On the Importance of History

Daniel Curtis and Joris Roosen, *reply by* Monica Green

Article

LETTERS

Issue

VOLUME 4, ISSUE 2

In response to "Black as Death" (Vol. 4, No. 1).

To the editors:

It is fitting that the economic historian who has worked most diligently over the past decade to gain a masterful understanding of the paleoscientific work behind research into historical climate and infectious diseases, Bruce Campbell, is reviewed by the scholar with arguably the strongest grasp of both the literature on the scientific aspects of plague dynamics and its history as reconstructed through the documents. Many scholars can master one side; Monica Green is one of the few that have mastered both.

Green has a resoundingly positive view on what the natural sciences can provide to our (re)interpretations of historical development: "the sciences are documenting events that historians had not previously known"; "in assessing the impact of the Black Death, we too must look beyond the chronicles of Boccaccio, de Mussis, and al-Wardi"; and "a new kind of paleoscience can make up for a lot that humans cannot see." We agree. The history of plague can no longer be written solely with recourse to the historical documents. An increasing emphasis on the contribution of the natural sciences in the study of plague leads to the question of what the future role of historians might be. We argue that some fundamental parts of the plague enigma cannot be solved without the contribution of historians working with manuscripts. It is our contention that the importance of history and historical data will only increase from its use in conjunction with new forms of scientific evidence and methods. In this response to Green's review of *The Great Transition*, we highlight three vital contributions that can still be made by historians to the study of plague and other diseases of the past.

Dating the aDNA

Green writes that "neither ancient nor modern genomes have exact clocks," and that "they can tell time relationally, but not exactly." As a result, historical documents play a pivotal role in

contextualizing and dating the results attained by aDNA studies. Green states that one of the most important advantages of the complete sequence of *Yersinia pestis* retrieved from the East Smithfield cemetery in 2011 is that analysis of historical documents has provided a clear and unambiguous dating for the cemetery's creation and closure in 1349.[1] Several pioneering bioarchaeological studies, such as those by Sharon DeWitte et al., had earlier acknowledged the vital contribution of historians in dating the burials at East Smithfield to contextualize the findings from skeletal analysis.[2] Without the contribution of historians, the misdating of aDNA evidence remains an ongoing problem for genetics research. This was the case in the misattribution of the Bergen-op-Zoom genome to the initial Black Death outbreak in a prominent 2010 study,[3] or the London genome as a *Yersinia pestis* strain, which later turned out to be attributable to the *Pestis secunda*.[4] This is a problem that requires scholars, such as Green, with an excellent grasp of the techniques and evidence used in the natural sciences, as well as the sources and contextual demands from the field of history.

More than just dating existing aDNA evidence, historians can also point future genetics research in the right direction by tracking down plague burial grounds from historical documents. Given that aDNA is "rare, expensive to extract, and still challenging to sequence," the precise dating of burial grounds can save time and money for future research. History and the natural sciences should be used together to achieve results unattainable by one scientific discipline in isolation.[5]

Plague Diagnosis

In previous publications, we have pointed out the problems created by the uncritical use of long-term data on historical plague outbreaks by those working in the natural sciences.[6] Here we will focus on the issue of retrospective plague diagnosis. The identification of the Black Death as an outbreak of *Yersinia pestis* was the subject of intense debate among scholars until just a few years ago,[7] and was resolved definitively by advances in the laboratory with irrefutable proof that plague was indeed the culprit. Similar proof is lacking for the many recurrences of the disease across the Second Pandemic period, and the problem of disease diagnosis for plague outbreaks throughout the late medieval and early modern period has rarely been addressed.

Scholars seeking to link long-term plague activity with climatic, environmental, and societal variables accept plague diagnosis based on either outdated literature or, for the most part, narrative sources using direct anecdotal references. aDNA testing is currently unable to provide the same *deus ex machina* solution it did for the Black Death. The recurrence of Second Pandemic plague across space and time is simply too vast, too frequent, and the analysis is too costly to provide laboratory testing for each plague outbreak. Green notes that "to date, only thirty complete *Yersinia pestis* genomes have been sequenced from aDNA"—barely scratching the surface of all historical plague activity. The result is paradoxical. While the genetic science to identify plague has become more and more advanced, scientific research on the dynamics of long-term plague activity accepts plague diagnoses made in earlier studies without any formal testing or standardized identification and diagnostic tools. Claims in the preexisting literature were, in fact, sometimes made at a time when there was not even consensus over what caused the Black Death.

Since we cannot expect aDNA testing to provide a diagnosis for every potential plague outbreak, we need to find a way for history and the natural sciences to work in unison to provide a solution. For a number of plague outbreaks, we now have evidence that *Yersinia pestis* was the pathogen responsible. For these outbreaks, historical documents can be used to reconstruct the most significant epidemiological characteristics to provide a plague-diagnosis tool to assess suspected outbreaks for which no aDNA evidence is currently available. Historical documents, after all, are not limited to the few chronicles Green mentions in her review, but can also include quantifiable data on different dimensions of mortality that is, to a certain extent, standardizable over time for a locality or comparable between localities. When combined, these documents offer a wide range of epidemiological characteristics ranging from severity, spread, seasonality, household and neighborhood clustering, longevity, and selectivity along many different dimensions including sex, age, and socioeconomic status.[8]

Thinking Globally in Disease History

In this third and final section, we focus on Green's assertion that:

> if [Campbell] has been less successful in the epidemiological parts of this investigation, that is because the methods of thinking globally in disease history are not yet as advanced as they are in the field of climate history.

Although likely correct, we think this is partially explained by differences in the qualities of the data employed in climate research and disease research. Climate data is more suitable for a global approach, rather than simply not thinking globally. We concur regarding the intrinsic benefits of opening up the plague story to take us beyond very restricted parts of Europe, but we should also remember that the greatest advances in our knowledge may come from working at a variety of scales—global, macro, regional, and local. Historical sources are a fickle resource distributed unevenly across space and time. Even when relatively abundant, they are often difficult to interpret and reveal only small segments of the phenomenon under investigation. We should be careful, for example, not to dismiss further research into plague activity in Europe as simply Eurocentrism, but see it as connected to more practical and tangible concerns. Much of the source material needed to shine a light on the epidemiological outcomes—and the local pressures dictating spatial and temporal variability—are more likely to be furnished from selected case studies, often in restricted parts of Europe. How then, we should be asking, does one construct a global disease history? Especially when there is no global data source that allows for such an enterprise? It may turn out to be the case that while the global climate acts as a guiding framework for plague activity, the actual epidemiological outcomes are still frequently dictated by social or environmental developments at much more restricted scales—perhaps even at the micro level. We do not need to match global datasets of climatic indicators with equally global datasets on plague activity, because we will never have global epidemiological information using standardized sources, and the modes of transmission of plague will often depend on events and processes happening at the micro level. By exploring the possibilities and limitations of historical sources on a multitude of scales, we can build a foundation to solidify global disease history as a mature scientific field.

Daniel Curtis and Joris Roosen

Monica Green *replies*:

I am pleased that my review of Professor Campbell's important monograph, *The Great Transition*, has prompted such thoughtful responses. It is a sign of the speed of research that already since June, when my review appeared, new genetics evidence has appeared that further complicates the central question I raised: can climate be invoked as a causal factor in disease history if we are unsure of the timing or even the locus of the biological events we wish to explain?

Daniel Curtis and Joris Roosen remind us how scant the aDNA evidence currently is, and how many are the challenges that lie ahead in moving beyond the often-flawed data on which so much historiography (and now, even science) of plague demographics has been based. I agree with them that the rich documentary record in European archives will always be attractive in creating "thick descriptions" of plague outbreaks. But I also would want to reclaim the term "global" from the sense in which they have used it (uniform forces and uniform effects), and instead reassert the sense in which I intended it. Curtis and Roosen ask how "does one construct a global disease history ... when there is no global data source that allows for such an enterprise?" But that global (or, in the medieval world, hemispheric) data source is right in front of us: this single-celled, very slowly changing organism, *Y. pestis*, which could produce similar levels of mortality even when its vectors and hosts were different. There are indeed many questions about plague's epidemiology that we can only hope to reconstruct from the rich archives of northern Europe. But the scope for analogical analyses based on *Y. pestis*'s genetic structure and knowledge of its vectors and hosts is considerable. Plague's many histories are also emerging from sources that have long been used to reconstruct histories outside of Europe, such as oral traditions, which yield evidence of plague's early modern history in East Africa.9 Even environmental disruptions can serve as evidence of plague's effects, an observation that is likely to prove important as work moves beyond a narrow focus on the Black Death to investigations of the Second Plague Pandemic throughout both Eurasia and Africa.

The very great achievement of Professor Campbell's book is in proving that, if we are to explain these events, the table at which researchers gather must be longer and have more methodological options on the menu than we ever imagined before.

Daniel Curtis is a researcher at the Leiden University Institute for History.

Joris Roosen is a PhD student at Utrecht University and is affiliated to the Research Institute for History and Art History.

Monica Green is an American historian and Professor of History at Arizona State University.

1. Kirsten Bos et al., "A Draft Genome of *Yersinia pestis* from Victims of the Black Death," *Nature* 478, no. 7,370 (2011): 506–10. ↩
2. Sharon DeWitte and James Wood, "Selectivity of the Black Death with Respect to Preexisting Health," *Proceedings of the National Academy of Sciences of the United States of America* 105, no. 5 (2008): 1436–41; Sharon DeWitte, "The Effect of Sex on Risk of Mortality during the Black Death in London, A.D. 1349–1350," *American Journal of Physical Anthropology* 139 (2009): 222–34. Sharon DeWitte, "Sex Differentials in Frailty in Medieval England," *American Journal of Physical Anthropology* 143 (2010): 285–97. ↩
3. Misidentified in Stephanie Haensch et al., "Distinct Clones of *Yersinia pestis* Caused the Black Death," *PLOS Pathogens* 6, no. 10 (2010): e1001134. As Green notes in fn. 22 of her review, this can only be isolated to some point later in the second half of the fourteenth century. ↩
4. Dating from 1359–62, broadly speaking, with regionally different chronologies of mortality effects. Misidentified in Kirsten Bos et al., "A Draft Genome of *Yersinia pestis* from Victims of the Black Death," *Nature* 478, no. 7,370 (2011). Green notes this herself in fn. 21 of her review. ↩
5. As remarked upon recently in Sharon DeWitte and Maryanne Kowaleski, "Black Death Bodies," *Fragments* 6 (2017): 16. ↩
6. Joris Roosen and Daniel Curtis, "Dangers of Noncritical Use of Historical Plague Data," *Emerging Infectious Diseases* 24, no. 1 (2018): 103–10. ↩
7. John Thielmann and Frances Cate, "A Plague of Plagues: The Problem of Plague Diagnosis in Medieval England," *Journal of Interdisciplinary History* 37, no. 3 (2007): 371–93. ↩
8. A similar positive reappraisal of the value of historical documents for plague research appeared in Guido Alfani and Marco Bonetti, "A Survival Analysis of the Last Great European Plagues: The Case of Nonantola (Northern Italy) in 1630," *Population Studies*(2018), doi:10.1080/00324728.2018.1457794. ↩
9. Monica Green, "Putting Africa on the Black Death Map: Narratives from Genetics and History," special issue of *Afriques* (forthcoming). ↩