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“The Lived Experience of the Black Death”

Megan Webb

The historiography of the Black Death includes a debate as to the exact epidemiology of the pathogen that struck Europe in 1348. Various historians have chimed in as to what, exactly, may have been the root cause of the pestilence – with theories ranging from bubonic plague to anthrax or influenza. There is also a question as to whether this debate is even relevant to the study of the Black Death – whether a confirmed medical diagnosis can illuminate a new understanding of the pestilence, or if the epidemiological debate only serves to obfuscate the Black Death’s greater historical consequences. The relevance of the debate is in how people experienced the pestilence as physical beings. The lived experience of the body is an important, and often insufficiently explored, sector of historical inquiry. The presentation, treatment, and attitudes associated with a specific disease are effected by its biology. Understanding the epidemiology of that disease is therefore integral to understanding a culture’s reactions to its incidence.

To appreciate the importance of the biological effects of disease on a society’s lived experience, it can be useful to look at modern examples. Polio provides an excellent example. Children who survive an infection of polio – and escape the neurological incapacitation that can result in disability up to paraplegia – have a fifty percent chance of suffering the similar effects of post-polio syndrome later in life. Similarly, children and adults who survived chicken pox unscathed can later be at risk for developing shingles. Syphilis, when left untreated, can cause its victims to go insane. The epidemiology of a specific disease can have far-reaching consequences both for the patients that suffer from it and the society as a whole.

Furthermore, absent an understanding of the biology of a disease, cultural and socioeconomic factors may obscure similarities in the history of epidemics. For example, if one were to compare the effects of the AIDS virus in the developed versus under-developed countries, without any biological knowledge one would assume these populations were suffering from two different diseases. The AIDS of the United States is predominantly understood to be a disease of gay men and intravenous drug users; it was for many years associated with immorality. On the other hand, the AIDS of Africa infects large segments of people who live in poverty, irrespective of gender or sexual orientation.

Nevertheless, while culture may steer how a disease interacts with the community, the disease's biology is what powers the boat. Just as we can only understand the similar spread of AIDS in both the developed and under-developed world by appreciating its biological properties, the key to understanding the incidence, spread, and consequences of the Black Death lies in the identification of the bacillus and how it interacts with the lived human experience. Investigations into previously unexplored historical subjects involving biology and ecology can shed light onto how the human body interacts with and is shaped by its environment.

Philip Ziegler's The Black Death is a work which gives an account of the pestilence based entirely on a survey of secondary sources authored by historians. Ziegler asserts that the epidemic which struck Europe in 1348 was the bubonic plague. The plague has struck in three great pandemics – first in Justinian's Byzantium in 542, then the Black Death, then a third pandemic in 1892. After comparing the three pandemics, Ziegler asserts that the symptoms as recorded by medieval chroniclers coincide exactly with the symptoms of modern plague –

namely buboes followed by neurological distress. Consequently, there is no question in his mind that these pandemics were all caused by the *Y. pestis* bacillus that was discovered in 1894.¹

Curiously, given his emphasis on the symptoms of the disease, Ziegler ignores evidence relating to the plague's transmission vector. The bubonic plague is spread by the flea *X. cheopis*, which subsists on the blood of rodents, especially rats. The spread of rats into areas inhabited by humans can introduce plague into that community as fleas begin to spread to human hosts. Yet, according to Ziegler, rats are not essential to the spread of plague. The rat flea can survive for long periods without a rat to feed on; therefore, rats are not entirely necessary for the spread of plague.² The prevalence of rats in this time period and its importance in plague transmission, however, as proven in later works, has shown that an historian ignores the rat at his or her own peril.

Ziegler's rejection of the biological elements that influence plague marks a lack of interest in exploring the lived experience of the plague. Whereas all of the following historians will note the many dissimilarities between symptoms of the Black Death and plague, Ziegler assumes them to be the exactly same. He leaves no room for the debate concerning epidemiology, going so far as to assert that there is no debate: it was definitely the plague. By leaving out the sources and evidence that contradict this assertion, Ziegler molds his historical argument into a fiction to his liking. Even if he is right, and the pestilence was caused by the bubonic plague, by forcing the evidence to support his truth claims, he is writing a work of fiction.

David Herlihy claims in The Black Death and the Transformation of the West that the fourteenth-century pestilence was most likely not bubonic plague. He bases this conclusion on

¹ Philip Ziegler, The Black Death. The John Day Company, New York, 1969, 20.

² Ziegler, 27.

contemporary medieval records of rodent activity, which he claims were not conducive to the spread of plague. He also cites differences between the reported symptoms of the two infections.³

Bubonic plague in human communities is predicated by a massive die-off in rat populations, as shown in China and India in 1892. Humans only catch the disease after the fleas have abandoned dead infected rats for the next available living host. No epizootic die-off was recorded by medieval chroniclers. Furthermore, the complex vector the disease takes for infection – which rarely occurs directly from person to person – does not coincide with the quick spread characteristic of the Black Death.⁴

In a comparison between the Black Death and the bubonic plague, the symptoms do not coincide. In plague, usually a single buboe forms at the groin, whereas during the Black Death buboes formed in several places on the body, usually on the neck. The buboe was not considered the sign of the pestilence; rather, the “plague girdle” or lenticulae designated this specific disease. Many of the symptoms are shared by other virulent epidemic diseases such as typhoid, anthrax, and influenza. None of these diseases, including bubonic plague, shares all of the symptoms of Black Death. It is possible this discrepancy can be attributed to a combination of several diseases striking concurrently; it is also possible that the Black Death was caused by a variant or mutation of plague that is symptomatically different from modern plague.⁵

If the Black Death was not caused by the plague, then all of the knowledge gained in the pandemic of 1892 cannot be retroactively applied to the past. One may question how much of our historical knowledge is, at its source, a projection of modern evidence onto past events. If

³ David Herlihy, The Black Death and the Transformation of the West. Harvard University Press, Cambridge, 1997, 7.

⁴ Herlihy, 26.

⁵ Herlihy, 30.

that modern evidence is incorrect, then our conclusions about the consequences of the pestilence may also be incorrect. Herlihy notes the discrepancies between the past and modern plague, but fails to apply his doubt to the historiography of the Black Death. He assumes that the diseases of the past are different from the diseases of today, and leaves it at that.

The Black Death: A History of Plagues 1345-1730 by Spicer and Naphy claims that the debate as to the correct identification of the pestilence cannot be resolved through examination of the sources. Chroniclers were not medically trained professionals and may not have been able to differentiate between the primary disease and any secondary infections producing conflicting symptoms. The disease identified in the 1890's may not be the cause of the first and second pandemics.⁶

Spicer and Naphy explain the discrepancies by attributing another disease altogether for the cause of the pestilence. They also suggest that a possible mutation of the bacillus caused the disease to present differently in modern plague than it did in medieval plague. This would account for the plague presenting different symptoms during each pandemic. However, the symptoms of the Black Death are present with other virulent diseases as well. Furthermore, any outbreaks of modern-type plague should have been preceded by a massive rodent mortality, but no records exist in western chronicles. Questions of the spread of the disease and the typical behavior of rats and rat fleas cast doubt on the plague being the cause.⁷

Spicer and Naphy's involvement in the epidemiological debate is to claim that the entire discussion is pointless. Since there is no clinical way to identify the germ, the question can never be fully resolved. The argument over epidemiology obscures the events themselves - knowing the cause would not answer any other questions about the short and long-term consequences of

⁶ Andrew Spicer and William Naphy, The Black Death: A History of Plagues: 1345-1730. Tempus, Charleston, 2000, 53.

⁷ Spicer and Naphy, 55.

the pestilence. Knowing the answer would not change the telling of its history. Bickering over the identification detracts historians from more relevant attempts to understand the impacts of and responses to the Black Death.⁸

Yet ignoring the biology of a disease also prevents us from isolating biological factors that may have led to cultural predispositions. The Black Death was a disease with both moral and bodily implications. Understanding the disease is necessary in order to understand people's reactions to it, and how they themselves understood it. For example, influenza and mononucleosis are both spread via the breath. If you sicken with the first, it is understood that you caught it because it is flu season. If you catch the latter, you are accused of catching it from someone specific that you have been kissing. The understanding of attitudes such as these stems both from culture and from the biological reality of the disease. Both are necessary to gain a fuller picture of the events.

Didier Raoult entered the epidemiological debate with "Molecular Identification.. of *Y. pestis* as the agent of medieval Black Death." In this article, Raoult weighs in on the debate regarding differences in symptoms and transmission of the bubonic plague. He seeks to end the controversy and improve understanding of bubonic epidemics through the application of DNA tests on archeological evidence. He tested skeletons that, due to information at the grave site, were hypothesized Black Death victims. In his testing, Raoult found *Y. pestis* in the dental pulp. Control tests for other possible diseases, such as those that share many symptoms with the pestilence, were negative. Raoult concludes that the Black Death was caused by the bubonic plague bacillus, and the debate is now over.⁹

⁸ Spicer and Naphy, 56.

⁹ Didier Raoult et alia, "Molecular Identification by 'Suicide PCR' of *Yersinia Pestis* as Agent of Medieval Black Death," *Proceedings of the National Academy of Sciences of the United States of America*, 97 (Nov. 2000): 12802.

Others could and have argued against Raoult's results as reliable historical evidence. Someone with the correct technical training could find fault with Raoult's methods or procedures; such criticisms are not appropriate for an historian to make. The point a historian could make is that, even if the persons examined had indeed contracted plague, it does not mean that every other case during the Black Death was bubonic. Until analysis is done on a much, much larger scale to rule out errors in sample size, the results are still inconclusive. While this type of analysis is important to settling the debate as Raoult claimed, it is not going to happen overnight. A confirmed case of plague from a grave site known to be from the Black Death does not infer that all cases of the Black Death were caused by the plague.

After Raoult ended the debate, Samuel Cohn, Jr.'s "The Black Death: End of a Paradigm" insisted on claiming that the Black Death was not caused by bubonic plague. His reasoning is based on the difference in signs and symptoms between the two diseases, as well as evidence of humans' immunity and adaptability to each.¹⁰

Cohn points out differences present in the transmission of the disease. The form of transmission is highly contagious in the pestilence. However, the complex rat-flea-human vector makes modern plague difficult to transmit person to person or at any great speed.¹¹ Cohn accedes that the presence of rats is an integral part of the spread of plague. However, during the Black Death, rats were present in insufficient numbers to account for a large-scale human epidemic.¹² Furthermore, Cohn doubts the results found by Raoult's DNA tests. Extraction of DNA is

¹⁰ Samuel K. Cohn, Jr. "The Black Death: End of a Paradigm," *The American Historical Review*. 107 (Jun. 2002), 703.

¹¹ Cohn, 711.

¹² Cohn, 714.

fraught with difficulties, he claims, which can result in erroneous attributions of plague. No other sites doing similar work in Europe have corroborated Raoult's findings.¹³

The main thrust of Cohn's argument against plague is that it just does not make sense for the epidemiology of the plague to shift so rapidly. If the plague caused both the 1348 and the 1892 epidemics, it experienced a drastic shift in both transmission and possibility for human immunity. It would have had to evolve from spreading directly between persons, to becoming dependent on the rat-flea-human vector. Usually disease transmission evolves the other way around, evolving from more complex to less complex transmission vectors. Evidence also suggests that humans rapidly adapted to the pestilence and over time domesticated it to a disease of childhood. No human groups ever developed immunity to the plague during the third pandemic.

Cohn's argument at this juncture makes sense, but historical truths cannot be built out of common-sense deductions alone. Where is his evidence that suggests such evolution is impossible in bacilli? No one developed immunity from the bubonic plague – but this evidence is taken from a period of a mere forty years, not the nearly three hundred in which the Black Death reigned. How long does it take a global community to develop immunity? Without the insights that historians can gain from scientific evidence, Cohn's main argument hinges on a guess.

Still, despite his guesswork, Cohn answers Spicer and Naphy's condemnation of the epidemiology debate. The debate is significant, he says, because different diseases result in different consequences for bodies and minds. Understanding those consequences can only be

¹³ Cohn, 735.

achieved through understanding the pathogen that caused them.¹⁴ Cohn understands that the lived experience of the body is an important part of historical inquiry.

Michael McCormick's "Rats, Communications, and Plague: Toward an Ecological History" addresses the arguments of previous plague historians. McCormick argues that the history of rats is important in the rise and fall of the ancient world and the development of the medieval economy.¹⁵ Rodents may have played a role in sustaining or transmitting plague. Therefore the population dynamics, geography, and migration of rodents are historically important.¹⁶

McCormick addresses each argument against plague as the cause of Black Death. Regarding lack of rat-die-off in sources, this is probably because either the medieval chroniclers did not care to make a note of an enzootic, or because they did not have a specific word for rat. Rodents were rodents – most were described using the same general term. Therefore, historians need to look for general rodent mortality in the sources, not specifically rat mortality. By using this logic it is apparent that Justinian sources during the first pandemic do indeed report a massive enzootic of rodents.¹⁷

Archaeology and other scientific methods of analysis are important in several respects. DNA for *Y. pestis* has been found in three medieval and early modern plague pits. McCormick predicts that it will probably be found in Justinian-era plague pits as well. The archaeology of rats has also expanded to describe medieval and ancient rat populations.¹⁸ It has shown that as the medieval human population in the 14th century increased and land transport systems

¹⁴ Cohn, 738.

¹⁵ Michael McCormick, "Rats, Communications, and Plague: Toward an Ecological History," *Journal of Interdisciplinary History*, 34 (2003):1.

¹⁶ McCormick, 3.

¹⁷ McCormick, 4.

¹⁸ McCormick, 5.

increased connections between these populations, rat demographics also expanded. Rat bones have been found in locations where historians had claimed rats did not exist – thus invalidating their claims that those areas could not have been infected with the plague bacillus.¹⁹

Scientific study has also shown link between precipitation and plague, indicating that the history of climate is also important in the study of human history. Mathematical modeling has shown that plague can remain within a rat population for years without causing an epidemic. There is no need for an outside source to cause every outbreak of plague. Rather, changes in climate, ecology, and rat behavior can predicate local plague outbreaks.²⁰

Of all the historians thus far surveyed who have contributed to this debate, McCormick makes the best case both for the incidence of bubonic plague during the pestilence and the relevance of its identification to historical research. His research delves into questions of how the person and the environment interact with each other. Some historians, such as Ziegler, looked on rats as unnecessary to understanding the plague. Others acknowledged the role of rats in plague transmission, but did not give them anything more than a cursory examination. McCormick places the role of rats and rodents as central to the study of plague. He bases his research on scientific findings such as those of Raoult, but provides actual context and analysis to legitimize the use of those findings in a historical work.

Beyond written historical sources, archaeology and archaeozoology can glean historical evidence from ancient rat populations and demographics. Mathematical models can compute the likelihood of periods of plague outbreak and disappearance based on that evidence. Merely noting the presence of rats and moving on does not add to historical knowledge. McCormick's

¹⁹ McCormick, 14.

²⁰ McCormick, 24.

method not only adds to it, but paves the way for future usage of scientific methods to supply evidence for other historical questions.

Interestingly, since McCormick posted his study, Raffaella Bianucci et al. published “A rapid diagnostic test detects plague in ancient human remains.” The results of testing of plague pit remains from locations across France match the results of Raoult et al. It confirms that in the case of Martigues, 1720, *Y. pestis* caused the plague epidemic.²¹

Bianucci acknowledges that previous evidence, such as that found by Raoult, has been looked upon with suspicion by the historical community. She anticipates that her study will be regarded with the same attitude. More corroborating DNA evidence, she concludes, is necessary for historians to begin acknowledging the positive plague results. The acceptance of those results and the insight they bring to the historical community hinges on the attitudes of historians such as McCormick. Most historians display an unwillingness to entertain such evidence and a predilection towards doubting science and all that it may offer.

The historical debate over the epidemiology of the Black Death hinges on whether the modern bubonic plague is caused by the same pathogen that caused the medieval pestilence. This debate is an enlightening case study into the usefulness of archaeological and scientific information as contributing to historical evidence. The lived experience of the body can only be understood when it is taken to be a biological body, which continuously interacts with the pathogens, animals, and ecology that are part of its environment. Just as it is necessary to include human activity in the study of urban and rural rats, it is necessary to include rats in the study of human plague epidemics.

²¹ Bianucci, Raffaella. “Technical Note: A Rapid Diagnostic Test Detects Plague in Ancient Human Remains: An Example of the Interaction Between Archaeological and Biological Approaches (Southeastern France, 16th-18th Centuries),” *American Journal of Physical Anthropology*, 136 (2008): 361.

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