-specific DNA in an exhaustive search using specimens from multiple putative European Black Death burial sites does not allow us to confirm the identification of *Y. pestis* as the aetiological agent of the Black Death...."

They went on to question the utility of the Marseilles team's method.[22] A pair of anthropologists[23] pointed out that the first Marseilles report had made several dubious assumptions. First, they had assumed that the graves were from the Black Death period, although the dating was vague. Second, they had used that speculation to assume that the teeth were from Black Death victims, on the surprising assertion that there was a 90% chance that *any* Marseilles burial from a 30-year period was a victim of this disease.[24] The anthropologists also suggested that, since the Marseilles lab had used *Y. pestis* in previous experiments, there was a possibility of contamination of the tooth pulp.[25] A further contribution to the discussion also implied that the Marseilles results were contaminated and urged further testing.[26] Several of the Marseilles team have continued to work on the tooth pulp search and reported in 2007 that they had found more evidence of *Y. pestis* DNA in teeth, this time from Vienne (the 7th–9th centuries) and Martigues (the 18th century); they argued that they had now proven the usefulness of the method.[27] Interesting as this research promises to be, it's too early for historians to be certain of the results. Only the participants of this heated debate could believe that it has settled the question of what caused the Black Death of the 14th century.

Historians who don't have time to make this a priority can easily form a false impression of the state of research on the Black Death, because scientists' work is often oversimplified by the popular press. There are many examples from the past few years. America Online (AOL) announced in 2008 that the plague, that "medieval scourge," was returning. *The New York Times*, *Daily Telegraph* and others reported in 2010 that the plague (i.e. the Black Death) had been proven to come from China. The BBC confirmed this and equated the plague with the Black Death, repeating all the standard themes. In August 2011, *The Guardian* told readers that rats "weren't the carriers of the plague after all," since the spread of the Black Death was so rapid it could only have been transmitted human-to-human. Soon after, the Associated Press distributed a story from Tubingen reporting that the medieval Black Death might be extinct. *Nature* reported in October 2011 that a study of London skeletons had shown "beyond doubt that *Y. pestis* was the true cause of the Black Death." The *Nature* article was then reported in countless newspapers and online news sites, including academic sites like MEDMED-L.[28] Such reports of scientific studies appearing in popular media have convinced many non-specialists that the *Y. pestis* diagnosis is settled – and by scientists, not historians.

We expect the popular media to oversimplify and stress what is exciting, bloody and terrible; they are selling news as entertainment. But textbooks? Since historians and scientists have not yet agreed, within and among their many disciplines, should textbooks treat the old identification of the Black Death with *Y. pestis* as a scientific fact? Should students be told, in effect, to find rats and fleas in the primary sources?

What Scientists and Historians Know So Far

Increased scientific interest in the bubonic plague was stimulated by two phenomena: the AIDS epidemic and the collapse of the Soviet Union. AIDS gives scientists greatly increased urgency for understanding how 'new' diseases cross from another species, how they are transmitted and how we may control them. The collapse of the Soviet system put a lot of scientific work that had been secret out into the public domain; the West learned that we weren't alone in experimenting with weaponized forms of diseases. Funding from Western governments for research into *Yersinia pestis* jumped dramatically after 1991.

So scientists have recently published mountains of data about *Y. pestis*, most of it in English. Debates over tooth pulp aside, they have established some facts about seven important aspects of the disease. Historians should compare what they have learned with the information from witnesses in the 14th century.

TRAVEL TIME

The bubonic plague moves to new regions by slow, gradual steps, since it moves at the pace of a rodent, and they're home-loving creatures who remain within small areas.[29] Even with steamships in the 19th century and airliners in the present, the bubonic plague is a plodding disease. But the Black Death was just the opposite. Biologists James Wood, Rebecca Ferrell and Sharon DeWitte-Aviña pointed out that it "moved from place to place with extraordinary speed. . . it swept across all of Europe in less than four years."[30] In fact, no human disease has ever been shown to travel so far, so fast.[31]

The bubonic plague remains in a region for decades, even centuries, without killing off its host population or the human population. It does not disappear, but becomes endemic in host animals, possibly even in the soil.[32] But 14th-century writers said that their disease spread across Europe at the pace of human traffic, and when it had passed through an area, it was gone until the next epidemic, years later.[33]

TIME TO KILL

The bubonic plague incubation period is 2–4 days before the onset of symptoms, with an outside range of 1–6 days.[34] Because of this short period, quarantine has no value against the bubonic plague. But 14th-century witnesses said that, while the disease seemed to kill within a few days of the appearance of symptoms – even in some cases with no outward symptoms – a person could travel a long distance from an infected place without sickening, only to fall ill after being received into a new place.[35] As a result, European cities experimented with periods of isolation, and settled on a quarantine of 40 days,[36] which appeared to be more effective than a 30-day isolation period. In the 14th century, those without apparent symptoms could incubate the disease for weeks before 'presenting,' as the doctors say.

At the same time, there is a wealth of evidence that the medieval disease could kill a new victim within a day of exposure; this is not simply a medieval *topos*, it is a demographic fact.[37] How this can be reconciled with its ability to travel long distances is not clear, but both features are missing in the modern disease.

SYMPTOMS

The symptoms of bubonic plague are dominated by swollen lymphatic nodules, usually accompanied by a rapidly rising fever; they may also include muscle pain, a stiff neck, chills,

nausea, intestinal problems and malaise.[38] If the variant is pneumonic, there is coughing (sometimes of blood), but pneumonic plague is a rare secondary development from bubonic plague.[39] No one dies of *Y. pestis* without passing days of such symptoms, and there is not much variation within the symptom cluster of each form. The 14th century also reported a range of symptoms, some of which are similar and some different. There might be muscle stiffness, tingling in the skin, a splitting headache, a high fever accompanied by a cough or without it. A person might bleed from the nose or vomit blood. Many witnesses mention a scattering of blood-blisters like freckles at random across the body that were a surer sign of death than the glandular bubo.[40] A few mention pea-sized, or egg-sized, swellings near the lymph nodes.[41] The dying person might give off a terrible stench, and might lie in a stupor for several days before death. But remarkably, witnesses also say that only a few of these symptoms, or none at all, might be present before a person died. [42]

TRANSMISSION

The modern bubonic plague is almost never passed from one human to another.[43] Even in its pneumonic form, it is a disease of rodents,[44] and a human must catch it from a flea that carried it from a rodent.[45] Almost invariably it requires a human being in contact with (the right kind of) flea from a vulnerable rodent, which is in contact with a flea from a resistant rodent: so two fleas and two rodents are needed to infect a human being. While the black rat, *Rattus rattus*, may be the vector, other rodents can be involved.[46]

Medieval witnesses, on the other hand, observed that the Black Death was passed from one person to another when they were close to each other – relatives embracing, friends clasping hands, priests anointing the sick, wives nursing their husbands. "Breath spread the infection among those speaking together," wrote Michele da Piazza in 1347,[47] and while this tells us nothing of the actual mechanism of transmission, it confirms the overwhelming testimony that the disease appeared to move from human to human. Medieval witnesses do not mention insects or rodents of any kind. (Animals will be discussed further below.) Some scientists and social scientists acknowledge this awkward evidence, while others dismiss it as unreliable.[48]

COUNTING THE SICK AND DEAD

In the best documented modern outbreaks of *Y. pestis*, the morbidity rate was far less than 10%, and the mortality rate among the sick was also usually under 5% (making a very low death rate in the general population).[49] This was true *before* the invention of antibiotics.[50] A comparison with estimates of medieval morbidity and mortality rates is complicated by occasional extreme statements from traumatized witnesses; this has led some modern readers to reject them all as examples of "the notorious medieval tendency to inflate numbers."[51] But, as Rosemary Horrox has said, "if the figures are exaggerated, they are not meaningless."[52] All witnesses agree that the proportion of a city population that fell sick when the Black Death arrived was so high as to be unheard-of, and that only a few of those who showed symptoms recovered. It is now possible to supplement anecdotal evidence with numerical evidence for some places and groups.[53] Archaeologists have helped with excavations of cemeteries, confirming some extraordinary mortality in densely packed cities like Florence, in numbers that make the 14th century estimates look much closer to the truth than we used to think. An overall loss of between 35–50% of the human population in the pandemic of 1347–53 is now considered conservative. [54]

RATS AND OTHER ANIMALS

Scientists find that the bubonic plague, rather inefficiently, generally requires two fleas and two rodents to infect a human being. But Black Death sources, as mentioned, include no rats or fleas.[55] Historians must beware of dismissing this evidence by making excuses that our forebears were just really dirty and did not notice dead vermin. What iron determination would it have taken to ignore "the sudden death of thousands, even millions, of rats falling from rafters, littering buildings, streets and lanes, not only in 1348, but in numerous other plagues until the nineteenth century"[56]? Yet, no contemporary report of the Black Death mentions rodents in Europe, the Middle East, Asia or Africa. No rats, mice, voles, bats, squirrels or any other rodents appear in eye-witness accounts of the medieval disease.

The absence of medieval rats is more striking because witnesses listed many other animal *except* rodents in their narratives of the Great Mortality, including pigs, dogs, cats, domestic fowl and wild birds, horses, cattle and sheep.[57] There are many reports of other species (not rodents) dying in impressive numbers during the Black Death. This does not mean it couldn't have been bubonic plague – other species[58] can also catch *Yersinia pestis* – but it makes the absence of rodents more noticeable. Historians must confront the primary evidence in its silence. It is not sufficient to suggest that even the privileged classes of Europe were so inured to the presence of rats that they could fail to see heaps of them dead in the streets and cloisters.

SEASON AND DURATION

Y. pestis is a disease chiefly of hot places – India, Africa, the American Southwest – which is most active in those regions in the winter.[59] The Black Death appears to have made no impact in southeast Asia, sub-Saharan Africa or other regions that now report bubonic plague.[60] Instead, it was as dramatic in northern Europe as in the Mediterranean or the Middle East.[61] While it might arrive at any time, in most parts of Europe, it did its worst in the summer and faded away as the weather cooled.[62]

Witnesses of the 14th century also said that the disease, when it took hold in a town, might burn brightly for three to six months, but did not become endemic. When the bubonic plague appears to go dormant in the summer, it returns again in the cooling autumn; but the Black Death left town after some months, and did not return for years.[63]

SUMMARY

These seven points of comparison give historians reason to wait before embracing a diagnosis. The *old* disease moved as fast and as far as human beings – much faster and further than is normal for rodents. It had an incubation period that may have been weeks, even a month. Victims sometimes died without marked symptoms; most displayed a variety of symptoms that might (or might not) include bubos, fever or coughing blood. The disease appeared to pass from one person to another. It had extremely high morbidity and mortality rates and was intensely contagious. Other animals might die in large numbers in the same period, but no medieval witness (in Europe or elsewhere) mentioned rodents. The medieval disease thrived in the summer, and in cold regions. After some months, it disappeared for years. The *modern* disease differs in each of those points. The more research is done, the more this is confirmed.

Ockham's Razor

As scientists examine the molecular structures and genetic history of *Yersinia pestis*, various mutations have been discovered, and others are suspected.[64] For a disease to jump from

one species to another usually requires a mutation.[65] To change from an endemic disease of one species to an epidemic disease in another, an organism must mutate at least once, probably twice (concerning both host and transmission). For an epidemic disease to disappear requires another change. No historian who accepts evolutionary biology will argue that the Black Death was created *ex nihilo* in the 1330s and miraculously extinguished in the 17th century; mutations are indicated. But to explain the substantial differences between bubonic plague and the 14th-century epidemic, the number of mutations that must be posited is remarkable – and they must be posited chiefly so that we can hold fast to the diagnosis of *Yersinia*. This seems to ask urgently for the application of Ockham's Razor: "plurality must not be posited without necessity." William of Ockham, who died in the Black Death, frequently invoked this principle of logical reasoning, sometimes called the Law of Economy. The simplest explanation consistent with the known facts, in this case an explanation that does not require a baker's dozen of hypothetical mutations, should be preferred.

For teaching historians, the Black Death can be an opportunity to show students how archaeology, microbiology, immunology, epidemiology and scores of other disciplines contribute to our understanding of the human past. But we should not treat our own material with contempt, or we will have failed our discipline and our students. Historians are uniquely qualified to assess the value and analyse the content of medieval primary sources and should not allow the glamour of science to make us forget our own expertise. Those who become too fervently attached to a theory must ignore any witness who stubbornly refuses to support that theory, as when Ole Benedictow argues that Boccaccio's famous description of the symptoms of the Black Death must be wrong, because it does not match the data on *Y. pestis*.[66] While the testimony of medieval witnesses is sometimes emotional, historians must weigh their credibility on a fair scale. Despising evidence that doesn't agree with our theories is not the way forward, in science or in history.

This is a time for our usual healthy skepticism, as well as careful research: let's wait until scientists can agree with each other, in conclusions that can be reconciled with the primary sources, before we teach students that we know what caused the Black Death.

Acknowledgement

This article was improved by the questions and suggestions of the editors and the anonymous readers of the journal, none of whom should be assumed to agree with it.

Short Biography

Phyllis Pobst is an ecclesiastical historian of the 14th century, for whom the Black Death was a secondary consideration until she began teaching a graduate seminar on the subject in 1999. Students' zeal turned the question of what caused this epidemic into a historiographical project which defines the course (and forces her to spend hundreds of hours reviewing scientific reports each term, in order to stay current with the field). Her major work, however, is editing the episcopal registers of 14th-century Norwich, beginning with *The Register of William Bateman, Bishop of Norwich 1344–1355* in two volumes (Boydell Press for The Canterbury & York Society, 1996, 2000); the records of Anthony de Bek and William de Ayremynne are nearly ready for another Canterbury & York volume. As the founding president of The 14th Century Society, she is active in its sessions at Kalamazoo each year. She holds a BA in Religious Studies from Gonzaga University, an MTS from Harvard Divinity School, an MSL from the Pontifical Institute of Mediaeval Studies in Toronto and an MA and PhD in Medieval Studies from the University of Toronto.

Notes

* Correspondence: Department of History, Arkansas State University, State University, AR 72467, USA. Email: ppobst@astate.edu.

[1] To avoid confusion, I will not use "plague" for the 14th-century pandemic, since it is strongly associated with the bubonic plague, *Yersinia pestis*.

[2] For a survey of these manuals, see Ann G. Carmichael, "Universal and Particular: the Language of Plague, 1348–1500," , Pestilential Complexities: Understanding Medieval Plague, ed. Vivian Nutton, 2008 (London: Wellcome Trust Centre for the History of Medicine at UCL, 2008), 27–52.

[3] Most historians believe the Black Death was last noted in Europe in the early 18th century, but Lars Walløe found references that he believes were to the same disease in eastern Europe in the 19th century: "Medieval and Modern Bubonic Plague: Some Clinical Continuities," Pestilential Complexities: Understanding Medieval Plague, ed. Vivian Nutton (London: Wellcome Trust Centre for the History of Medicine at UCL, 2008): 60–63.

[4] Samuel K. Christakos et al., 2005, 2002, The Black Death Transformed: Disease and Culture in Early Renaissance Europe (New York: Oxford University Press, 2003), 2.

[5] E.g. William J. Duiker and Jackson J. Spielvogel, *The Essential World History*, 6th edn. (Boston: Wadsworth, 2011), 321; similar statements are found in most other college history textbooks.

[6] Philip Ziegler, *The Black Death* (New York: Harper & Row, 1969). References here are to the Pelican edition (Harmondsworth: Penguin, 1970).

[7] As Ziegler acknowledged (*Black Death*, 311).

[8] With a new title, The Black Death of 1348 and 1349, 2nd edn. (London: George Bell and Sons,1908).

[9] 'Preface to the Second Edition,' vi–vii; the entire preface is about this subject. In the text, he makes the point more simply: "The epidemic would appear to have been some form of the ordinary Eastern or bubonic plague" (Gasquet, 1908, *The Black Death*, 2nd edn., 8).

[10] Countless reference works cited him, such as *The New International Encyclopædia*, 17 vols. (New York: Dodd, Mead, 1902–05), vol. 3, *s.v.* "Black Death."

[11] Ziegler, *Black Death*, 25.

[12] E.g. Kristy Wilson, 2008 Bowers, "Black Death (1347–1352)," *Encyclopedia of Pestilence*, *Pandemics and Plagues*, edited by Joseph P. Byrne, 2004 (Westport, CT: GreenWood and DeWitte-Aviña, 2003, 2008); Sandra W. Moseng, 2007, 2008, "Bubonic Plague,"*ibid*.

[13] Among recent general studies are Ole J. Benedict, 1996ow, The Black Death 1346–1353: *the Complete History* (Woodbridge: BoydEll, 1979, 2004); Joseph P. Byrne, *The Black Death* (Westport, CT: Greenwood, 2004); John Kelly, 2005, The Great Mortality: an Intimate History of the Black Death, the Most Devastating Plague of All Time (New York: HarperCollins, 2005); John Hatcher, 2008, *The Black Death*: *a Personal History* (Philadelphia: Da Capo Press, 2008).

[14] Major critics of the *Yersinia pestis* diagnosis have been Graham Twigg, 1984, The Black Death: a Biological Reappraisal (London: Batsford, 1984); Samuel K. Centers for Disease Control and Prevention, 1996, 2005, 2002, "The Black Death: the End of a Paradigm," *American Historical Review* 107:3 (2002), 703–738, *The Black Death Transformed* and "Epidemiology of the Black Death"; Susan Scott and Duncan, 2005 and Christopher Duncan and Scott, 2004, *Biology of Plagues*: *Evidence from Historical Populations* (Cambridge: Cambridge University Press, 2005). For an overview of the controversy, see Lester K. Little, 2007, "Plague Historians in Lab Coats," *Past and Present* 213 (2011), 267–290.

[15] E.g. John Shrewsbury, 1970, 2007 and Frances Cate, "A Plague of Plagues: the Problem of Plague Diagnosis in Medieval England," *Journal of Interdisciplinary Studies* 37:3 (2007), 371–393; and Little, "Plague Historians," 267–290.

[16] For a more cautious view, see Andrew Cunningham, 1992, "Transforming Plague: the Laboratory and the Identity of Infectious Disease," in The Laboratory Revolution in Medicine, ed. A. Cunningham and Perry Williams (Cambridge: Cambridge University Press, 1992): 238–244.

[17] For a similar debate over the survival of disease DNA in ancient human bones, see Ingrid Welford and Bossak, 2009a, 2005 and Gisela Welford and Bossak, 2009a, 2005, "Detection of *Yersinia pestis* DNA in Two Early Medieval Skeletal Finds from Aschheim (Upper Bavaria, 6th Century A.D.)," *American Journal of Physical Anthropology* 126:1 (January 2005), 48–55; and Ian Achtman et al., 2004, 2008, 2010, 2006 and Mark G. Thomas, "Evaluating Bacterial Pathogen DNA Preservation in Museum Osteological Collections," *Proceedings of the Royal Society B*: *Biological Sciences* 273 (22 March 2006), 645–653.

[18] Michel Drancourt and Raoult, 2010 *et al.*, "Detection of 400-year-old *Yersinia pestis* DNA in Human Dental Pulp: an Approach to the Diagnosis of Ancient Septicemia," *Proceedings of the National Academy of Sciences* 95 (1998): 12637–12640; the graves were from 1590 in Lambec, and 1722 in Marseilles.

[19] The dating of the graves was speculative: "dug between the 13th and late 14th centuries" (Didier Raoult, *et al.*, "Molecular Identification by 'Suicide PCR' of *Yersinia pestis* as the Agent of Medieval Black Death," *Proceedings of the National Academy of the Sciences* 97:23 (2000): 12800–12803).

[20] Alan Cooper and Poinar, 2000 and Hendrik N. Poinar, "Ancient DNA: Do It Right or Not At All," *Science* 289 (18 August 2000): 1139. The animus in this letter is clear; note also that Poinar co-led the Smithfield cemetery DNA project reported in *Nature* in October 2011 (see note 29 below).

[21] In a paper to the British Society for General Microbiology (reported by Debora MacKenzie, "Case Reopens on Black Death Cause," *New Scientist* of 11 September 2003); one of the group, Alan Cooper, pointed out that the Marseilles team reported "a suspiciously high survival rate" for DNA in such a warm climate. The full study was published in 2004 (see next note).

[22] M. T. P. Gilbert et al., 2004, *et al.*, "Absence of *Yersinia pestis*-Specific DNA in Human Teeth from Five European Excavations of Putative Plague Victims," *Microbiology* 150 (2004): 341–354; the final version was written in September 2003.

[23] James Wood and Sharon DeWitte-Aviña, "Was the Black Death Yersinial Plague?" *The Lancet: Infectious Diseases* 3 (June 2003), 327.

[24] Amazingly, they calculated that 90% of the population of Marseilles had died of the Black Death within 30 years, so that "any remnant of this period has a 90% probability of being related to the Black Death" (Raoult, *et al.*, "Molecular Identification," 12802); Wood and DeWitte-Aviña called this "a statistical non sequitur" ("Was the Black Death," 327).

[25] Raoult called this "unsubstantiated speculation that does not contribute seriously to the debate on the origins of the Black Death" (*The Lancet: Infectious Diseases* 3 (2003): 328).

[26] Michael B. Prentice and Rahalison, 2007, *et al.*, "Was the Black Death Caused by *Yersinia pestis*?" *The Lancet: Infectious Diseases* 4 (February 2004): 72.

[27] Michel Drancourt and Raoult, 2010, *et al.*, "Yersinia pestis Orientalis in Remains of Ancient Plague Patients," *Emerging Infectious Diseases* 13:2 (February 2007): 332–333.

[28] Most examples here are from the web: Nicholas Wade, "Europe's Plagues Came From China, Study Finds," 31 Oct. 2010, accessed 3 Nov. 2010 at http://www.nytimes.com/2010/11/01/health/01plague.html ; Malcolm Moore, "Black Death may have originated in China," 1 Nov. 2010, accessed 11 Nov. 2010 at http://www.telegraph.co.uk/news/worldnews/asia/china/8102278/Black-Death-may-have-originated-in-China.html; Mike Ibeji, "Black Death: the Disease," 17 Feb. 2011, accessed 11 June 2012 at http://www.bbc.co.uk/history/british/middle_ages/blackdisease_01.shtml#three; Maev Kennedy, "Black Death study lets rats off the hook: Plague of 1348–49 spread so fast in London the carriers had to be humans not black rats, says archaeologist," 17 Aug. 2011, accessed 18 Aug. 2011 at http://www.guardian.co.uk/world/2011/aug/17; Randolph E. Schmid, "Study: Medieval Plague may be extinct," 29 Aug. 2011, accessed 30 Aug. 2011 at http://www.seattlepi.com/news/article/Study-Medieval-plague-may-be-extinct-2146079.php; see a different emphasis in http://www.geekosystem.com/researchers-discover-origin-black-death/; Edward C. Holmes, "Plague's progress," *Nature* (27 October 2011): 465 (a summary of Kirsten I. Bos, *et al.*, "A Draft Genome of *Yersinia pestis* from Victims of the Black Death, *ibid*. 478–510); MEDMED-L is a forum for scholars of medieval medicine, administered by Arizona State University.

[29] The black rat, for instance, has a home range of about 300 feet: Christopher Duncan and Susan Scott, "What Caused the Black Death?" *Postgraduate Medical Journal* 81 (2005): 317.

[30] James W. Wood *et al.*, "The Temporal Dynamics of the Fourteenth-Century Black Death: New Evidence from English Ecclesiastical Records," *Human Biology* 75: 4 (August 2003): 427–49, accessed 15 June 2012 at http://galenet.galegroup.com/servlet/HWRC/.

[31] The Black Death travelled as far as six kilometers a day: George Christakos et al., 2005, *Interdisciplinary Public Health Reasoning and Epidemic Modelling: the Case of the Black Death* (Berlin: Springer, 2005), 223, 230.

[32] Saravanan Ayyadurai, *et al.*, "Long-term Persistence of Virulent *Yersinia pestis* in Soil," *Microbiology* 154: 9 (September 2008): 2865–71.

[33] The same disease was pandemic in Europe in 1361–63, and did not return until 1369; see also Morrison *et al.*, "Epidemics," 528–535.

[34] Centers for Disease Control and Prevention, 2003 (CDC), "Imported Plague—New York City, 2002," *Morbidity and Mortality Weekly Report* 52:31 (August 8, 2003): 727; CDC, "Prevention of Plague: Recommendations of the Advisory Committee on Immunization Practices (ACIP)," *Morbidity and Mortality Weekly Report* 45: RR-14 (1996): 2.

[35] For these seven points, see the accounts of Gabriele de' Mussis, Giovanni Boccaccio, Guglielmo and Abrigeto Cortusii, John of Reading, Robert of Avesbury, Geoffrey le Baker and others, collected in *The Black Death*, edited by Rosemary Horrox, 1994 (Manchester: University of Manchester Press, 1994), Part I, 14–92; Michael Dols, 1974, "Ibn Al-Wardī's Risālah al-Naba' 'an Al-Waba': a Translation of a Major Source for the Black Death in the Middle East," *Near Eastern Numismatics, Iconography, Epigraphy and History: Studies in Honor of George C. Miles*, ed. Dickran K. Kouymjian (Beirut: American University of Beirut, 1974), 443–455; and Christos S. Bartsocas, 1966, "Two Fourteenth Century Greek Descriptions of the 'Black Death'," *Journal of the History of Medicine and Allied Sciences* 21 (1966): 394–400.

[36] Duncan and Scott calculated a 32-day incubation period in the medieval disease: "What Caused?", 315–320, and *Return of the Black Death: the World's Greatest Serial Killer* (Chichester: Wiley, 2004), 162–163; see also Paul S. Sehdev, 2002, "The Origin of Quarantine," *Clinical Infectious Diseases* 35:9 (2002): 1071–1072; G. F. Gensini et al., 2004, *et al.*, "The Concept of Quarantine in History: from Plague to SARS," *Journal of Infection* 49 (2004): 257–61.

[37] In addition to Gabriele de' Mussis, Giovanni Boccaccio, Jean de Venette, the Neuberg chronicler and other narrative sources (Horrox, *The Black Death*, 21, 28, 55, 60), see Samuel K. Christakos et al., 2007, 2002 and Guido Alfani, "Households and Plague in Early Modern Italy," *Journal of Interdisciplinary History* 38 (2007): 177–205; their data from 15th–17th century sources show an interval of zero days within households.

[38] CDC, "Prevention of Plague," 2; Susan E. Dufel and Darling, 2011 and Robert G. Darling, "Plague," *Medscape Reference* (3 June 2011), accessed 25 June 2012 at http://emedicine.medscape.com/article/829233-overview#showall.

[39] Michael B. Prentice and L. Rahalison, "Plague," *The Lancet* 369: 9568 (7 April 2007): 1196–1207; Raymond Gani and Leach, 2004 and Stephen Leach, "Epidemiologic Determinants for Modeling Pneumonic Plague Outbreaks," *Emerging Infectious Diseases* 10:4 (April 2004); accessed 25 June 2012 at http://wwwnc.cdc.gov/eid/article/10/4/03-0509_article.htm.

[40] The presence of *multiple* bloody pustules scattered across the body (not chiefly in lymph nodes) was a defining mark of the medieval disease, not only in the 14th century but also among physicians in the following centuries: Cohn, "Epidemiology of the Black Death," 89, 91.

[41] Especially in the neck or throat: Cohn, "Epidemiology of the Black Death," 90.

[42] Horrox, *The Black Death*, 14–92.

[43] Jacob L. Kendall et al., 2012, 2011l, "Risk of Person-to-Person Transmission of Pneumonic Plague," *Clinical Infectious Diseases* 40:8 (15 April 2005): 1166–1172; the best study of human-to-human *Y. pestis* transmission is of an outbreak in Nepal in 1967 that resulted in one case of pneumonic plague: Robert Raoult, 2003, 2007, "Ecology, Evolution and Epidemiology of Plague," *Plague and the End of Antiquity*: *the Pandemic of 541–750*, ed. Lester K. Little (Cambridge: Cambridge University Press, 2007): 231–289.

[44] This has been clear for a century: Wu Lien-Teh, , "First Report of the North Manchurian Plague Prevention Service," *Journal of Hygiene* 13 (1913–1914): 237–90.

[45] Occasionally another mammal may transport the fleas: see Dufel and Darling, "Plague," http://emedicine.medscape.com/article/829233-overview#showall; and M. L. Gupta and Sharma, 2007 and A. Sharma, "Pneumonic Plague, Northern India, 2002" [letter], *Emerging Infectious Diseases* 13:4 (April 2007), accessed 25 June 2012 at http://wwwnc.cdc.gov/eid/article/13/4/05-1105.htm.

[46] Dufel and Darling, "Plague," http://emedicine.medscape.com/article/829233-overview#showall.

[47] Michele da Piazza, in Horrox, *The Black Death*, 36.

[48] See for example the heated exchange over body lice as a possible vector: Saravanan Ayyadurai *et al.*, "Body Lice, *Yersinia pestis* Orientalis, and Black Death," *Emerging Infectious Diseases* 16:5 (May 2010), 892; Mark R. Walløe, 2008, 2009a and Brian H. Bossak, letter to the editor re. "Body Lice, *Yersinia pestis* Orientalis, and Black Death," *Emerging Infectious Diseases* 16:10 (October 2010): 1649; M. Drancourt and D. Raoult, "In Response," *ibid*., 1650–51.

[49] Many writers say that the pneumonic form of *Y. pestis* is far more dangerous than the bubonic form, but in the most-deadly modern outbreak of pneumonic plague, in 1911 Manchuria, fewer than 0.3% of the population was affected: Mark Gamsa, 2006, "The Epidemic of Pneumonic Plague in Manchuria, 1910–1911," *Past and Present* 190 (2006): 147–184. See also Dufel and Darling, "Plague," at http://emedicine.medscape.com/article/829233-overview#showall.

[50] For antibiotics and *Y. pestis*, see T. V. Inglesby et al., 2000, *et al.*, "Plague as a Biological Weapon: Medical and Public Health Management," *Journal of the American Medical Association*, 283 (2000): 2286.

[51] Horrox, "Introduction," *The Black Death*, 3.

[52] *Ibid*.

[53] See James W. Wood *et al.*, "The Temporal Dynamics of the Fourteenth-Century Black Death: New Evidence from English Ecclesiastical Records," *Human Biology* 75 (2003): 427 ff.; Richard W. Emery, 1967, "The Black Death of 1348 in Perpignan," *Speculum* 42 (1967): 611–623; Ann G. Byrne, 2004, 2011, 1978, "Epidemic Diseases in Early Renaissance Florence," (Ph.D. dissertation, Duke University, 1978); Barbara E. Megson, 1998, "Mortality Among London Citizens in the Black Death," *Medieval Prosopography* 19 (1998): 125–133.

[54] Callaway, 2011, 1978, "Epidemic Diseases," 50: "over 50,000 died within the city that year [1348], out of a population already pruned back to just under 100,000."

[55] For a discussion of whether black rats were rare in Europe, see David E. Davis, 1986, "The Scarcity of Rats and the Black Death: an Ecological History," *Journal of Interdisciplinary History* 16:3 (1986): 455–470; G. Karisson, 1996, "Plague without Rats: the Case of Fifteenth-Century Iceland," *Journal of Medieval History* 22:3 (1996): 263–284; and Michael McCormick, 2003, "Rats, Communications, and Plague: Toward an Ecological History," *Journal of Interdisciplinary History* 34:1 (2003): 1–25.

[56] Cohn, "Epidemiology of the Black Death," 76, n.11, and 78.

[57] E.g. Boccaccio, Ralph Higden, Thomas Walsingham and Henry Knighton (Horrox, *The Black Death*, 28, 63, 66, 77, 88); and Bartsocas, "Two Fourteenth Century," 395; and Cohn, *The Black Death Transformed*, 131–134.

[58] See note 45 above.

[59] Christakos *et al.*, "Recent Results," 700–720; Plague Research Commission, 1907 Commission, "The Epidemiological Observations Made by the Commission in Bombay City," *The Journal of Hygiene* 7 (1907): 724–786.

[60] Carmichael, "Universal and Particular," 29.
[61] Karisson, "Plague Without Rats," 263–284; P. Oeding, 1989, "Plague Epidemics in Bergen and Population Crises," *Tidsskrift for den Norske Laegeforening* 109 (December 10, 1989): 3569–73; Oeding, "The Black Death in Norway," *Tidsskrift for den Norske Laegeforening* 110 (June 30, 1990): 2204–08; Ole Georg Morrison et al., 1985, "The Complex Plague—Reconsiderations of an Epidemic from the Past," *Tidsskrift for den Norske Laegeforening* 127 (December 13, 2007): 3272–75.
[62] Cohn graphed this seasonal pattern in "The Black Death: End of a Paradigm," 703–738. See also Morrison *et al.*, "Epidemics," 532–533; Mark R. Welford and Brian H. Bossak, "Validation of Inverse Seasonal Peak Mortality in Medieval Plagues, Including the Black Death, in Comparison to Modern Yersinia pestis-variant Diseases," *Public Library of Science ONE* 4(12) (2009): e8401; Welford and Bossak, "Did Medieval Trade Activity and a Viral Etiology Control the Spatial Extent and Seasonal Distribution of Black Death Mortality?" *Medical Hypotheses* 72: 6 (2009): 749–52; Carmichael, "Epidemic Diseases," 53–74; Wood *et al.*, "Temporal Dynamics," 427–49 (http://galenet.galegroup.com/servlet/HWRC/); Twigg, *Black Death*, 171–186; Cohn, *Black Death Transformed*, 140–187; J. F. D. Shrewsbury, *A History of the Bubonic Plague in the British Isles* (Cambridge: Cambridge University Press, 1970), 123–125.
[63] Morrison *et al.*, "Epidemics," 528–535.
[64] See, for instance, Mark Achtman *et al.*, "Microevolution and History of the Plague Bacillus, *Yersinia pestis*," *Proceedings of the National Academy of Sciences* 101: 51 (December 21, 2004): 17837–17842; Jovanka T. Koo *et al.*, "Global Discovery of Small RNAs in *Yersinia pseudotuberculosis* Identifies *Yersinia*-specific Small, Noncoding RNAs Required for Virulence," *Proceedings of the National Academy of Sciences* 108: 37 (published online ahead of print, August 29, 2011): E709-717.
[65] William H. McNeill, 1976, *Plagues and Peoples* (New York: Doubleday, 1976), discusses this in chapter 2, and identifies a number of contagious diseases that passed from animals, usually domesticated, to humans.
[66] In *The Black Death 1346–1353*, *op.cit.*, 26.

Bibliography

(This is not an exhaustive list, as it includes only works cited, but it may help those new to the controversies begin their own research.)

Achtman, Mark *et al*. "Microevolution and History of the Plague Bacillus, *Yersinia pestis*." *Proceedings of the National Academy of Sciences* 101: 51 (December 21, 2004): 17837–17842.

Ayyadurai, Saravanan, Florent Sebbane, Didier Raoult, and Michel Drancourt. "Body Lice, *Yersinia pestis* Orientalis, and Black Death." *Emerging Infectious Diseases* 16:5 (May 2010): 892.

Ayyadurai, Saravanan, Linda Houhamdi, Hubert Lepidi, Claude Nappez, Didier Raoult and Michel Drancourt. "Long-term Persistence of Virulent *Yersinia pestis* in Soil." *Microbiology* 154: 9 (September 2008): 2865–71.

Barnes, Ian and Mark G. Thomas. "Evaluating Bacterial Pathogen DNA Preservation in Museum Osteological Collections." *Proceedings of the Royal Society B: Biological Sciences* 273 (22 March 2006): 645–653.

Bartsocas, Christos S. "Two Fourteenth Century Greek Descriptions of the 'Black Death'." *Journal of the History of Medicine and Allied Sciences* 21 (1966): 394–400.

Benedict, Carol. *Bubonic Plague in Nineteenth-Century China*. Palo Alto: Stanford University Press, 1996.

Benedictow, Ole J. *The Black Death 1346–1353: the Complete History*. Woodbridge: Boydell, 2004.

Bossak, Brian H., and Mark R. Welford. "Did Medieval Trade Activity and a Viral Etiology Control the Spatial Extent and Seasonal Distribution of Black Death Mortality?" *Medical Hypotheses* 72: 6 (2009): 749–52.

Bowers, Kristy Wilson. "Black Death (1347–1352)." *Encyclopedia of Pestilence, Pandemics and Plagues*. Edited by Joseph P. Byrne. Westport, CT: Greenwood, 2008.

Byrne, Joseph P. *The Black Death*. Westport, CT: Greenwood, 2004.

Callaway, Ewen. "Plague Genome: the Black Death Decoded." *Nature* 478 (2011), 444–446. Published October 25, 2011, corrected October 28, 2011: http://www.nature.com/news/2011/111025/full/478444a.html#B2

Carmichael, Ann G. "Epidemic Diseases in Early Renaissance Florence." Ph.D. dissertation, Duke University, 1978.

Carmichael, Ann G. "Universal and Particular: the Language of Plague, 1348–1500." *Pestilential Complexities: Understanding Medieval Plague*. Edited by Vivian Nutton. Medical History, Supplement 27. London: Wellcome Trust Centre for the History of Medicine at UCL, 2008: 17–52.

Centers for Disease Control and Prevention. "Imported Plague—New York City, 2002." *Morbidity and Mortality Weekly Report* 52:31 (August 8, 2003): 725–728.

Centers for Disease Control and Prevention. "Prevention of Plague: Recommendations of the Advisory Committee on Immunization Practices (ACIP)." *Morbidity and Mortality Weekly Report* 45: RR-14 (1996): 1–15.

Christakos, George, Ricardo A. Olea, Marc L. Serre, Hwa-Lung Yu, and Lin-Lin Wang. *Interdisciplinary Public Health Reasoning and Epidemic Modelling: the Case of the Black Death*. Berlin: Springer, 2005.

Christakos, George, Ricardo A. Olea, and Hwa-Lung Yu. "Recent Results on the Spatiotemporal Modelling and Comparative Analysis of Black Death and Bubonic Plague Epidemics." *Public Health* 121:9 (September 2007): 700–720.

Cohn, Samuel K. "The Black Death: End of a Paradigm." *American Historical Review* 107:3 (2002): 703–738.

Cohn, Samuel K. *The Black Death Transformed: Disease and Culture in Early Renaissance Europe*. New York: Oxford University Press, 2003.

Cohn, Samuel K. "Epidemiology of the Black Death and Successive Waves of Plague." *Pestilential Complexities: Understanding Medieval Plague*. Edited by Vivian Nutton. Medical History, Supplement 27. London: Wellcome Trust Centre for the History of Medicine at UCL, 2008: 74–100.

Cohn, Samuel K. and Guido Alfani. "Households and Plague in Early Modern Italy." *Journal of Interdisciplinary History* 38 (2007): 177–205.

Cooper, Alan and Hendrik N. Poinar. "Ancient DNA: Do It Right or Not At All." *Science* 289 (18 August 2000): 1139.

Cunningham, Andrew. "Transforming Plague: the Laboratory and the Identity of Infectious Disease." In *The Laboratory Revolution in Medicine*, ed. Andrew Cunningham and Perry Williams. Cambridge: Cambridge University Press, 1992: 238–244.

Davis, David E. "The Scarcity of Rats and the Black Death: an Ecological History." *Journal of Interdisciplinary History* 16:3 (1986): 455–470.

Dols, Michael. "Ibn Al-Wardī's Risālah al-Naba' 'an Al-Waba': a Translation of a Major Source for the Black Death in the Middle East." *Near Eastern Numismatics, Iconography, Epigraphy and History: Studies in Honor of George C. Miles*. Ed. Dickran K. Kouymjian. Beirut: American University of Beirut, 1974): 443–455.

Drancourt, Michel and Didier Raoult. "In Response." *Emerging Infectious Diseases* 16:10 (October 2010): 1650.

Drancourt, Michel, Gérard Aboudharam, Michel Signoli, Olivier Dutour and Didier Raoult. "Detection of 400-year-old *Yersinia pestis* DNA in Human Dental Pulp: an Approach to the Diagnosis of Ancient Septicemia." *Proceedings of the National Academy of Sciences* 95 (1998): 12637–12640.

Drancourt, Michel, Michel Signoli, La Vu Dang, Bruno Bizot, Véronique Roux, Stéfan Tzortzis and Didier Raoult. "Yersinia pestis Orientalis in Remains of Ancient Plague Patients." *Emerging Infectious Diseases* 13:2 (February 2007): 332–333.

Dufel, Susan E. and Robert G. Darling. "Plague." *Medscape Reference*: *Drugs*, *Diseases* & *Procedures* (June 3, 2011), accessed June 26, 2012 at http://emedicine.medscape.com/article/829233-overview#showall.

Duncan, Christopher and Susan Scott. "What Caused the Black Death?" *Postgraduate Medical Journal* 81 (2005): 315–320.

Duncan, Christopher and Susan Scott. *Return of the Black Death: the World's Greatest Serial Killer*. Chichester: Wiley, 2004.

Ell, Stephen R. "Some Evidence for Interhuman Transmission of Medieval Plague." *Reviews of Infectious Diseases* 1: 3 (1979): 563–566.

Emery, Richard W. "The Black Death of 1348 in Perpignan." *Speculum* 42 (1967): 611–623

Encyclopedia of Pestilence, Pandemics and Plagues. Edited by Joseph P. Byrne. Westport, CT: Greenwood, 2008.

Gamsa, Mark. "The Epidemic of Pneumonic Plague in Manchuria, 1910–1911." *Past and Present* 190 (2006): 147–184.

Gani, Raymond and Stephen Leach. "Epidemiologic Determinants for Modeling Pneumonic Plague Outbreaks." *Emerging Infectious Diseases* 10:4 (April 2004). Serial online, accessed June 26, 2012 at http://wwwnc.cdc.gov/eid/article/10/4/03-0509_article.htm

Gasquet, F. A. *The Black Death of 1348 and 1349*. 2nd edn. London: George Bell and Sons, 1908.

Gensini, Gian Franco, Magdi H. Yacoub, and Andrea A. Conti. "The Concept of Quarantine in History: from Plague to SARS." *Journal of Infection* 49 (2004): 257–61.

Gilbert, M., Thomas P., Jon Cucculi, William White, Niels Lynnerup, Richard W. Titball, Alan Cooper and Michael B. Prentice. "Absence of *Yersinia pestis*-Specific DNA in Human Teeth from Five European Excavations of Putative Plague Victims." *Microbiology* 150 (2004): 341–354.

Gupta, M. L., and A. Sharma. "Pneumonic Plague, Northern India, 2002" [letter]. *Emerging Infectious Diseases* 13:4 (April 2007). Online serial accessed June 26, 2012 at http://wwwnc.cdc.gov/eid/article/13/4/05-1105.htm

Hatcher, John. *The Black Death: a Personal History*. Philadelphia: Da Capo Press, 2008.

Horrox, Rosemary. *The Black Death*. Manchester: University of Manchester Press, 1994.

Inglesby, T. V., *et al.* "Plague as a Biological Weapon: Medical and Public Health Management." *Journal of the American Medical Association*, 283 (2000): 2281–90.

Karisson, G. "Plague Without Rats: the Case of Fifteenth-Century Iceland." *Journal of Medieval History* 22:3 (1996): 263–284.

Kelly, John. *The Great Mortality: an Intimate History of the Black Death, the Most Devastating Plague of All Time*. New York: HarperCollins, 2005.

Kendall, E. J., J. Montgomery, J. A. Evans, C. Stantis, and V. Mueller. "Mobility, Mortality, and the Middle Ages: Identification of Migrant Individuals in a 14th Century Black Death Cemetery Population." *American Journal of Physical Anthropology*, online December 7, 2012. DOI: 10.1002/ajpa.22194.

Koo, Jovanka T., Trevis M. Alleyne, Chelsea A. Schiano, Nadereh Jafari and Wyndham W. Lathem. "Global Discovery of Small RNAs in *Yersinia pseudotuberculosis* Identifies *Yersinia*-specific Small, Noncoding RNAs Required for Virulence." *Proceedings of the National Academy of Sciences* 108: 37 (published online ahead of print, August 29, 2011): E709-717.

Kool, Jacob L. "Risk of Person-to-Person Transmission of Pneumonic Plague." *Clinical Infectious Diseases* 40:8 (15 April 2005), 1166–1172.
Little, Lester K., ed. *Plague and the End of Antiquity: the Pandemic of 541–750*. Cambridge: Cambridge University Press, 2007.
Little, Lester K. "Plague Historians in Lab Coats." *Past and Present* 213:1 (2011): 267–290.
McCormick, Michael. "Rats, Communications, and Plague: Toward an Ecological History." *Journal of Interdisciplinary History* 34:1 (2003): 1–25.
McNeill, William H. *Plagues and Peoples*. New York: Doubleday, 1976.
Megson, Barbara E. "Mortality Among London Citizens in the Black Death." *Medieval Prosopography* 19 (1998): 125–133.
Morrison, A. S., J. Kirshner and A. Molho. "Epidemics in Renaissance Florence." *American Journal of Public Health* 75: 5 (1985): 528–535.
Moseng, Ole Georg. "The Complex Plague—Reconsiderations of an Epidemic from the Past." *Tidsskrift for den Norske Laegeforening* 127 (December 13, 2007): 3272–75.
Moss, Sandra W. "Bubonic Plague." *Encyclopedia of Pestilence, Pandemics and Plagues*. Edited by Joseph P. Byrne. Westport, CT: Greenwood, 2008.
Nutton, Vivian. "Introduction." *Pestilential Complexities: Understanding Medieval Plague*. Edited by Vivian Nutton. Medical History, Supplement 27. London: Wellcome Trust Centre for the History of Medicine at UCL, 2008.
Oeding, P. "Plague Epidemics in Bergen and Population Crises." *Tidsskrift for den Norske Laegeforening* 109 (December 10, 1989): 3569–73.
Oeding, P. "The Black Death in Norway." *Tidsskrift for den Norske Laegeforening* 110 (June 30, 1990): 2204–08.
Plague Research Commission. "The Epidemiological Observations Made by the Commission in Bombay City." *The Journal of Hygiene* 7 (1907): 724–786, plus maps.
Prentice, Michael B. and L. Rahalison. "Plague." *The Lancet* 369: 9568 (April 7, 2007): 1196–1207.
Prentice, Michael B., M. Thomas P. Gilbert and Alan Cooper. "Was the Black Death Caused by *Yersinia pestis*?" *The Lancet: Infectious Diseases* 4 (February 2004): 72.
Raoult, Didier. Author's reply to "Was the Black Death Yersinial Plague?" *The Lancet: Infectious Diseases* 3 (2003): 328.
Raoult, Didier, Gérard Aboudharam, Eric Crubézy, Georges Larrouy, Bertrand Ludes and Michel Drancourt. "Molecular Identification by 'Suicide PCR' of *Yersinia pestis* as the Agent of Medieval Black Death." *Proceedings of the National Academy of the Sciences* 97:23 (2000): 12800–12803.
Sallares, Robert. "Ecology, Evolution and Epidemiology of Plague." *Plague and the End of Antiquity: the Pandemic of 541–750*. Edited by Lester K. Little. Cambridge: Cambridge University Press, 2007: 231–289.
Scott, Susan and Christopher Duncan. *Biology of Plagues: Evidence from Historical Populations*. Cambridge: Cambridge University Press, 2005.
Sehdev, Paul S. "The Origin of Quarantine." *Clinical Infectious Diseases* 35:9 (2002), 1071–1072.
Shrewsbury, J. F. D. *A History of the Bubonic Plague in the British Isles*. Cambridge: Cambridge University Press, 1970.
Theilmann, John and Frances Cate. "A Plague of Plagues: the Problem of Plague Diagnosis in Medieval England." *Journal of Interdisciplinary Studies* 37:3 (2007): 371–393.
Twigg, Graham. *The Black Death: a Biological Reappraisal*. London: Batsford, 1984.
Walløe, Lars. "Medieval and Modern Bubonic Plague: Some Clinical Continuities." *Pestilential Complexities: Understanding Medieval Plague*. Edited by Vivian Nutton. Medical History, Supplement 27. London: Wellcome Trust Centre for the History of Medicine at UCL, 2008: 59–73.
Welford, Mark R. and Brian H. Bossak. "Did Medieval Trade Activity and a Viral Etiology Control the Spatial Extent and Seasonal Distribution of Black Death Mortality?" *Medical Hypotheses* 72: 6 (2009a): 749–752.
Welford, Mark R. and Brian H. Bossak. Letter in response to "Body Lice, *Yersinia pestis* Orientalis, and Black Death." *Emerging Infectious Diseases* 16:10 (October 2010): 1649.
Welford, Mark R. and Brian H. Bossak. "Validation of Inverse Seasonal Peak Mortality in Medieval Plagues, Including the Black Death, in Comparison to Modern Yersinia pestis-variant Diseases." *Public Library of Science ONE* 4(12) (2009b): e8401.
Wiechmann, Ingrid and Gisela Grupe. "Detection of *Yersinia pestis* DNA in Two Early Medieval Skeletal Finds from Aschheim (Upper Bavaria, 6th Century A.D.)." *American Journal of Physical Anthropology* 126:1 (January 2005): 48–55.
Wood, James W., Rebecca J. Ferrell, and Sharon N. DeWitte-Aviña. "The Temporal Dynamics of the Fourteenth-Century Black Death: New Evidence from English Ecclesiastical Records." *Human Biology* 75: 4 (August 2003): 427–49. Accessed at http://galenet.galegroup.com/servlet/HWRC/.
Wood, James and Sharon DeWitte-Aviña. "Was the Black Death Yersinial Plague?" *The Lancet: Infectious Diseases* 3 (June 2003): 72.
Wu Lien-Teh. "First Report of the North Manchurian Plague Prevention Service." *Journal of Hygiene* 13 (1913–1914): 237–90.
Ziegler, Philip. *The Black Death*. New York: Harper & Row, 1969.

FORDHAM UNIVERSITY

Search

IHSP

Main
Ancient
Medieval
Modern

Subsidiary Sourcebooks
African
Eastern Asian
Global
Indian
Jewish
Islamic
Lesbian/Gay
Science
Women

Special Resources
Byzantium
Medieval Music

Medieval Sourcebook: Boccaccio: The Decameron - Introduction

The onset of the Black Death, was described by Giovanni Boccaccio (1313-1375).

I say, then, that the years of the beatific incarnation of the Son of God had reached the tale of one thousand three hundred and forty eight, when in the illustrious city of Florence, the fairest of all the cities of Italy, there made its appearance that deadly pestilence, which, whether disseminated by the influence of the celestial bodies, or sent upon us mortals by God in His just wrath by way of retribution for our iniquities, had had its origin some years before in the East, whence, after destroying an innumerable multitude of living beings, it had propagated itself without respite from place to place, and so calamitously, had spread into the West.

In Florence, despite all that human wisdom and forethought could devise to avert it, as the cleansing of the city from many impurities by officials appointed for the purpose, the refusal of entrance to all sick folk, and the adoption of many precautions for the preservation of health; despite also humble supplications addressed to God, and often repeated both in public procession and otherwise by the devout; towards the beginning of the

Saints' Lives

Ancient Law
Medieval Law

Film: Ancient
Film: Medieval
Film: Modern

About IHSP
IHSP Credits

spring of the said year the doleful effects of the pestilence began to be horribly apparent by symptoms that shewed as if miraculous.

Not such were they as in the East, where an issue of blood from the nose was a manifest sign of inevitable death; but in men a women alike it first betrayed itself by the emergence of certain tumors in the groin or the armpits, some of which grew as large as a common apple, others as an egg, some more, some less, which the common folk called *gavoccioli*. From the two said parts of the body this deadly gavocciolo soon began to propagate and spread itself in all directions indifferently; after which the form of the malady began to change, black spots or livid making their appearance in many cases on the arm or the thigh or elsewhere, now few and large, then minute and numerous. And as the gavocciolo had been and still were an infallible token of approaching death, such also were these spots on whomsoever they shewed themselves. Which maladies seemed set entirely at naught both the art of the physician and the virtue of physic; indeed, whether it was that the disorder was of a nature to defy such treatment, or that the physicians were at fault - besides the qualified there was now a multitude both of men and of women who practiced without having received the slightest tincture of medical science - and, being in ignorance of its source, failed to apply the proper remedies; in either case, not merely were those that covered few, but almost all within three days from the appearance of the said symptoms, sooner or later, died, and in most cases without any fever or other attendant malady.

Moreover, the virulence of the pest was the greater by reason the intercourse was apt to convey it from the sick to the whole, just as fire devours things dry or greasy when they are brought close to it, the evil went yet further, for not merely by speech or association with the sick was the malady communicated to the healthy with consequent peril of common death; but any that touched the clothes the sick or aught else that had been touched, or used by these seemed thereby to contract the disease.

So marvelous sounds that which I have now to relate, that, had not many, and I among them, observed it with their own eyes, I had hardly dared to credit it, much less to set it down in writing, though I had had it from the lips of a credible witness.

I say, then, that such was the energy of the contagion of the said pestilence, that it was not merely propagated from man to mail, but, what is much more startling, it was frequently observed, that things which had belonged to one sick or dead of the disease, if touched by some other living creature, not of the human species, were the occasion, not merely of sickening, but of an almost instantaneous death. Whereof my own eyes

(as I said a little before) had cognisance, one day among others, by the following experience. The rags of a poor man who had died of the disease being strewn about the open street, two hogs came thither, and after, as is their wont, no little trifling with their snouts, took the rags between their teeth and tossed them to and fro about their chaps; whereupon, almost immediately, they gave a few turns, and fell down dead, as if by poison, upon the rags which in an evil hour they had disturbed.

In which circumstances, not to speak of many others of a similar or even graver complexion, divers apprehensions and imaginations were engendered in the minds of such as were left alive, inclining almost all of them to the same harsh resolution, to wit, to shun and abhor all contact with the sick and all that belonged to them, thinking thereby to make each his own health secure. Among whom there were those who thought that to live temperately and avoid all excess would count for much as a preservative against seizures of this kind. Wherefore they banded together, and dissociating themselves from all others, formed communities in houses where there were no sick, and lived a separate and secluded life, which they regulated with the utmost care, avoiding every kind of luxury, but eating and drinking moderately of the most delicate viands and the finest wines, holding converse with none but one another, lest tidings of sickness or death should reach them, and diverting their minds with music and such other delights as they could devise. Others, the bias of whose minds was in the opposite direction, maintained, that to drink freely, frequent places of public resort, and take their pleasure with song and revel, sparing to satisfy no appetite, and to laugh and mock at no event, was the sovereign remedy for so great an evil: and that which they affirmed they also put in practice, so far as they were able, resorting day and night, now to this tavern, now to that, drinking with an entire disregard of rule or measure, and by preference making the houses of others, as it were, their inns, if they but saw in them aught that was particularly to their taste or liking; which they, were readily able to do, because the owners, seeing death imminent, had become as reckless of their property as of their lives; so that most of the houses were open to all comers, and no distinction was observed between the stranger who presented himself and the rightful lord. Thus, adhering ever to their inhuman determination to shun the sick, as far as possible, they ordered their life. In this extremity of our city's suffering and tribulation the venerable authority of laws, human and divine, was abased and all but totally dissolved for lack of those who should have administered and enforced them, most of whom, like the rest of the citizens, were either dead or sick or so hard bested for servants that they were unable to execute any office; whereby every man was free to do what was right in his own eyes.

Not a few there were who belonged to neither of the two said parties, but kept a middle course between them, neither laying t same restraint upon their diet as the former, nor allowing themselves the same license in drinking and other dissipations as the latter, but living with a degree of freedom sufficient to satisfy their appetite and not as recluses. They therefore walked abroad, carrying in the hands flowers or fragrant herbs or divers sorts of spices, which they frequently raised to their noses, deeming it an excellent thing thus to comfort the brain with such perfumes, because the air seemed be everywhere laden and reeking with the stench emitted by the dead and the dying, and the odours of drugs.

Some again, the most sound, perhaps, in judgment, as they were also the most harsh in temper, of all, affirmed that there was no medicine for the disease superior or equal in efficacv to flight; following which prescription a multitude of men and women, negligent of all but themselves, deserted their city, their houses, their estates, their kinsfolk, their goods, and went into voluntary exile, or migrated to the country parts, as if God in visiting men with this pestilence in requital of their iniquities would not pursue them with His wrath wherever they might be, but intended the destruction of such alone as remained within the circuit of the walls of the city; or deeming perchance, that it was now time for all to flee from it, and that its last hour was come.

Of the adherents of these divers opinions not all died, neither did all escape; but rather there were, of each sort and in every place many that sickened, and by those who retained their health were treated after the example which they themselves, while whole, had set, being everywhere left to languish in almost total neglect. Tedious were it to recount, how citizen avoided citizen, how among neighbors was scarce found any that shewed fellow-feeling for another, how kinsfolk held aloof, and never met, or but rarely; enough that this sore affliction entered so deep into the minds of men a women, that in the horror thereof brother was forsaken by brother nephew by uncle, brother by sister, and oftentimes husband by wife: nay, what is more, and scarcely to be believed, fathers and mothers were found to abandon their own children, untended, unvisited, to their fate, as if they had been strangers. Wherefore the sick of both sexes, whose number could not be estimated, were left without resource but in the charity of friends (and few such there were), or the interest of servants, who were hardly to be had at high rates and on unseemly terms, and being, moreover, one and all, men and women of gross understanding, and for the most part unused to such offices, concerned themselves no further than to supply the immediate and expressed wants of the sick, and to watch them die; in which service they themselves not seldom perished with their gains. In consequence of which dearth of

servants and dereliction of the sick by neighbors, kinsfolk and friends, it came to pass-a thing, perhaps, never before heard of-that no woman, however dainty, fair or well-born she might be, shrank, when stricken with the disease, from the ministrations of a man, no matter whether he were young or no, or scrupled to expose to him every part of her body, with no more shame than if he had been a woman, submitting of necessity to that which her malady required; wherefrom, perchance, there resulted in after time some loss of modesty in such as recovered. Besides which many succumbed, who with proper attendance, would, perhaps, have escaped death; so that, what with the virulence of the plague and the lack of due attendance of the sick, the multitude of the deaths, that daily and nightly took place in the city, was such that those who heard the tale-not to say witnessed the fact-were struck dumb with amazement. Whereby, practices contrary to the former habits of the citizens could hardly fail to grow up among the survivors.

It had been, as to-day it still is, the custom for the women that were neighbors and of kin to the deceased to gather in his house with the women that were most closely connected with him, to wail with them in common, while on the other hand his male kinsfolk and neighbors, with not a few of the other citizens, and a due proportion of the clergy according to his quality, assembled without, in front of the house, to receive the corpse; and so the dead man was borne on the shoulders of his peers, with funeral pomp of taper and dirge, to the church selected by him before his death. Which rites, as the pestilence waxed in fury, were either in whole or in great part disused, and gave way to others of a novel order. For not only did no crowd of women surround the bed of the dying, but many passed from this life unregarded, and few indeed were they to whom were accorded the lamentations and bitter tears of sorrowing relations; nay, for the most part, their place was taken by the laugh, the jest, the festal gathering; observances which the women, domestic piety in large measure set aside, had adopted with very great advantage to their health. Few also there were whose bodies were attended to the church by more than ten or twelve of their neighbors, and those not the honorable and respected citizens; but a sort of corpse-carriers drawn from the baser ranks, who called themselves

becchini and performed such offices for hire, would shoulder the bier, and with hurried steps carry it, not to the church of the dead man's choice, but to that which was nearest at hand, with four or six priests in front and a candle or two, or, perhaps, none; nor did the priests distress themselves with too long and solemn an office, but with the aid of the becchini hastily consigned the corpse to the first tomb which they found untenanted. The condition of the lower, and, perhaps, in great measure of the middle ranks,

of the people shewed even worse and more deplorable; for, deluded by hope or constrained by poverty, they stayed in their quarters, in their houses where they sickened by thousands a day, and, being without service or help of any kind, were, so to speak, irredeemably devoted to the death which overtook them. Many died daily or nightly in the public streets; of many others, who died at home, the departure was hardly observed by their neighbors, until the stench of their putrefying bodies carried the tidings; and what with their corpses and the corpses of others who died on every hand the whole place was a sepulchre.

It was the common practice of most of the neighbors, moved no less by fear of contamination by the putrefying bodies than by charity towards the deceased, to drag the corpses out of the houses with their own hands, aided, perhaps, by a porter, if a porter was to be had, and to lay them in front of the doors, where any one who made the round might have seen, especially in the morning, more of them than he could count; afterwards they would have biers brought up or in default, planks, whereon they laid them. Nor was it once twice only that one and the same bier carried two or three corpses at once; but quite a considerable number of such cases occurred, one bier sufficing for husband and wife, two or three brothers, father and son, and so forth. And times without number it happened, that as two priests, bearing the cross, were on their way to perform the last office for some one, three or four biers were brought up by the porters in rear of them, so that, whereas the priests supposed that they had but one corpse to bury, they discovered that there were six or eight, or sometimes more. Nor, for all their number, were their obsequies honored by either tears or lights or crowds of mourners rather, it was come to this, that a dead man was then of no more account than a dead goat would be to-day.

From Boccaccio,

TheDecameron,. M. Rigg, trans. (London: David Campbell, 1921), Vol. 1, pp. 5-11

New Hypothesis for Cause of Epidemic among Native Americans, New England, 1616–1619

John S. Marr and John T. Cathey

In the years before English settlers established the Plymouth colony (1616–1619), most Native Americans living on the southeastern coast of present-day Massachusetts died from a mysterious disease. Classic explanations have included yellow fever, smallpox, and plague. Chickenpox and trichinosis are among more recent proposals. We suggest an additional candidate: leptospirosis complicated by Weil syndrome. Rodent reservoirs from European ships infected indigenous reservoirs and contaminated land and fresh water. Local ecology and high-risk quotidian practices of the native population favored exposure and were not shared by Europeans. Reduction of the population may have been incremental, episodic, and continuous; local customs continuously exposed this population to hyperendemic leptospiral infection over months or years, and only a fraction survived. Previous proposals do not adequately account for signature signs (epistaxis, jaundice) and do not consider customs that may have been instrumental to the near annihilation of Native Americans, which facilitated successful colonization of the Massachusetts Bay area.

Retrospective studies have inherent, sometimes insurmountable, biases, but speculation on past events by historians and anthropologists is commonplace and offers grist for future studies. We offer an alternative hypothesis for the cause of an epidemic among Native Americans in the years immediately before the arrival of the Pilgrims in Massachusetts. During 1616–1619, many persons died of a disease that presumably spared nearby European fishermen and traders (*1*). The more severe manifestations were fever, headache, epistaxis, jaundice, and skin lesions. Speculations as to the cause have included plague, yellow fever, and smallpox (*2–7*), as well as influenza, chickenpox, typhus, typhoid fever, trichinosis, cerebrospinal meningitis, and syndemic infection of hepatitis B virus (HBV) and hepatitis D virus (HDV) (Table 1) (*6–11*). We propose another disease: leptospirosis, accompanied by Weil syndrome. With its more severe manifestations, this syndrome is consistent with available clinical information, the nidality of *Leptospira* organisms, the introduction of rodent reservoirs, and the presence of favorable ecologic niches. Practices of the local population placed it repeatedly in high-risk exposures to epidemic and hyperendemic environments.

Author affiliations: Virginia Commonwealth University School of Medicine, Richmond, Virginia, USA (J.S. Marr); and King Faisal Specialist Hospital and Research Centre, Riyadh, Saudi Arabia (J.T. Cathey)

DOI: 10.3201/eid1602.090276

Epidemiology

The limited information available notes the following clinical manifestations of the illness: headache and fever with visible signs of epistaxis and jaundice. Mode of

Table 1. Summary of published interpretations suggesting or discounting possible causes of an epidemic among Native Americans, New England, 1616–1619*

Cause	Suggested	Discounted
Yellow fever	Webster (*2*)	Cook (*1*), Williams (*3*), Carter (*4*), Bratton (*6*)
Plague	Williams (*3*)	Carter (*4*), Hoornbeek (*5*), Bratton (*6*), Crosby (*9*)
Influenza	Carter (*4*)	
Smallpox	Bratton (*6*), Holmes (*7*)	Cook (*1*), Webster (*2*), Williams (*3*), Hoornbeek (*5*)
Chickenpox	Hoornbeek (*5*), Cronon (*10*)	Bratton (*6*)
Typhus	Lescarbot (*11*)	Williams (*3*), Bratton (*6*)
HBV/HDV	Speiss and Speiss (*8*)	
Leptospirosis	This study	

*HBV, hepatitis B virus; HDV, hepatitis D virus.

transmission was not known. Weather and seasonality are unknown, although tree ring data suggest greater than average rainfall in eastern Massachusetts during 1615–1625 (*12*). The duration of the epidemic (or epidemics) reportedly ranged from 3 to 6 years. Estimated death rates (which lack reliable numerator and denominator data) range from one third of the local population to as high as 90% (*1*,*13*). The Patuxet (Plimouth) Native American village was severely depopulated (*14*). Referring to conditions along the Newfoundland and Maine coasts, where some believe the epidemic may have originated, Pierre Biard, a Jesuit missionary, noted: "They [the Indians] are astonished and often complain that since the French mingle and carry on trade with them, they are dying fast, and the population is thinning out" (*15*). In New England, Smith noted "three plagues in three years successively neere two hundred miles along the coast" of southern Massachusetts to Cape Cod and inland for 15 miles (*16*). Bennett suggested a 50–60-mile interior extension, which corresponds to the area of native corn horticulture (*17*).

By 1616, several subtribes of the Wampanoag (Pokanoket) Nation were living between the present-day borders of eastern Rhode Island and southeastern Maine (Figure 1). The Patuxet village was localized to an area in and around Plymouth harbor (Figure 2). Demographers and historians disagree about the total size of the Wampanoag Nation, but Salisbury considers an estimate of 21,000–24,000 as "not unrealistic for this region" (*13*). Gookin also estimated 3,000 men living in Massachusetts before the epidemic (*18*), which when extrapolated for family size is consistent with Salisbury's overall estimate. Salisbury estimated that the size of the Patuxet tribe before the epidemic was 2,000.

No estimates are available of the number of Portuguese, Breton, and Bristol fishermen; Basque whalers; French fur traders; or English codders who had established a presence on the North Atlantic coast since the early sixteenth century (*10*). In 1578, an observer noted 100 Spanish sails, 20–30 Basque whalers, ≈150 French and Breton fishing ships, and 50 English sails along the coast of Newfoundland (*19*). English traders and fishermen had daily contact with indigenous persons but lived on ships or in segregated enclaves on land where salt-dried codfish stations (favored by the English) were built along Massachusetts Bay.

Ecology

Indigenous ecology was cataloged in 1604 when hundreds of coastal plants, trees, and animals (but not "vermine") were described (*20*). Before 1620, there were no peridomiciliary animals except for small dogs and mice (*10*), although other rodents (e.g., squirrels) were common. Precolonization and postcolonization English written accounts do not mention rats, the numbers of which may have been influenced by the presence of cats, but aboard

Figure 1. Native American tribes of southeastern Massachusetts in ≈1620.

ships rats must have been common. An earlier explorer noted "Tant qu'on eut des cuirs on ne s'avisa point de faire la guerre aux rats…" ("As long as there is a cargo of skins, it makes no sense to kill the rats.") (*11*). The black rat (*Rattus rattus*) was common in coastal England at the time (yet to be displaced by the brown rat [*R. norvegicus*] nearly 100 years later) (*21*); the black rat and mice were universal companions on ships and must have established themselves early on the coastal mainland, seeking harborage in and around Native American households. Once established, rats and mice would become chronic carriers of disease agents, contaminating water and soil and infecting other commensal rodents (e.g., the local mouse *Peromyscus leucopus*) and other mammals. Fresh and stored food items such as maize, beans, squash, pumpkin, roots, nuts, berries, meat, fish, and shellfish, were also susceptible to leptospiral contamination.

Previous Explanations

One hundred years ago, Williams collected all known information about the epidemic in an article that included 23 primary references, 22 of which contained eyewitness accounts or reports (*3*). He concluded that the disease may have been bubonic plague and supported his proposal by noting that there were abundant fleas in Indian dwellings, survivors had sores suggestive of buboes, and plague was endemic in London during 1606–1611. Eleven of his 23 primary sources disagreed, as did Carter, who without further elaboration stated that he thought the epidemic was influenza (*4*). Despite allusions to icterus, Williams discounted yellow fever (as did Carter); he also dismissed other febrile illnesses with jaundice, yet he cited Gookin from 1674: "I have discoursed with old Indians, who were then youths, who say that the bodies all over were exceedingly yellow, describing it by a yellow garment they showed me, both

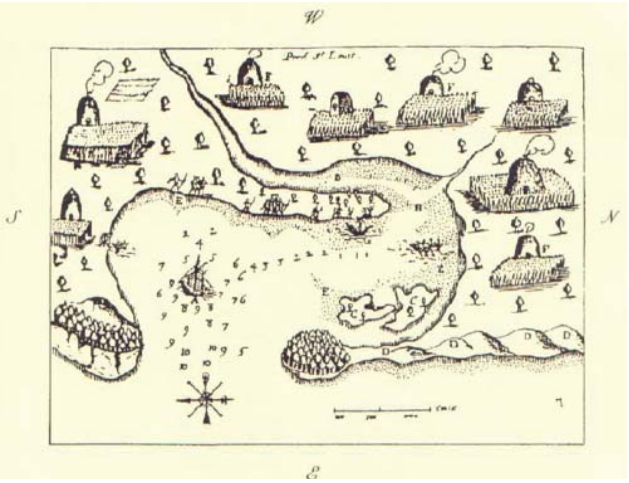

Figure 2. Plymouth, Massachusetts, harbor showing extensive Native American settlement (a sketch by Samuel de Champlain from his voyage of 1606).

before they died and afterwards." Trumbull, another eyewitness, noted that the Indian word for the disease meant "a bad yellowing" (*3*). A recent analysis interpreted it as caused by a confluent form of smallpox (*6*). Clinical and epidemiologic information about classical explanations and some of the more recent suggestions are summarized in Table 2.

Discussion

The causes of most historical epidemics may never be proven. The new science of paleomicrobiology may provide some answers, but the question will remain about whether a person died of a specific disease or with the disease. However, even when proper evidence is limited, this limitation should not dissuade speculation about the causes of ancient afflictions. Our hypothesis is not meant to be a definite answer but a heuristic for others to criticize and explore. Alfred Crosby, one of America's foremost medical historians, coined the term "virgin soil epidemics" to describe immunologically unexposed populations exposed to Old World diseases and cited the 1616–1619 epidemic as an example (*9*). He also proposed that environmental and behavioral factors were equally important (*22*). The Massachusetts epidemic supports this observation, and evidence may indicate that "genetic weakness" was not as important as the intimate and repeated exposure to an infectious agent among the Indians not shared by Europeans.

All previously proposed explanations for the epidemic are consistent with an Old World importation into a susceptible population (except for Webster's, who thought yellow fever was of autochthonous origin). Despite its manifestation and subsequent visitations along coastal America in later years, yellow fever is not a plausible explanation given the routes of the trans-Atlantic slave trade at the time. Transportation of the disease, its vector, and human cargo from Africa to the New World was limited to the Caribbean and Central and South America; little evidence exists that any ships visited the New England coast after disembarking slaves (*23*). Alternative arthropod-borne and other non-arthropod–borne viral hemorrhagic fevers are even less plausible candidates.

Clinical descriptions of other proposed diseases (plague, chickenpox, typhus, typhoid fever, and meningitis) are largely inconsistent with the syndrome described and were dismissed by Bratton. Citing Oliver Wendell Holmes, Sr. (*7*), Bratton concluded that the disease was smallpox, explaining that the confluent form of pustular smallpox might mimic jaundice (*6*). In 1799, Webster had discounted smallpox because "the Indians, who were perfectly acquainted with the disease [smallpox] after the English arrived, always gave a very different account of it..." (*2*). Two diseases not mentioned by Bratton (trichino-

Table 2. Factors related to some of the postulated causes of an epidemic among Native Americans, New England, 1616–1619*

Factor	Yellow fever	Plague	Influenza	Smallpox	Chickenpox	Typhus	HBV/HDV	Leptospirosis
Characteristic signs and symptoms								
Headache/fever	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Jaundice	Yes	No	No	No	No	No	Yes	Yes
Hemorrhages	Yes	Yes	No	Yes	No	Yes	No	Yes
Skin lesions†	Bruises	Buboes	No	Pustules	Rash	Rash	No	Rash
Epidemiologic								
High attack rate	Yes	Yes	Yes	Yes‡	Yes	Yes	Yes	Yes
High death rate	Yes	Yes	No	Yes	No	Yes	Yes	Yes
Endemic in Europe	No	Yes	Yes	Yes	Yes	Yes	No	Yes
Suitable arthropod vector	No	Yes	NA	NA	NA	Yes	NA	NA
Suitable reservoir host	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Native susceptibility	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
European susceptibility	Yes	Yes	Yes	No	No	Yes	Yes	Yes

*HBV, hepatitis B virus; HDV, hepatitis D virus; NA, not applicable.
†Sign mentioned by only 1 person (Thomas Dermer) and possibly referred to another unrelated disease outbreak.
‡Native Americans only.

sis and HBV/HDV infections) are also unlikely. Pigs were absent in the New World, and the finding of a single pig bone in an undated midden makes a most unlikely explanation for the epidemic. Syndemic HBV/HDV infection presupposes aboriginal HBV carriage, HDV importation, and (in the opinion of Speiss and Speiss) an enteric mode of transmission (*8*).

In 1886, Adolf Weil originally described a constellation of signs and symptoms that is now eponymic for Weil syndrome (his first patient experienced nasenbluten [nosebleed] on the second day of illness) (*24*). Inada and Ido identified the causative organism 30 years later (*25*). Subsequent studies have demonstrated that rodents have high rates of leptospiral carriage and shedding (*26*). Severe (icteric) leptospirosis was also known as infectious jaundice, epidemic jaundice, and icto-hemorrhagic fever (*27*). Early outbreaks in the United States were recorded by Neill, including a Union Civil War Surgeon General's report of a large number of "hepatic and haematic disorders" estimated to have affected >71,000 troops during the War (*28*).

In 1965, Heath et al. summarized the history of leptospirosis in the United States, analyzing 483 cases reported during 1949–1961 (*29*,*30*). Twenty-five percent were caused by *L*. serovar Icterohemorrhagiae. Today, *L*. Icterohemorrhagiae and other serovars (Canicola, Autumnalis, Hebdomidis, Australis, and Pomona) are endemic in the United States, and isolated instances within the United States continue to be reported (*31*). More recent reports from the Centers for Disease Control and Prevention (*32*,*33*) and ProMED mail (*34*) demonstrate that leptospirosis is a worldwide, reemerging infection with identifiable risk factors, including immersion in fresh water, exposure to contaminated soil, and antecedent heavy rains (*35*,*36*). Unlike hookworm disease, another Old World soil-borne disease that established itself in the more hospitable American South, leptospirosis is a more cosmopolitan fellow traveler and is still recognized as a zoonosis in New England.

Contemporary medical texts conflate signs, symptoms, and death rates of mild leptospiral infection with Weil syndrome, relying on more recent citations in which the nature of exposure, duration, and responsible *Leptospira* spp. are often not known. Interventional measures (removal from known sources, prompt diagnosis and treatment, and early prevention and control measures) may have decreased overall case-fatality rates and limited the extent of the outbreaks. Nosebleed is rarely mentioned in the recent literature, but "hemorrhages, starting with epistaxis" are noted in a 1944 text on tropical diseases, which also cites high death rates (32% in Europe and 48% in Japan) (*27*). These surprisingly high death rates in early Japanese reports were attributed to repeated intimate exposure to contaminated water by barefooted mine workers and rice farmers.

Unlike the European experience, epidemics in Japan were rare, and endemic exposures were more common (*27*). A recent population-based seroepidemiologic study found leptospiral seropositivity rates of 28% in an annually flooded area of the Amazon basin (*37*). *Leptospira* spp. were found to cause seasonal outbreaks of a mysterious disease (tentatively named Andaman hemorrhagic fever) during periods of rice paddy sowing and harvesting in the late 1980s on the Andaman Islands in the Indian Ocean (*38*). Subsequent studies found that leptospiral seroposivity was as high as 62.5% (among agricultural workers) in the Andaman Islands and that the case-fatality rate was 42.9% among hospitalized patients with severe leptospirosis and pulmonary symptoms.

Endemicity and subsequent high case-fatality rates, similar to those reported from Japan, are consistent with a leptospiral etiology for the 1616–1619 epidemic. The Patuxets may not have associated sickness with their environment or traditional ways of living and may have attributed their affliction to many causes, but not to countless exposures and reexposures to the agent. Sporadic, focal mini-epidemics may have played out and coalesced into what was construed as a single "plague" by outside observers. Except for more severe cases of liver failure, the most common cause of death for leptospirosis (renal or respiratory insufficiency) would have not been recognized. The Indian lifestyle, which included constant exposure to rodents and their excreta on land and in water, exposed them to the leptospiral life cycle (Figure 3) (*39*,*40*). Bare feet were common in and around houses. Although a rare portal of entry, mucosal exposure may have occurred from ingestion of corn buried in the ground in rodent-accessible baskets and from rodent-contaminated foods in wigwams (weetas). Dermal abrasions offered cutaneous portals of entry. Attendance of the ill and burial of the dead (including those who died from Weil syndrome) would have attracted others who shared local food, water, and camp grounds. It was common practice for entire families to enter sweat lodges followed by immediate immersion in cooling streams and ponds; sweat lodges were considered vivifiers and cure-alls for illnesses, a practice that may have reexposed the already ill to contaminated water. Once the spirochete established its presence in numerous foci, it survived for months in water, mud, and moist soil and caused infection in additional mammalian reservoirs. A reduction in the populace may have been incremental, episodic, and continuous; daily needs and customs may have exposed the Indians to leptospirosis over many months or years, with only a small fraction of the population eventually surviving. Suggestions that the disease persisted among the Indians after 1619 (perhaps through 1630) support the premise of endemic nidality and selective Indian vulnerability. The fate of nearby European cod fishermen is unknown, but they did not share

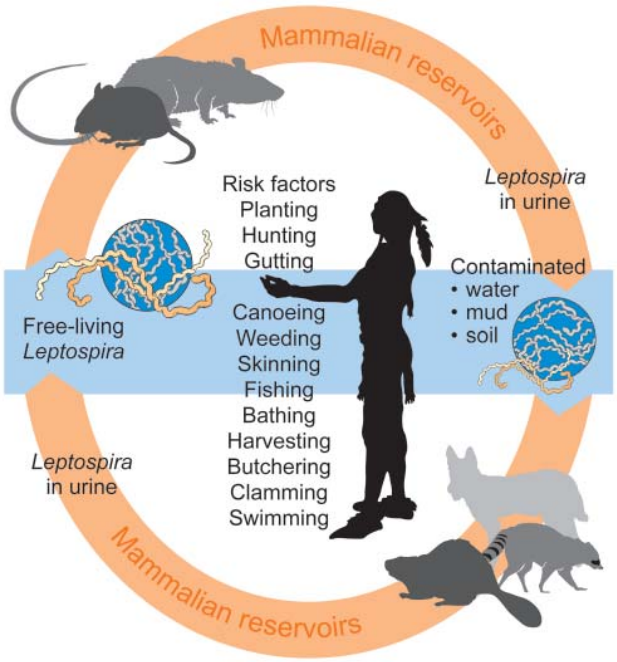

Figure 3. Leptospiral life cycle.

most of the Indians' risk factors. Boots would have limited transmission from fresh water exposures, bathing was not a common practice, and work in a saline environment may have curtailed transmission. An occasional case of febrile illness on board ship would have been attributed to many other causes. Disease and death may have occurred among the fishermen but are not recorded.

The exact duration and extent of the epidemic(s) will never be known, but our suggestion offers an alternative explanation. Persistent leptospiral exposures resulted in more severe cases of Weil syndrome and jaundice, a sign that would have been reported by observers; the cause of death from other (anicteric) leptospiral infection would not have been recognized. Our proposal is consistent with the historical clinical descriptions, estimated death rates, importation and distribution of its reservoir host, inoculation of the agent in multiple suitable nidalities, spread to other mammalian reservoirs, hyperendemicity, ecologic factors favoring repeated exposure and transmission, and known high-risk activities of the indigenous population.

The name Squanto has entered American history and folklore as the one of the last of the Patuxets who assisted the Pilgrims in 1620. He was one of the few survivors of an epidemic that was crucial to the success of the Plymouth and Massachusetts Bay colonies because remaining Indians had little capacity to resist the new settlers. Two years later, after having fever and a nosebleed, Squanto died of what was then referred to as "the Indean disease."

Acknowledgments

We thank Alfred W. Crosby, Asim A. Jani, Grayson B. Miller, Myron G. Schultz, and Jack Woodall for critical comments; Philip McEldowney for literature search/retrieval; Stefanie Nauhardt Parker for translation; Mariana Ruiz-Villarreal and David Connell for providing the leptospiral life cycle; and Reina Tejano and Samuel de Champlain for providing the maps.

Dr Marr is a professor at Virginia Commonwealth University School of Medicine, Richmond, Virginia, and at East Carolina University School of Medicine, Greenville, North Carolina. His research interests include public health history and historical epidemics and diseases.

Mr Cathey is senior editor of the Annals of Saudi Medicine at King Faisal Specialist Hospital, Riyadh, Saudi Arabia, and a professional medical writer. His research interest is historical epidemics.

References

1. Cook SF. The significance of disease in the extinction of the New England Indians. Hum Biol. 1973;45:485–508.
2. Webster N. A brief history of epidemic and pestilential diseases. Hartford (CT): Hudson and Goodwin; 1799.
3. Williams H. The epidemic of the Indians of New England, 1616–1620, with remarks on Native American infections. Johns Hopkins Hospital Bulletin. 1909;20:340–9.
4. Carter H. Yellow fever: an epidemiological and historical study of its place of origin. Baltimore: Williams and Wilkins; 1931.
5. Hoornbeek B. An investigation into the cause or causes of the epidemic which decimated the Indian population of New England 1616–1619. The New Hampshire Archeologist. 1976–1977;19:35–46.
6. Bratton TL. The identity of the New England Indian epidemic of 1616–19. Bull Hist Med. 1988;62:351–83.
7. Holmes OW. The medical profession in Massachusetts. Medical essays, 1842–1882. Boston: Houghton-Mifflin; 1896 [cited 2009 Sep 17]. http://www.gutenberg.org/files/2700/2700.txt
8. Spiess A, Spiess BD. New England pandemic of 1616–1622: cause and archeological implication. Man in the Northeast. 1987;34:71–83.
9. Crosby AW. Virgin soil epidemics as a factor in the aboriginal depopulation in America. William Mary Q. 1976;33:289–99. DOI: 10.2307/1922166
10. Cronon W. Changes in the land: Indians, colonists and the ecology of New England. New York: Hill and Wang; 1983.
11. Lescarbot M. The history of New France. Toronto: The Champlain Society; 1907.
12. Cook ER, Woodhouse CA, Eakin CM, Meko DM, Stahle DW. Long-term aridity changes in the western United States. Science. 2004;306:1015–8. DOI: 10.1126/science.1102586
13. Salisbury N. Manitou and Providence: Indians, Europeans, and the making of New England 1500–1643. New York: Oxford University Press; 1982.
14. Sturtevant W, Trigger BC. Handbook of North American Indians. Washington: Smithsonian Institution; 1978.
15. Thwaites RG. The Jesuit relations and allied documents: travels and explorations of the Jesuit missionaries. Cleveland (OH): Burrows Brothers; 1897.
16. Smith J. New England's trials. Peter Force, editor: tracts and other papers; 1622.

17. Bennett M. The food economy of the New England Indians 1605–1675. J Polit Econ. 1955;63:369–97. DOI: 10.1086/257706
18. Gookin D. Historical collections of the Indians in New England: of their several nations, numbers, customs, manners, religion, and government, before the English planted there. Fiske J, editor. New York: Arno Press; 1972. p. 7–12.
19. Grant W, Jameson JF. Voyages of Samuel de Champlain 1604–1618. New York: Charles Scribner's Sons; 1907.
20. Smith J. A description of New England (1616): an online electronic text edition. Royster P, editor: Lincoln (NE): University of Nebraska Press; 2006 [cited 2009 Sep 21]. http://works.bepress.com/paul_royster/7
21. Zinsser H. Rats, lice and history. Boston: Little, Brown and Company; 1938.
22. Jones DS. Virgin soils revisited. William Mary Q. 2003;60:703–42.
23. Eltis D, Halbert M. Voyages: the trans-Atlantic slave trade database [cited 2008 Dec 31]. http://www.slavevoyages.org
24. Weil A. Ueber eine eigenthumlich, mit milztumor, icterus und nephritis einhergehende, acute infectionskrankheit. Deutsch Archiv für Klinische Medizin. 1886;39:209–32.
25. Inada R, Ido Y. A report of the discovery of the causal organism (a new species of spirocheta) of Weil's disease. Tokyo Ijishinshi. 1908;1915:351–60.
26. Aviat F, Blanchard B, Michel V, Blanchet B, Branger C, Hars J, et al. *Leptospira* exposure in the human environment in France: a survey in feral rodents and in fresh water. Comp Immunol Microbiol Infect Dis. 2009;32:463–76. DOI: 10.1016/j.cimid.2008.05.004
27. Strong R. Stitt's diagnosis, prevention and treatment of tropical diseases. 7th ed. Philadelphia: Blakiston; 1944.
28. Neill M. The problem of acute infectious jaundice in the United States. Public Health Rep. 1918;33:717–26.
29. Heath CW, Alexander AD, Galton MM. Leptospirosis in the United States. Analysis of 483 cases in man, 1949–1961 (Part 1). N Engl J Med. 1965;273:857–64.
30. Heath CW, Alexander AD, Galton MM. Leptospirosis in the United States. Analysis of 483 cases in man, 1949–1961 (Part 2). N Engl J Med. 1965;273:915–22.
31. Meites E, Jay MT, Deresinski S, Shieh WJ, Zaki SR, Tompkins L, et al. Reemerging leptospirosis, California. Emerg Infect Dis. 2004;10:406–12.
32. Gaynor K, Katz AR, Park SY, Nakata M, Clark TA, Effler PV. Leptospirosis on Oahu: an outbreak associated with flooding of a university campus. Am J Trop Med Hyg. 2007;76:882–5.
33. Centers for Disease Control and Prevention. Update: outbreak of acute febrile illness among athletes participating in Eco-Challenge-Sabah 2000 Borneo, Malaysia, 2000. JAMA. 2001;285:728–30. DOI: 10.1001/jama.285.6.728
34. Leptospirosis—Sri Lanka. (07): (North Western Province) ProMed. 2008; Dec 31 [cited 2009 Sep 17]. http://www.promedmail.org Archive no. 20081231.4127.
35. Levett PN. Leptospirosis. Clin Microbiol Rev. 2001;14:296–326. DOI: 10.1128/CMR.14.2.296-326.2001
36. Ricaldi JN, Vinetz JM. Leptospirosis in the tropics and in travelers. Curr Infect Dis Rep. 2006;8:51–8. DOI: 10.1007/s11908-006-0035-3
37. Johnson MA, Smith H, Joeph P, Gilman RH, Bautista CT, Campos KJ, et al. Environmental exposure and leptospirosis, Peru. Emerg Infect Dis. 2004;10:1016–22.
38. Vijayachari P, Sugunan AP, Sharma S, Roy S, Natarajaseenivasan K, Sehgal SC. Leptospirosis in the Andaman Islands, India. Trans R Soc Trop Med Hyg. 2008;102:117–22. DOI: 10.1016/j.trstmh.2007.08.012
39. Russell H. Indian New England before the Mayflower. Hanover (NH): University Press of New England; 1980.
40. Karr R. Indian New England 1524–1674: a compendium of eyewitness accounts of Native American life. Pepperill (MA): Branch Line Press; 1999.

Address for correspondence: John S. Marr, 6315 Pig Mountain Road, Free Union, VA 22940, USA; email: jsmarr4@earthlink.net

etymologia

Cryptococcus gattii

[krip″ to-kok′əs ga-te-i]

This yeast genus takes its name from the Greek *kryptos*, hidden, and *kokkos*, berry. The pathogen has been recently recognized as a distinct species that causes infection (with cutaneous, pulmonary, and neurologic manifestations) in both humans and animals. The species was named for Italian mycologist Franco Gatti who, with Roger Eeckels, described an atypical strain of *C. neoformans* in the cerebrospinal fluid of a Congolese Bantu boy with cryptococcosis in 1970.

Sources: Vanbruseghem R, Takashio M. An atypical strain of *Cryptococcus neoformans* (San Felice) Vuillemin 1894. Part II. *Cryptococcus neoformans* var *gattii* var. nov. Am Soc Belge Med Trop. 1970;50:695–702; Springer DJ, Chaturvedi V. Projecting global occurrence *Cryptococcus gattii*. Emerg Infect Dis. 2010;16:14–20; Dorland's illustrated medical dictionary, 31st ed. Philadelphia: Saunders Elsevier; 2007.

To his Worshipfull Friend M. SAMVEL PVRCHAS, *Preacher of the Word, at the Church a little within* Ludgate, London.

Sir,

IT was the nineteenth of May, before I was fitted for my discouery, when from *Monahiggan* I set sayle in an open Pinnace of fiue tun, for the Iland I told you of. I passed alongst the Coast where I found some antient Plantations, not long since populous now vtterly void; in other places a remnant remaines, but not free of sicknesse. Their disease the Plague, for wee might perceiue the sores of some that had escaped, who described the spots of such as vsually die. When I arriued at my Sauages natiue Country (finding all dead) I trauelled alongst a daies iourney Westward, to a place called *Nummastaquyt*, where finding Inhabitants, I dispatched a Messenger a dayes iourney further West, to *Poconaokit* which bordereth on the Sea; whence came to see me two Kings, attended with a guard

1619 *May*

Monhegan

CHAP. III.

Of a great mortality that happened amongſt the Natives of New England, neere about the time that the Engliſh came there to plant.

IT fortuned ſome few yeares before the Engliſh came to inhabit at new Plimmouth, in New England, that upon ſome diſtaſt given in the Maſſachuſſets bay by Frenchmen, then trading there with the Natives for beaver, they ſet upon the men at ſuch advantage that they killed manie of
* 23 them, burned their ſhipp, * then riding at Anchor by an Iſland there, now called Peddocks Iſland,[1] in memory of Leonard Peddock[2] that landed there, (where many wilde

in writing his *New England's Proſpect*, in 1633, remarks (p. 78), that "Some have thought they [the Indians] might be of the diſperſed Jews, becauſe ſome of their words be near unto the Hebrew; but by the ſame rule they may conclude them to be ſome of the gleanings of all nations, becauſe they have words which ſound after the Greek, Latin, French, and other tongues."

There is in the *Magnalia* (book III. part iii.) a lengthy but highly characteriſtic paſſage, in which Mather recounts the points of reſemblance which the evangeliſt Eliot ſaw between the Indians and "the poſterity of the diſperſed and rejected Iſraelites."

[1] Peddock's, or Pettick's, Iſland, ſtill ſo called, is one of the largeſt iſlands in Boſton Bay. It lies directly oppoſite to George's Iſland and Hull, from which laſt it is ſeparated by a narrow channel, and is between Weymouth and Quincy bays, on the eaſt and weſt. See Shurtleff's *Deſcription of Boſton*, p. 557.

[2] Leonard Peddock ſeems to have been in the employment of the Council for New England. In the records of the Council for the 8th of November, 1622, is the following entry: "Mr. Thomſon is ordered to pay unto Leo: Peddock £10 towards his paynes for his laſt Imployments to New England." Subſequently, on the 19th of the ſame month: "It is ordered that a Letter be written from the Counſell to Mr. Weſton, to deliver to Leonard Peddock, a boy Native of New England called papa Whinett belonging to Abbadakeſt, Sachem of Maſſachuſetts, which boy Mr Peddock is to carry over with him" (*Proceedings of the American Antiquarian Society*, April, 1867, pp, 70, 74).

Andrew Weſton had returned to England in the *Charity*, leaving Weſſaguſſett in September, 1622 (*ſupra*, 7). He would

wilde Anckies[1] haunted that time, which hee thought had bin tame,) diſtributing them unto 5. Sachems, which were Lords of the feverall territories adjoyninge: they did keepe them ſo longe as they lived, onely to ſport themſelves at them, and made theſe five Frenchmen fetch them wood and water, which is the generall worke that they require of a ſervant.[2] One of theſe five men, out livinge the reſt, had learned ſo much of their language as to rebuke them for their bloudy deede, ſaying that God would be angry with them for it, and

Five Frenchmen kept by the Salvages.

would ſeem to have brought over the Indian boy in queſtion with him. From the entry in the records of the Council for New England, juſt quoted, it would appear that Leonard Peddock was in New England during the ſummer of 1622. The reference to him in the text is additional evidence that Morton was there at the ſame time, and in company with Weſton.

[1] This is undoubtedly a miſprint for Auckies, which was a ſailor's corruption for Auks. The Great Auk (*Alca impennis*) is probably referred to. This bird, now ſuppoſed to be extinct, was formerly common on the New England coaſt. Audubon, writing in 1838, ſays: "An old gunner, reſiding on Chelſea Beach, near Boſton, told me that he well remembered the time when the Penguins were plentiful about Nahant and ſome other iſlands in the bay." (*Am. Ornithological Biog.*, vol. iv. p. 316.) Profeſſor Orton, alluding to this paſſage, in the *American Naturaliſt* (1869, p. 540), expreſſes the opinion that the Razor-billed Auk was the bird referred to; but Profeſſor F. W. Putnam adds, in a foot-note, that "the 'old hunter' was undoubtedly correct in his ſtatement, as we have bones of the ſpecies taken from the ſhell-heaps of Marblehead, Eagle Hill in Ipſwich, and Plum Iſland." Dr. Jeffries Wyman found them in the ſhell-heaps at Cotuit. See *Mem. Hiſt. of Boſton*, vol. i. p. 12.

There is an elaborate paper on the Great Auk, under the title of "The Gareſowl and its Hiſtorians," by Profeſſor Alfred Newton, in the *Natural Hiſtory Review* for 1865, p. 467.

[2] Morton would ſeem to be miſtaken in this ſtatement. Between 1614 and 1619 two French veſſels were loſt on the Maſſachuſetts coaſt. One was wrecked on Cape Cod, and the crew, who ſucceeded in getting on ſhore, were moſt of them killed by the ſavages, and the remainder enſlaved in the way deſcribed in the text. Two of theſe captives were ſubſequently redeemed by Captain Dermer (Bradford, p. 98). The other veſſel was captured by the ſavages in Boſton Bay, and burned. This is the veſſel referred to by Morton as riding at anchor off Peddock's Iſland. The circumſtances of the capture are deſcribed in Phinehas Pratt's narrative (IV. *Maſs. Hiſt. Coll.*, vol. iv. pp. 479, 489). All the crew, he ſays, were killed, and the ſhip, after grounding, was burned. Pratt's ſtatement is diſtinct, and agrees with Bradford's, that the captives among the Indians were the ſurvivors from the veſſel wrecked on Cape Cod, not from that captured in Boſton Bay.

and that hee would in his diſpleaſure deſtroy them; but the Salvages (it ſeemes boaſting of their ſtrenght,) replyed and ſayd, that they were ſo many that God could not kill them.[1]

The Plague fell on the Indians.

But contrary wiſe, in ſhort time after the hand of God fell heavily upon them, with ſuch a mortall ſtroake that they died on heapes as they lay in their houſes; and the living, that were able to ſhift for themſelves, would runne away and let them dy, and let there Carkaſes ly above the ground without buriall. For in a place where many inhabited, there hath been but one left a live to tell what became of the reſt; the livinge being (as it ſeemes) not able to bury the dead, they were left for Crowes, Kites and vermin to pray upon. And the bones and ſkulls upon the ſeverall places of their habitations made ſuch a ſpectacle after my comming into thoſe partes, that, as I travailed in that For-

The livinge not able to bury the dead.

reſt

[1] Pratt's account of this ſurvivor among the French crew is to be found in IV. *Maſs. Hiſt. Coll.*, vol. iv. pp. 479, 489. He ſays that "one of them was wont to read much in a book (ſome ſay it was the New Teſtament). and that the Indians enquiring of him what his book ſaid, he told them it did intimate that there was a people like French men that would come into the country and drive out the Indians." The account given by Mather (*Magnalia*, B. I. ch. ii. § 6) is curiouſly like that in the text. After quoting the ſubſtance of Pratt's ſtatement he adds: "Theſe infidels then blaſphemouſly replied, 'God could not kill them;' which blaſphemous miſtake was confuted by a horrible and unuſual plague, whereby they were conſumed in ſuch vaſt multitudes that our firſt planters found the land almoſt covered with their unburied carcaſes; and they that were left alive were ſmitten into awful and humble regards of the Engliſh by the terrors which the remembrance of the Frenchman's prophecy had imprinted on them."

Pratt, whom Mather followed, claims to have derived his knowledge of theſe events during the winter of 1622–3 directly from ſavages concerned in them. The probability is that the tradition of the French captive, and his book and prophecy, was a common one among the ſettlers both at Plymouth and about Boſton Bay. Pratt apparently had a habit, as he grew old, of appropriating to his own account many of the earlier and more ſtriking incidents of colonial hiſtory. (Mather's *Early New England*, p. 17.)

reſt nere the Maſſachuſſets, it ſeemed to mee a new found Golgatha.

* But otherwiſe, it is the cuſtome of thoſe Indian * 24
people to bury their dead ceremoniouſly and carefully,
and then to abandon that place, becauſe they have no deſire the place ſhould put them in minde of mortality: and this mortality was not ended when the Browniſts of new Plimmouth were ſetled at Patuxet in New England: and by all likelyhood the ſickneſſe that theſe Indians died of was the Plague, as by conference with them ſince my arrivall and habitation in thoſe partes, I have learned.[1] And by this meanes

[1] The myſterious peſtilence, which in the years 1616 and 1617 ſwept away the New England Indians from the Penobſcot to Narraganſett Bay, is mentioned by all the earlier writers, and its character has recently been ſomewhat diſcuſſed. There can be no doubt that it practically deſtroyed the tribes, eſpecially the Maſſachuſetts and the Pokanokets, among which it raged. The former were reduced from a powerful people, able, it is ſaid, to muſter three thouſand warriors, to a mere remnant a few hundred ſtrong. The Pokanokets were in ſome localities, notably at Plymouth, actually exterminated, and the country left devoid of inhabitants (I. *Maſs. Hiſt. Coll.*, vol. i. p. 148; Young's *Chron. of Pilg.*, p. 183). Winſlow gave a deſcription of the deſolation created by this peſtilence, and of the number of the unburied dead, very like that in the text (Young's *Chron. of Pilg.*, pp. 183, 206). On this ſubject, ſee alſo, Bradford, pp. 102, 325; Johnſon, p. 16; Wood's *Proſpect*, p. 72; III. *Maſs. Hiſt. Coll.*, vol. vi. p. 57.

No definite concluſion as to the nature of this peſtilence has been reached by medical men. It has been ſuggeſted that it was the yellow-fever (Palfrey, vol. i. p. 99, *n*). As, however, it raged equally in the depth of the ſevereſt winter as in ſummer, this could not have been the caſe (III. *Maſs. Hiſt. Coll.*, vol. vi. p. 57; Bradford, p. 325). Other modern medical authorities have inclined to the opinion that it was a viſitation of ſmall-pox (Dr. Holmes in *Maſs. Hiſt. Soc., Low. Inſt. Lect.*, 1869., p. 261; Dr. Green's *Centennial Addreſs before the Maſs. Med. Soc.*, June 7, 1881, p. 12). In ſupport of this hypotheſis Captain Thomas Dermer is quoted, who, ſailing along the coaſt in 1619–20, wrote "we might perceive the ſores of ſome that had eſcaped, who deſcribed the ſpots of ſuch as uſually die" (Purchas, vol. iv. p. 1778). On the other hand, none of the contemporaneous writers who ſpeak of the diſeaſe ever call it the ſmall-pox, though all of them were perfectly familiar with ſmall-pox, and a very large portion of them probably bore its marks. Dermer ſpeaks of it as "the plague." Bradford, when the ſame peſtilence raged on the Connecticut, deſcribed it as "an infectious fever." Dr. Fuller, the firſt New England phyſician, then died of it (Bradford, p. 314). He could

meanes there is as yet but a ſmall number of Salvages in New England, to that which hath beene in former time, and the place is made ſo much the more fitt for the Engliſh
2 *Sam.* 24. Nation to inhabit in, and erect in it Temples to the glory of God.

CHAP. IV.

Of their Houſes and Habitations.

THe Natives of New England are accuſtomed to build them houſes much like the wild Iriſh; they gather Poles in the woodes and put the great end of them in the ground, placinge them in forme of a circle or circumference, and, bendinge the topps of them in forme of an Arch, they bind them together with the Barke of Walnut trees, which is wondrous tuffe, ſo that they make the ſame round
*25 on the Topp *for the ſmooke of their fire to aſſend and

could not but have been familiar with the ſmall-pox and its ſymptoms; and it would ſeem moſt improbable that he ſhould have died of that diſeaſe among his dying neighbors, and not have known what was killing him. Moreover, in 1633–4 the ſmall-pox did rage among the Indians, and Bradford, in giving a fearfully graphic account of its ravages, adds, "they [the Indians] fear it more than the plague." Joſſelyn alſo draws the ſame diſtinction, ſaying (*Two Voyages*, p. 123): "Not long before the Engliſh came into the country, happened a great mortality amongſt [the Indians]; eſpecially where the Engliſh afterwards planted, the Eaſt and Northern parts were ſore ſmitten by the contagion; firſt by the plague, afterwards, when the Engliſh came, by the ſmall-pox."

It would ſeem, therefore, that the peſtilence of 1616–7 was clearly not the ſmall-pox. More probably it was, as Bradford ſays, "an infectious fever," or ſome form of malignant typhus, due to the wretched ſanitary condition of the Indian villages, which had become overcrowded, owing to that proſperous condition of the tribes which Smith deſcribes as exiſting at the time of his viſit to the coaſt in 1614 (III. *Maſs. Hiſt. Coll.*, vol. vi. p. 109).