

Climate change and major plagues in the Roman period

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Abstract

This thesis examines the climate of the ancient Mediterranean from the second century BC to the sixth century AD, as well as major outbreaks of disease, with the principal aim of investigating potential explanations for apparent connections between climate change and the major pandemics of the Roman world. In addition, I consider the influence of the three great Roman “plagues” on society, assessing whether their effects, or lack thereof, are as apparent as scholars suggest.

Incorporating paleoclimatic and paleoenvironmental research into the study of ancient history and classical archaeology has become an important addition to the sphere of classical studies; as has the history of disease through the use of archaeogenetics and phylogenetics. These are the fields of science that I utilise in this thesis to explore possible relationships between climate and environmental change and outbreaks of the Antonine Plague (AD 165-190), the Plague of Cyprian (AD 251/2-270), and the first wave of the Justinianic Plague (AD 541-544). It becomes apparent that significant climatic and environmental variability probably contributed to the diseases of the Antonine and Justinianic pandemics reaching the Empire, while climate change does not appear to be linked with the Plague of Cyprian.

The debates concerning the impact of the three pestilences on Roman society continue to be vigorous. However, both exaggerated and understated interpretations of their effects dominate the histories of these pandemics, in spite of the evidence. This thesis approaches the question of impact with caution, by avoiding generalisations and focusing on case studies that depict local effects. I argue that impact should be assessed regionally, and that in many cases the consequences of the “plagues” are not at all clear.

Ultimately, the thesis provides a method to synthesise evidence from a range of historical and scientific disciplines with the aim of explaining how ecological calamities affected the Roman world.

Acknowledgments

My interest in history dates back to my youth; something about imagining how the people before us lived managed to keep my attention from primary school to secondary school. That interest evolved into fascination in undergrad at Columbia University. My love for the ancient world developed there, as I took ancient history courses, Greek, and Latin. But it was not until study under William Harris that the Romans became most important to me. His comprehensive and interdisciplinary approach to research – which at the time was new to me – and his constant investigations of longstanding hypotheses, has had a lasting impact on me. To William, and his passion for the Roman world, I owe a huge debt of gratitude.

My parents are equally important to my current position. My father, whose decades-long career as a dedicated educator has been a constant source of inspiration, provided much encouragement to pursue something I am truly interested in. My mother's continual support as we discussed the next stage of my academic path was essential to my success. Without their love and support, I might still be stuck in a nine-to-five job.

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Introduction

The influences of climate, environmental change and pandemics on the ancient world are relatively new but energetic topics in the realm of classical studies. The study of past climate and disease is ever more important today, as the global climate is changing at an unprecedented pace and the world is in the midst of the COVID-19 pandemic; that it is possible that our changing climate and environment played a role in the disease's emergence, and may still be playing a role in its transmissibility (both are still being investigated), make this thesis especially relevant.¹

Research on climate, environment and disease in the ancient world has gained momentum in the first two decades of the 21st century. Seminal studies focusing on the former, such as Horden and Purcell's *The Corrupting Sea*, Lukas Thommen's *An Environmental History of Greece and Rome*, William Harris' edited volume on *The Ancient Mediterranean Between Science and History*, Walter Scheidel's edited volume *The Science of Roman History: Biology, Climate, and the Future of the Past*, and the many 21st-century collaborative papers on the impact of climate and environmental change on ancient societies, have broadened the field, ushering in new methods in our approach to studying ancient history and archaeology.² Concentration on the emergences and effects of diseases on ancient societies has gained equal traction. With works such as Scheidel's *Death on the Nile: Disease and the Demography of Roman Egypt*, Lester K. Little's edited collection *Plague and the End of Antiquity: The Pandemic*

¹ Climatological, Meteorological and Environmental (CME) factors in the COVID-19 pandemic, virtual symposium August 4-6, 2020, outcome statement, <https://public.wmo.int/en/events/meetings/covid-19-symposium/outcomes>, date accessed: November 15th, 2020; Luterbacher *et al* 2020, 45.

² Horden and Purcell 2000; Thommen 2012; Harris 2013; Scheidel 2018.

of 541-570, Elio Lo Cascio's edited volume *L'impatto della "peste antonina"*, and Kyle Harper's *The Fate of Rome: Climate, Disease and the End of an Empire*, the study of infectious diseases as an element that helped shape the ancient world has been thrust to the forefront of classical studies.³ Yet, in the many works concerning ecological calamities of the ancient Mediterranean, there has not been an in-depth study focusing on the interrelationship of these factors – i.e. on the possible influence of climate change on the rise and spread of pandemics in the classical world, let alone a review of that question for the dramatic shifts in climate that coincide with the three pandemics of the Roman period.

The question has been broached in some of the works above, particularly *Plague and the End of Antiquity* (for the Justinianic Plague) and the more recent *The Fate of Rome* (mainly the Justinianic Plague), as well as in several 21st-century articles, most notably an article by Peregrine Horden in 2005 (the Justinianic Plague), an article by Benoît Rossignol and Sébastien Durost in 2007 (the Antonine Plague), a paper by Timothy Newfield in 2018 (the Justinianic Plague), and an article by Richard Duncan-Jones also in 2018 (the Antonine Plague).⁴ But beyond asking questions, pointing out general broad connections and suggesting likelihoods, or positing indirect scenarios – such as the increasing nutritional deficiency brought on by a changing climate that may leave populations more susceptible to disease – we do not have studies that investigate, biologically, the potential influence of significant climatic variability on the major Roman pestilences.

³ Scheidel 2001; Little 2007; Lo Cascio 2012; Harper 2017.

⁴ Horden 2005, 153; Rossignol and Durost 2007; Newfield 2016; Duncan-Jones 2018.

A few months before the submission of this thesis, Jürg Luterbacher and colleagues published their article, “Past pandemics and climatic variability across the Mediterranean”, in which they claim to “explore potential associations between pandemic disease and climate over the past 2,500 years in Mediterranean history”.⁵ Their paper focuses little on exploring links, and looks more at the ancient climatic picture, suggesting that there are many periods with significant climatic events but no pandemics, which complicates potential investigation. They also suggest that a reason why there have not been more studies on the links between ancient climate and disease is that, for most of the pandemics, the diseases have not been identified scientifically. However, there need not be a pandemic for every climatic event, and we should not halt investigation into links between climate and pandemics because we do not know for certain which disease was the cause. If we want to truly investigate, we should focus on further exploring the pandemics based on the evidence afforded to us by ancient authors. With their help diagnosing the diseases, we can investigate, biologically and epidemiologically, whether there may be a link with climate change.

That is exactly what this thesis does. It explores the possibility that the diseases related by ancient authors were in fact influenced by climatic events and change. It focuses on the three Roman pandemics that we know most about through the written evidence and archaeology: the Antonine Plague (AD 165–possibly 190); the Plague of Cyprian (AD 251/2–270); and the first wave of the Justinianic pandemic (AD 541–544). It should be noted that use of the term “plague” for the Antonine and the third-century pandemic do not denote actual plague (e.g. bubonic

⁵ Luterbacher *et al.* 2020.

plague). The word was used by ancient writers as a term for any widespread outbreak of disease. Its continued use in scholarship is more traditional than it is precise. The sixth-century Justinianic pandemic, however, was actual plague; confirmed without doubt in 2013 when ancient DNA (aDNA) analysis of skeletal remains from a mass grave in Germany returned results positive for *Yersinia pestis*, the bacterium that causes plague (Chapter 4).⁶ It makes investigation of a link between climate and disease for this pandemic a little easier. However, based on the written evidence, the other two plagues do not appear to be plague pandemics. The Antonine Plague, discussed in Chapter 2 is probably an ancestor of the modern smallpox virus, while the Plague of Cyprian, the topic of Chapter 3, appears to have been a viral haemorrhagic fever (VHF) – most likely an ancestor of Ebolavirus (EBOV). Thus, without scientific confirmation, some of the analysis within this thesis is, of course, speculative. However, as the succeeding chapters prove, it is highly likely that the pandemics described by ancient authors in the second and third centuries were the diseases above. Before aDNA analysis confirmed that the Justinianic pandemic was true plague, the written evidence pointed to bubonic plague as the cause. This bodes well for our diagnostic skills as historians of disease. Hopefully, archaeogenetics will continue to improve so that we may scientifically confirm the pandemics of the second and third century. Until then, our working hypotheses, I think, are very good ones.

The other important aspect of this thesis, detecting past climatic change, is not simple or straightforward. However, to this point we have come a long way in paleoclimatology (the study of past climate), so that our trust in climatic

⁶ Harbeck *et al.* 2013.

reconstructions and pinpointing past climatic events is solidifying; especially because many climatic proxies cohere with one another. There are also some that do not cohere, and that suggests either yet-to-be-understood complications in certain climatic proxies (possibly caused by past anthropogenic forcing), or notable variations between micro-climates. The latter notion is something we must keep in mind, as the ancient Mediterranean world *was not* one grand climatic region in which Roman provinces reacted to climatic forcing such as volcanic eruptions in the same way. Similarly, we should not extrapolate a climatic reconstruction from one region to another, such as applying to Egypt a tree-ring-width record that serves as a proxy for temperature for central Europe. It is also important to note the difference in precision amongst the various proxies – for example, tree-ring-width series typically have much higher resolution (annual-to-decadal) than glacier-fluctuation data (century-scale) – as well as recognising the complexities of a micro-climate and how that may or may not be displayed well in a climatic reconstruction (e.g. speleothems of northern Turkey). Fortunately, our data for the planet's principal climate forcing mechanism, volcanic eruptions, are highly resolved, and cohere with important climate reconstructions.⁷ Here, precision of the climate history is key, as I must investigate correlations between regional climatic trends with the origins and the paths of three pandemics.

As crucial as the climate history and the historical evidence for disease are the scientific studies on the effects of climatic factors and season on the diseases most likely responsible for the pandemics. Unfortunately, these studies are not many – we have more for one disease (bubonic plague) than the others – but from the studies

⁷ Sigl *et al.* 2015; Büntgen *et al.* 2016; McConnell *et al.* 2020a.

available we can glean the information needed to determine what role climate change may have played in the rise and spread of the diseases during the Roman period. For smallpox, the disease probably responsible for the Antonine pandemic, there are 20th-century studies on how the virus reacts in various ambient temperatures, in different rainfall regimes, and in a range of percentages of humidity, as well as studies on the seasonality of the disease.⁸ However, because the virus was eradicated in 1980, such investigation has ceased, and in an age where laboratory research has been improving rapidly. For the probable cause of the Plague of Cyprian, viral haemorrhagic fever, there was almost no research on the potential relationship between climate and the forms of VHF relevant to this thesis until 2017, when a major study with a multitude of authors was published on the form most likely connected to the third-century pandemic: Ebola virus.⁹ Fortunately for scholarship on the Justinianic Plague, there are numerous studies concerning the effects of climatic variability on bubonic plague, the form probably responsible for the majority of sixth-century plague cases.¹⁰ It is by using these scientific studies on diseases with the studies on past climate that I propose, where possible, potential explanations of an apparent connection between climate change and the Roman pandemics.

This thesis begins with a chapter that is a focused climate history of the Roman period from 200 BC until AD 600. We begin by reviewing the proxies for detecting past climate and climate-forcing mechanisms. The focus then turns to the

⁸ Rogers 1926; Rogers 1928; Downie and Dumbell 1947; MacCallum and McDonald 1957; Huq 1976; Fenner *et al.* 1988; Nishiura and Kashiwagi 2009.

⁹ Dudas *et al.* 2017.

¹⁰ Parmenter *et al.* 1999; Hinnebusch, Fischer and Schwan 1998; Hinnebusch *et al.* 2002; Stenseth *et al.* 2006; Ari *et al.* 2011; Schmid *et al.* 2015; Hinnebusch, Jarrett and Bland 2017.

Roman period starting with the second century BC, for a climatic picture of the Mediterranean world during the so-called Roman Climate Optimum (200 BC-AD 150), and showing how climate became more turbulent in the latter half of the second century AD. The chapter then homes in on the periods that precede and coincide with each pandemic (AD 150 – 200; AD 245 – 270; AD 530 – 550). These sections use all available climatic proxies to illustrate, regionally, how climate looked before and during the three pandemics, particularly around the areas where the written evidence indicates the disease first reached the Empire.

Each of the three succeeding chapters is devoted to the three pandemics in chronological order. Chapter 2 takes a thorough look at the Antonine Plague, surveying the relevant scholarship and debates up to 2020. A discussion of the written evidence follows, in which I gather all accounts from ancient authors who are most likely talking about the second-century pestilence. The written evidence helps us define the chronology of the Antonine pandemic, suggests something of its geographical extent, relates symptomology which is our principal source for diagnosing the pestilence, and aids in assessing its impact on the Empire. The chapter then moves to analyse diagnoses of the pandemic, looks at the phylogenetic history of the probable disease, and discusses important epidemiological characteristics. Next is a section on the influence of climate on the suspected disease, in which I use the scientific studies discussed above to elucidate whether climate can affect the incidence of the virus. The last section of the chapter focuses on archaeology that may be connected to the Antonine Plague, and what it might tell us

about the impact of the second-century pandemic, weighing in on a debate that has been active since Gibbon.¹¹

Chapter 3 investigates the Plague of Cyprian, with a structure that differs slightly from the preceding chapter. This is due to the much more limited scholarship on the third-century pandemic. Given that Kyle Harper's article from 2015 is the only comprehensive study of this pestilence, in which he is the first to gather all of the sources relevant to the Plague of Cyprian, the ancient written evidence and the modern historiography are discussed together in the same section. The next section examines the archaeological and numismatic evidence that seem to be connected to this pandemic. The section that follows is an analysis of symptomology and epidemiology, concluding with a tentative diagnosis of the pestilence. Then I investigate whether climatic factors could have influenced the disease that appears to be responsible for the Plague of Cyprian, and whether third-century climate change may have had any bearing on its rise and spread. Lastly, the chapter considers the plague's impact and its relationship to the "crisis of the third century".

The last and longest chapter is devoted to the Justinianic Plague; longest because the disease responsible has been confirmed and we have the most evidence for it. There is much to relate on the historiography, as research and scholarship on the sixth-century pandemic have been plentiful, and the debates most lively. There is also a fair amount of detailed ancient written evidence from regions throughout the Empire that needs examining. The archaeological evidence is far more rewarding than the previous chapters, as it has provided confirmation of the identity of the

¹¹ Gibbon 1806, vol. 4, ch. 43.

disease and its presence throughout Europe in the sixth century. A discussion on the phylogenetic history follows, during which important aspects of the plague's evolution are revealed. Given that the plague cycle has been well studied, there is much to analyse in terms of epidemiology, which includes a review of reservoirs and vectors. The crux of the chapter, an assessment of the influence of climate on the plague cycle, is the penultimate section, in which I examine scientific studies on the climate/plague link and discuss how it may relate to sixth-century climate change and the pandemic's first wave. In the final section, there is a discussion of the Justinianic Plague's impact on the Empire in the scope of a case study, and whether we can disentangle its effects from the damage caused by sixth-century climate change.

What becomes apparent upon reading this thesis is that nothing is universal. Mediterranean climate is not similar between regions or centuries, the diseases that caused the pandemics all seem to be different and each affected the Empire in a unique way. Although, in two of the cases we can trace close chronological conjunctions between climate change and the outbreak of pandemics, and even suggest plausible biological and epidemiological causation, climate change did not influence the rise and spread of all three of the great Roman plagues, despite what some broadly suggest.¹² In an age where research on climate change and disease, including the influence of climatic change on disease, is as important as it has ever been, investigating these questions through the lens of the past may prove to be useful for future research. Of course, climate change and disease affected the Roman world in ways different from how these issues are transforming the modern world.

¹² Harper 2017.

However, the afflictions that such ecological calamities brought to the Romans are important to the history of climate, disease, and the ancient world, and fully understanding how these extraneous pressures shaped their world may help us better understand ours.

Roman Climate: 200 BC – AD 600

Introduction

This chapter presents a climate history for the Roman period from the second century BC to the sixth century AD, giving both a macro view of the general climate under which the Romans lived from 200 BC – AD 150, and focusing on climatic trends surrounding each of the three major plagues (the Antonine Plague: AD 160 – 190; the Plague of Cyprian: AD 250 – 270; and the first wave of the Justinianic Plague: AD 530 – 550). We will observe what the Romans were accustomed to climatically, as well as the periods of climatic variability preceding and coinciding with each epidemic, which might have led to biological vulnerability for parts of the Empire. While we should keep in mind that the Empire is notable for its “stability and relative warmth”, this is with respect to the entire Holocene epoch, which had a few millennial phases of extreme climatic change.¹³ The Roman period had its bouts of damaging climatic variability too, both economically and demographically-speaking. Here we examine these climatic events and trends and consider that some coincided with the worst epidemics the Empire had seen.

There are a number of notable years, decades, and even centuries where the Empire underwent significant climatic shifts that strayed from the norm; major temperature shifts occurring in the mid-1st century BC, the latter half of the second century AD, the late third/early fourth century AD, and in the first half of the sixth century AD are a few that stand out. The Roman Warm Period, recently re-named

¹³ Harper and McCormick 2018, 21.

the Roman Climate Optimum (RCO), was a 350-year period of relative climatic stability lasting from 200 BC to AD 150.¹⁴ While one major temperature anomaly does occur near the middle of these three and a half centuries – mid-1st century-BC volcanic events – many regions experienced warmer and wetter conditions for the majority of this period.¹⁵ This phase was far from ubiquitous in the Empire, but, on the whole, much of the Roman world did benefit from advantageous conditions. That began to change in the latter half of the second century AD, as temperatures and precipitation in central Europe, central Asia, Anatolia, the Near East, and central and eastern Africa fluctuated, in some cases quite dramatically. Roman climate rebounded in the early third century, but again deteriorated later in the century. From there, in general, climate was unstable, with the Empire experiencing its greatest shock following a string of sixth-century volcanic eruptions that helped generate the Late Antique Little Ice Age or LALIA (AD 536 – 660). The LALIA coincided with the early Islamic conquests and the contraction of the Eastern Roman Empire.

This chapter will first review climate forcing mechanisms potentially responsible for balance and disruption during the Roman period, followed by an overview of the steadiest climatic phase (the RCO), and finally the climatically turbulent post-RCO phase (AD 150 – 660), with a particular focus on the intervals surrounding each major plague.

¹⁴ Harper and McCormick 2018, 25.

¹⁵ Büntgen *et al.* 2011; Büntgen *et al.* 2016; Sigl *et al.* 2015, 544-545; McConnell *et al.* 2020a; Oppenheimer 2020; Strunz and Braeckel 2020; McConnell *et al.* 2020b.

Climate Forcing from 200 BC – AD 600

What can cause unfavourable climatic changes across the globe? The two most important natural external climate-forcing mechanisms are solar activity and volcanism.

Solar activity

For solar activity the effect is relatively straightforward: less solar activity usually coincides with cooler temperatures on Earth.¹⁶ One way of measuring solar activity is by quantifying radionuclides ^{14}C (Carbon-14, or radiocarbon) and ^{10}Be (Beryllium-10) in natural archives like tree rings and ice cores respectively.¹⁷ When cosmic ray particles enter the Earth's atmosphere they react with atmospheric nitrogen, oxygen, and argon, creating ^{10}Be and ^{14}C in the atmosphere.¹⁸ These isotopes are eventually stored in polar ice sheets and tree rings, helping us measure Total Solar Irradiance – the rate at which solar energy reaches the earth's surface. Production of ^{10}Be and ^{14}C is reduced with greater solar activity (an inverse relationship), therefore lower levels of ^{10}Be and ^{14}C are indicative of warming, while an increase in the radionuclides represents a cold phase.¹⁹

In Figure 1, the values gathered from ^{14}C archives in trees from South Germany, Ireland, California and Washington State and modelled to demonstrate global solar activity (TSI) show that TSI rose early in the second century BC, just after the start of the so-called RCO.²⁰ More importantly, however, the ^{14}C data show

¹⁶ Gray *et al.* 2010, 2-6; Beer, Mende and Stellmacher 2000, 403-405.

¹⁷ Steinhilber *et al.* 2012, 5967-5968; Gray *et al.* 2010, 2-4; Harper and McCormick 2018, 18.

¹⁸ Steinhilber *et al.* 2012, 5967.

¹⁹ Gray *et al.* 2010, 2-4; McCormick *et al.* 2012, 210-212; Harper and McCormick 2018, 18.

²⁰ Stuiver *et al.* 1998, 1044; Vieira *et al.* 2011; Harper and McCormick 2018, 18-20.

stability for most of the RCO. In the early second century AD we see a notable decline in solar activity followed by a quick recovery.²¹ A much more substantial decrease is apparent in the third century, falling to its lowest mark since the fourth century BC in the AD 250s. From there, TSI fluctuates significantly for the rest of the Roman period, aside from a relatively stable span from the mid-sixth to the early-seventh centuries AD (Fig. 1).

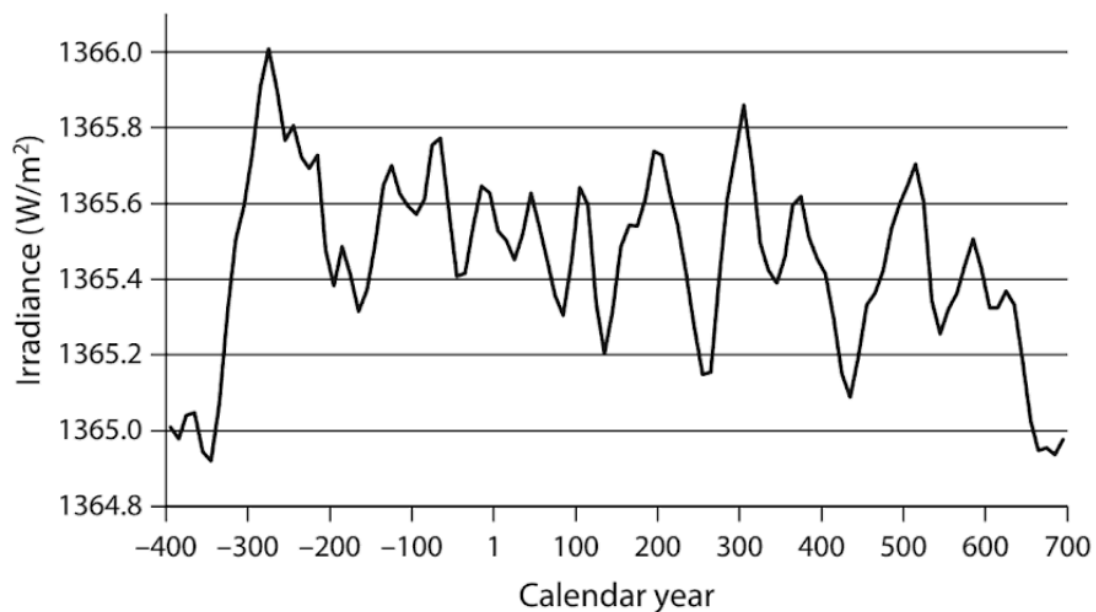


Figure 1. Total Solar Irradiance measured from cosmogenic isotope ^{14}C (radiocarbon). Data: https://www2.mps.mpg.de/projects/sun-climate/data/tsi_hol.txt. Figure: (Harper and McCormick 2018; Fig. 1.2)

The ^{10}Be data gathered from ice cores from Greenland and Antarctica (Fig. 2) tell a similar story. Total Solar Irradiance is *very* stable during the first two centuries of the Roman Climate Optimum. There is a drop in TSI at the turn of the millennium, followed by a quick rise and even larger second drop, all in the first century AD.²² Solar activity climbs again, remaining stable for nearly all of the second century AD. TSI takes a considerable fall in the first half of the third century,

²¹ Vieira, Solanki, Krivova and Usoskin 2011; Harper and McCormick 2018.

²² F. Steinhilber, Beer and Fröhlich 2009; Harper and McCormick 2018, 18-20.

dropping to the second-lowest point of the series in Roman times at around AD 250. But TSI begins to rebound in the latter half of the century. Like the ^{14}C data, the ^{10}Be

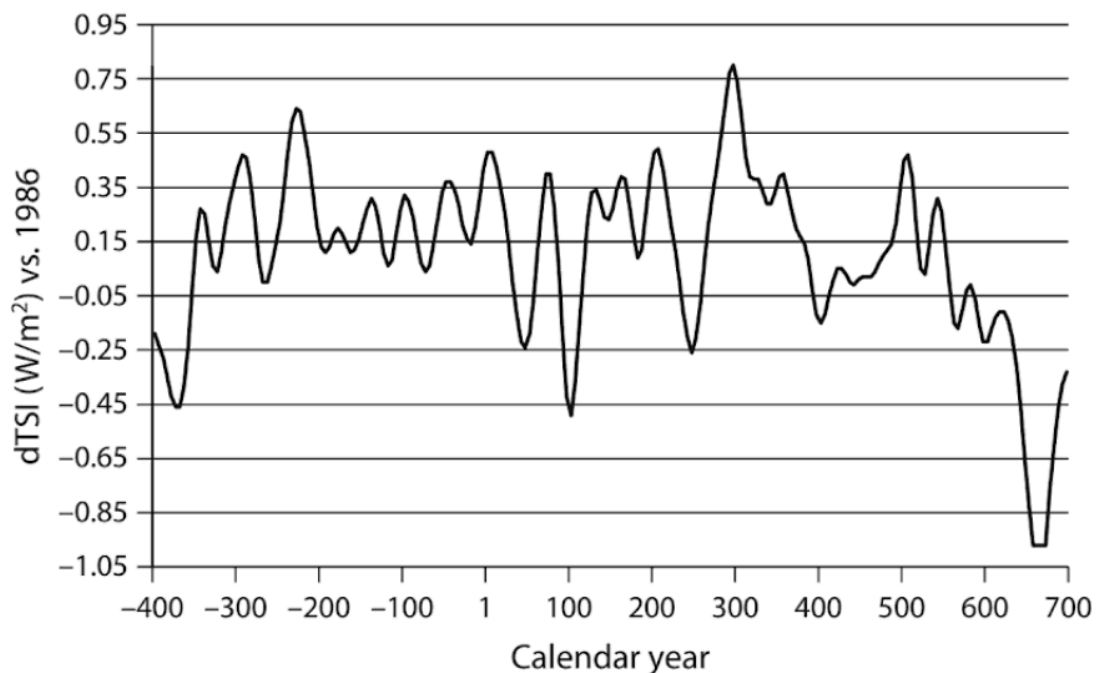


Figure 2. Total Solar Irradiance measured from cosmogenic isotope ^{10}Be . Data from: ftp://ftp.ncdc.noaa.gov/pub/data/paleo/climate_forcing/solar_variability/steinhilber2009tsi.txt. (Figure: Harper and McCormick 2018; Fig 1.2)

data demonstrate high levels of TSI for the fourth century AD. We see a decline at the turn of the century, with moderate stability until AD 600, when solar activity plummets to the lowest levels of the series.

The ^{14}C and ^{10}Be data displaying total solar irradiance show broad similarity throughout the Roman period. Solar activity during the Roman Climate Optimum was stable until around the late-first/early-second century AD. Both data sets show large reductions in TSI in the third century, high activity in the fourth century, moderate levels in the late-fifth and sixth centuries, and a massive reduction in the seventh century.

Volcanic activity

Volcanism is another common climate-forcing mechanism that disrupts the earth's radiative equilibrium. Historically, large volcanic eruptions have often been the cause of notable cooling phases, sometimes felt globally. After an eruption, sulphur is released into the stratosphere with the volcano's plume. The sulphur combines with water to create sulphuric acid and sulphate particles. These sulphate aerosols can remain in the stratosphere for around 4 years, having a reflective effect against the sun, which in turn cools the planet. The post-eruption cooling effect is evident in comparing the evidence for historic volcanic activity and corresponding summer temperatures revealed through tree-growth data (more on tree-ring data below). Fifteen of the sixteen largest reductions in tree-growth between 500 BC and AD 1000 came less than one year after a notable volcanic eruption (figures 3 and 4).²³ But how do we detect and measure past volcanic eruptions? The post-eruption sulphuric aerosols are key to pinpointing historic volcanic activity. After multiple years in the stratosphere, the aerosols eventually fall and are preserved in ice at Greenland and Antarctica.²⁴ To find them, scientists drill ice cores near the poles and later scan them for traces of sulphates. The sulphur isotope levels in each annual layer of ice can indicate the intensity and location of past eruptions. Global volcanic aerosol forcing (GVF) is a measurement (W/m^2) of the intensity of past eruptions based on the sulphate signal in the ice-core layers. Eruptions detected in ice at both poles are usually given a higher GVF rating, since aerosols from the eruption reached both

²³ Sigl *et al.* 2015, 546-547.

²⁴ Stothers 2002, 1; Zielinski 1995, 20937-20940; Sigl *et al.* 2015, 543-544;

ends of the earth, hence its “global” forcing.²⁵ Tephrochronology is another method used to pinpoint the location of past eruptions. Tephra – chemical material produced by a volcanic eruption – is unique to a volcanic field, and thus

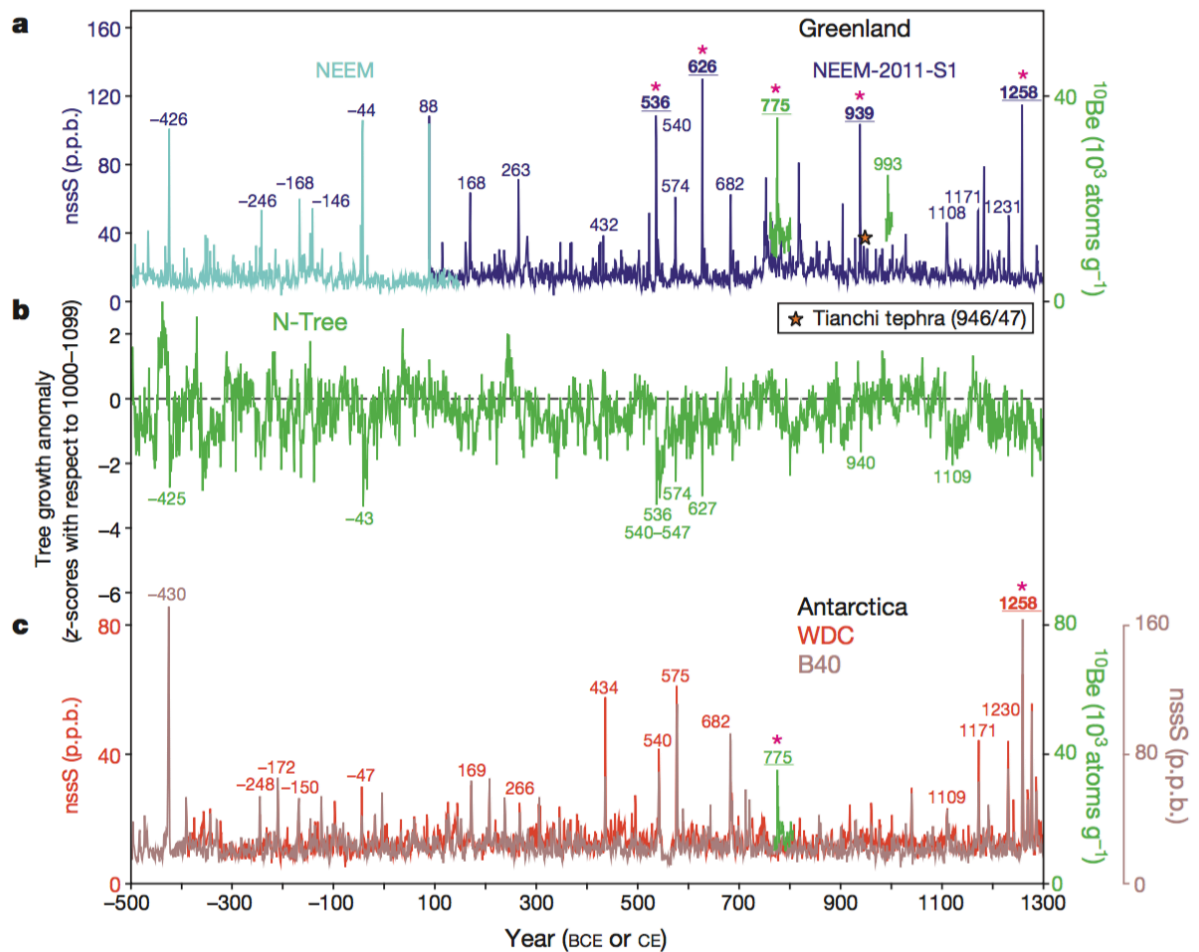


Figure 3. Volcanism from 500 BC – AD 1300: **a)** Non-sea-salt sulphur records from the Greenland NEEM and NEEM-2011-S1 ice cores; **b)** Tree-ring growth anomalies (we will hear more about the tree-ring records later in this chapter); **c)** Non-sea-salt sulphur records from Antarctic WDC ice core. (Sigl et al. 2015, Fig. 2)

²⁵ Despite the GVF rating, eruptions with lower GVF values can still have devastating regional effects climatically. In the case of the AD 79 eruption of Vesuvius, Pompei and Herculaneum were directly affected by the volcano’s molten rock and hot ash, but the region was not severely affected indirectly by climate-forcing, as the eruption seems not to have had long-term climatic consequences.

acts as a volcano's "fingerprint" geologically and, if the eruption is large enough that tephra reaches the poles, it can sometimes be detected in the annual layers of ice cores.²⁶ It is with these methods that scientists create a volcanic record.

The timing of eruptions in antiquity is now clear in the volcanic record, but this was not always the case. Back in 1994, a study led by Gregory Zielinski used sulphate data from the Greenland Ice Sheet Project 2 ice core (GISP2) to create a record of volcanism going back to 7000 BC.²⁷ Scholars had been referencing this volcanic record for twenty years until a major change came. In 2015, Michael Sigl led

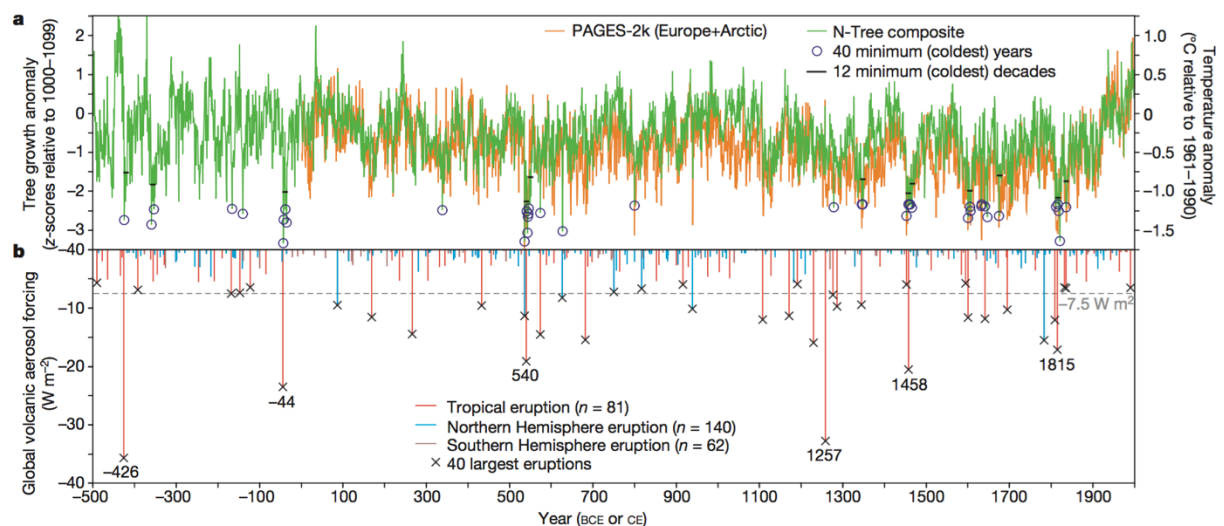


Figure 4. **a)** 2500 years of tree-ring growth anomalies; **b)** Global volcanic aerosol forcing from bipolar sulphate composite records. (Sigl et al. 2015, Fig. 3)

a study that recalibrated the ice-core chronologies and succeeded in matching them to the tree-ring chronologies, finding that the eruption chronology was off by 5-10 years.²⁸ Exploiting the recent discovery of two short-lived, sharp increases in cosmogenic nuclides in trees and ice cores in the late first millennium AD that provide unambiguous tie points between the two archives, as well as the much more

²⁶ Lowe and Alloway 2015, 4-7.

²⁷ Zielinski *et al.* 1994; Zielinski 1995.

²⁸ Sigl *et al.* 2015.

detailed ice-core chemical records produced by modern analytical systems, Sigl and his collaborators derived new ice-core chronologies for both Greenland and Antarctica. The result was that ice-core and tree-ring chronologies aligned during the first millennium, which led to re-dating many eruptions.

The new volcanic record from the study led by Sigl shows that during the Roman Climate Optimum there were relatively few eruptions compared to other periods; and from the eruptions we do see, few seem to have had a large global impact.²⁹ Between 200 BC and AD 150, the noteworthy sulphate signatures we see are at: 168 BC, 146 BC, 43 BC, and AD 88 in the Greenland ice cores; and at 172 BC, 150 BC, and 47 BC in the core from Antarctica (figures 3 and 4).³⁰ The eruption with highest GVF during the RCO is the 43 BC eruption, now known to be from the Okmok volcano in Alaska, from which sulphuric aerosols reached both poles; however, the high GVF number is due more to its massive signal in the Northern Hemisphere (the second largest signal on record for the Northern Hemisphere after an Icelandic eruption in AD 1783).³¹ More importantly, aside from the 43 BC event, and possibly the AD 88 eruption, there was very little globally-significant volcanic activity during the RCO (Table 1); and there was virtually no serious activity between 42 BC and AD 88 (figures 3 and 4).

In the post-Roman-Climate-Optimum phase (post-RCO), between AD 150 and 200 the Greenland cores register a notable eruption at 168, and the Antarctic core has a signal at 169 (Fig. 3). These signals are from the same eruption. However, more

²⁹ Sigl *et al.* 2015.

³⁰ Although the years do not exactly match up, some of the sulphate signals between the Greenland and Antarctic cores could be from the same eruption; aerosols might have reached the poles at slightly different times, which is partly why there is a suggested error tolerance of ± 2 .

³¹ Sigl *et al.* 2015, extended data table 4; McConnell *et al.* 2020.



Figure 5. The location of two major volcanic eruptions of the Roman period. Scientists believe the volcano Ksudach, in far eastern Russia, was responsible for the eruption attested in ice cores at around AD 163; while scientific analysis has confirmed that Ilopango was the eruption that occurred at AD 540. Other eruptions of the Empire are yet to be attributed. The Roman Empire (ca. second century AD) is shaded red. (Map courtesy of Andrew Wilson)

recent data show that that eruption date should be moved to around 170, and, further, there is another eruption happening 6-7 years earlier around 163.

First, some history on the detection of these two eruptions. In the 1994 eruption chronology, Zielinski listed two eruptions happening early in the second half of the second century. The first in AD 152 (± 2) was assigned to Ibusuki, Japan, the location of a volcanic field that includes the volcano Kaimondake. The second was logged at AD 161 (± 2) and attributed to the volcano Ksudach in southern Kamchatka (far eastern Russia).³² However, new ice-core data and Sigl et al.'s recalibration of the ice-core chronology moved the 152 (± 2) eruption to around AD 161, and Sigl's analysis of the signal led the scientists to suggest that it was probably

³² Zielinski *et al.* 1994, 949-952; Zielinski 1995, 20939. Their attribution of volcanic events is based on the historical volcanic record and sampling analysis done by Simkin *et al.* 1994.

the Ksudach volcano in Russia – given the strong unipolar signal in the ice cores and Ksudach’s position as a high-latitude volcano (Fig. 5) - while they moved Zeilinski’s AD 161 eruption to 169 and tagged it as an “unattributed event.”³³ But this was not the last adjustment to these two eruption dates.

After discussion with the third author of Sigl et al. 2015, ice core scientist Joe McConnell, he showed me his new team’s most recent modifications to the Greenland ice-core chronologies based on annual layer counting of new high-resolution chemical measurements in the NGRIP2 (North Greenland Ice Core Project) core from north-central Greenland. Evaluation of this new chronology against tree-ring measurements suggests <2 year dating uncertainty throughout the Common Era.³⁴ He linked the GIPS2 and GRIP (Greenland Ice Core Project) timescales with the new NGRIP2 ice core timescale by comparing electrical conductivity data with his team’s NGRIP2 non-sea-salt-Sulphur data. This produced a new age-scale that differs by roughly ten years from Zielinski’s 1995 record. He then used the new age scale to re-date the data from another Greenland ice core, NEEM-2011-S1 from Sigl et al. 2015, as seen in Figure 6 (note that the two “x” axes for the ice cores are slightly offset). On the NGRIP2 age scale (the bottom axis of Figure 5), which McConnell favours – due to the more recent analysis of NGRIP2

³³ Michael Sigl pers. comm; Sigl et al. 2015, 543-549 and the supplemental materials: their article focuses mainly on addressing the adjustment of the ice core chronologies, but with particular focus on a series of eruptions in sixth century CE that contributed to the Late Antique Little Ice Age (536 to about 665 CE). However, the supplemental materials lists all of the volcanic events (spikes in sulphate), including those from the second century. Upon discussion with Michael Sigl, he explained that the signal for the early 160s eruption fits with the Ksudach volcano in Russia. He and Joe McConnell (third author) clarified that the late 160s eruption is probably a low-latitude volcano, but because sulphate from its plume registered near both poles at Greenland and Antarctica, it is difficult to identify, hence the “unattributed event” tag.

³⁴ McConnell *et al.* 2018, 5726-5731.

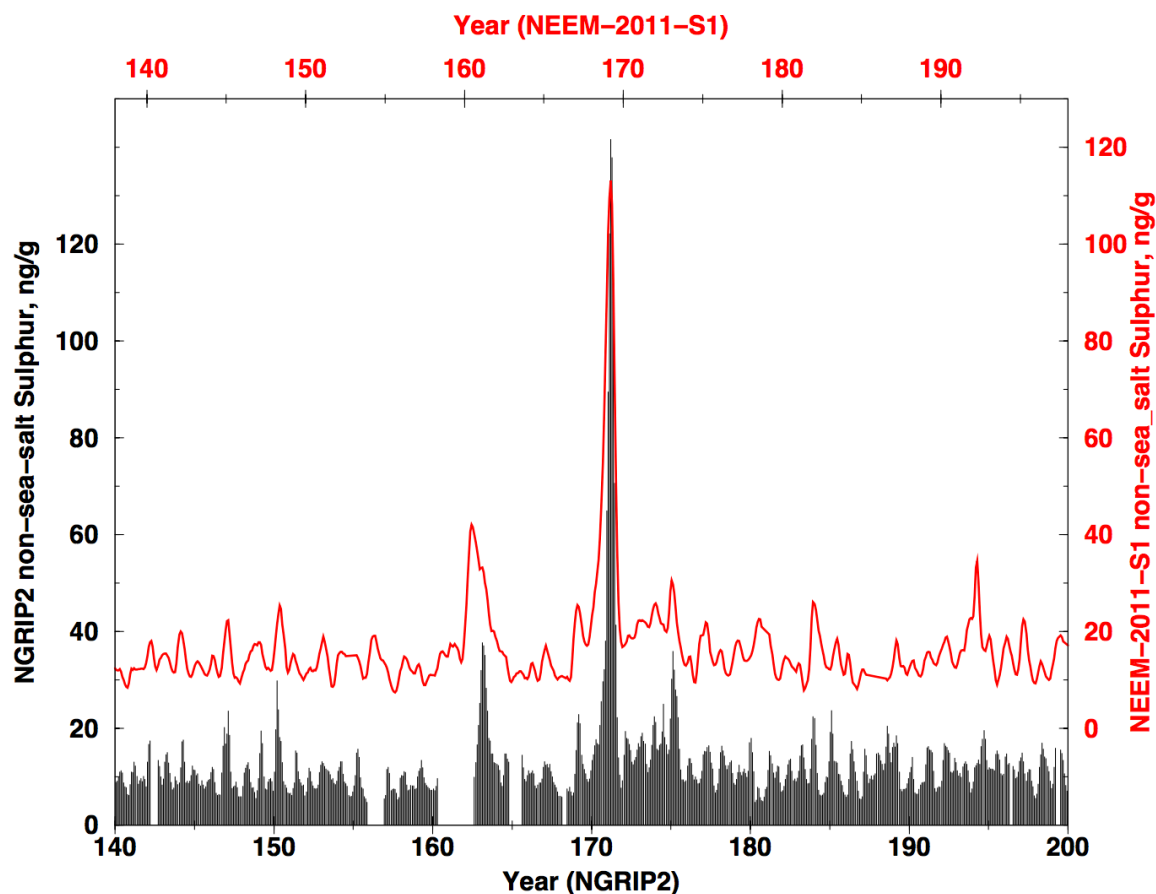


Figure 6. Sulphur measurements ($\sim$ monthly) from Greenland ice cores NGRIP2 (bottom “x” axis) and NEEM-2011-S1 (top “x” axis). Note that the two “x” axes are slightly offset. Data from: McConnell *et al.* 2018: NGRIP2; and Sigl *et al.* 2015: NEEM-2011-S1. (my figure)

using updated high-resolution chemical measurements – an eruption occurs around 163, while a second eruption happens at 170; the NEEM-2011-S1 scale (the top axis) puts both eruptions slightly earlier but the differences are well within the uncertainties of Sigl *et al.*’s NEEM-2011-S1 age scale. Thus, we now have current and near-precise age markers for two post-150 eruptions: an eruption around AD 163, probably from the Ksudach volcano in far eastern Russia, and another, larger eruption happening around 170, the location of which is still unknown.

For the third century AD, there is a significant signal at 263 in the Greenland cores, and one at 266 in the core from Antarctica.³⁵ These are probably two different

³⁵ Sigl *et al.* 2015, Extended Data Table 4.

eruptions. However, it is possible that error tolerance might be the cause of a signal lag, accounting for the three-year difference between core signals, even though Sigl and company suggest only a ± 2 -year error tolerance. Nevertheless, whether this is a single eruption, or two consecutive eruptions, Sigl and company list the signal at AD 266 as the eleventh largest eruption of the past 2500 years; enough to cause cooling in the later third century.

We see no notable sulphate signals for the fourth century, and one seemingly massive eruption for the fifth century (the 22nd largest of the past 2500 years), at 432 in the Greenland cores and 434 in the Antarctica core. From then it is quiet, until a devastating series of eruptions in the first half of the sixth century.

The first eruption occurs at AD 536, more visible in ice from the Northern Hemisphere than the Southern Hemisphere, but still registering the eighteenth highest GVF value in the past 2500 years (Table 1); this eruption remains unattributed.³⁶ A second eruption occurs five years later at 540. This eruption was even larger, with a high sulphur signal in both Greenland and Antarctica, amounting to the fifth largest eruption in Sigl et al.'s chronology (Table 1 and figures 3 and 4); the eruption was recently attributed to the Ilopango volcano in El Salvador (Fig. 5).³⁷ A third, less powerful eruption happened seven years later at 547. While not significant on its own, the eruption in 547, combined with the events at 536 and 540, probably contributed further to a phase of severe climatic variability across the globe; more on this below. In the later sixth century, we see a signal at around 574 at

³⁶ Sigl *et al.* 2015, 543-545.

³⁷ Sigl *et al.* 2015, Extended Data Table 4; Dull *et al.* 2019: via tephra analysis and radiocarbon dating of subfossil trees that were buried by the eruption, the scientists confirmed Ilopango as the source of the 540 eruption.

both poles, that was the tenth largest eruption since 500 BC.

The data show that the Roman period was not brimming with volcanic activity, but it was not exactly lacking it either. The three-and-a-half-century RCO phase was relatively quiet, aside from the 43 BC and AD 88 eruptions; based on the tree-ring data, the former appears to have been far more serious than the latter (Fig. 4 and Table 1). The ice cores show a few smaller eruptions in the early second century BC, but no other notable spikes in sulphur in those 350 years. The second half of the second century AD was not packed with eruptions either, but two events

Rank	Year	Volc. SO ₄ ²⁻ Greenland (kg km ⁻²)	Volc. SO ₄ ²⁻ Antarctica (kg km ⁻²)	Global forcing* (W m ⁻²)	Cold year	N-Tree (z scores; 1000-99)	T _{Europe/Arctic} (°C; 1961-90)	Volcano†/ Region
1	-426	99.8	78.2	-35.6	-425	-2.74		UE -426
2	1258	90.4	73.4	-32.8	1258	-1.43	-0.91	Samalas/Indonesia
3	-44	100.6	15.4	-23.2	-43	-3.33		Chiltepe?/Nicaragua
4	1458	39.0	63.6	-20.5	1459	-2.31	-1.03	Kuwaie/Vanuatu
5	540	61.2	34.4	-19.1	541	-2.57	-1.48	Ilopango?/El Salvador
6	1815	39.7	45.8	-17.1	1816	-2.51	-1.55	Tambora/Indonesia
7	1230	56.4	23.1	-15.9	1230	-1.71	-0.65	UE 1230
8	1783	135.8		-15.5	1783	-1.16	-0.97	Laki/Iceland
9	682	38.4	38.7	-15.4	682	-0.95	-0.96	Pago?/New Britain
10	574	38.3	34.1	-14.5	574	-2.46	-0.94	Rabaul?/New Britain
11	266	61.0	11.3	-14.5	268	-1.70	-0.72	UE 266
12	1809	34.6	25.4	-12.0	1810	-2.18	-1.23	UE 1809
13	1108	48.3	11.6	-12.0	1109	-1.99	-1.15	UE 1108
14	1641	44.2	14.9	-11.8	1641	-2.31	-1.19	Parker/Philippines
15	1601	39.2	18.7	-11.6	1601	-2.62	-1.50	Huaynaputina/Peru
16	169	39.1	18.4	-11.5	170	-0.80	-0.94	UE 169
17	1171	37.0	19.5	-11.3	1171	-0.91	-0.88	UE 1171
18	536	99.0		-11.3	536	-3.36	-1.74	UE 536
19	1695	28.6	22.5	-10.2	1696	-1.63	-1.28	UE 1695
20	939	88.7		-10.1	940	-1.81	-1.44	Eldgjá/Iceland
21	1286	27.6	20.8	-9.7	1288	-1.49	-0.65	Quilotoa?/Ecuador
22	433	20.6	27.2	-9.6	432	-0.45	-0.25	UE 433
23	87	83.1		-9.5	87	-0.22	-0.49	UE 87
24	1345	27.9	19.1	-9.4	1346	-2.18	-1.48	El Chichon?/Mexico
25	626	72.2		-8.2	627	-3.00	-0.93	UE 626

Table 1. The 25 most powerful volcanic eruptions from 500 BCE – 2000 CE based on Global Volcanic Forcing (GVF), or “Global forcing”, from sulphate measurements in ice cores from Greenland and Antarctica. Note the separate columns for sulphate measurement in Greenland cores and Antarctic cores. (Sigl *et al.* 2015, Extended Data Table 4)

not seven years apart (at 163 and 170) had the potential for climatic disruption (Fig. 6). The third and fifth century each had a sizeable eruption, at 262 and 434. The sixth century was the Roman period's most volcanically-active century, with large eruptions at 536 and 540, a smaller eruption at 547, and another large eruption at 574. It is safe to say that the Roman period was not a "quiet" volcanic period. But of the whole period, the RCO was the quieter span, and it is here that we will begin investigating climatic trends throughout the Roman world.

200 BC – AD 150: the "Roman Climate Optimum"

The period from 200 BC – AD 150, today referred to as the "Roman Climate Optimum" (RCO), was a stretch of generally favourable climatic conditions lasting from the late Republic to the early Empire.³⁸ While the RCO is not represented universally across the Roman world, many regions in the Empire experienced warmer and wetter conditions during this period.³⁹

Tree-ring data

Tree rings are typically regarded as the best source of paleoclimatic data, owing to the high-resolution chronologies dating they provide.⁴⁰ If both the tree's growth and its surrounding environment are well understood, tree rings can be used as a high-precision climate proxy for annual temperature and precipitation.

³⁸ In the space of 6 years, Michael McCormick and Kyle Harper revised their range for the "Roman Climate Optimum," affirming in 2012 that the period of stability spanned 100 BCE – 200 CE, but amending it in 2018 to 200 BC – AD 150, noting the apparent instability starting in 150 CE for both parts of the Empire: Harper and McCormick 2018, 34-35; McCormick *et al.* 2012, 174-175.

³⁹ Büntgen *et al.* 2011; Büntgen *et al.* 2016; Nicolussi and Patzelt 2000; Göktürk 2011.

⁴⁰ Hughes 2002, 96-97; Harper and McCormick 2018, 24.

The tree-ring data show a relatively stable climatic span for most of the so-called Roman Climate Optimum. Tree-ring-width data from Alpine conifers (temperature) and oaks (precipitation) from the Alps in central Europe demonstrate a warm and wet 350 years (Fig. 7), with sudden but relatively

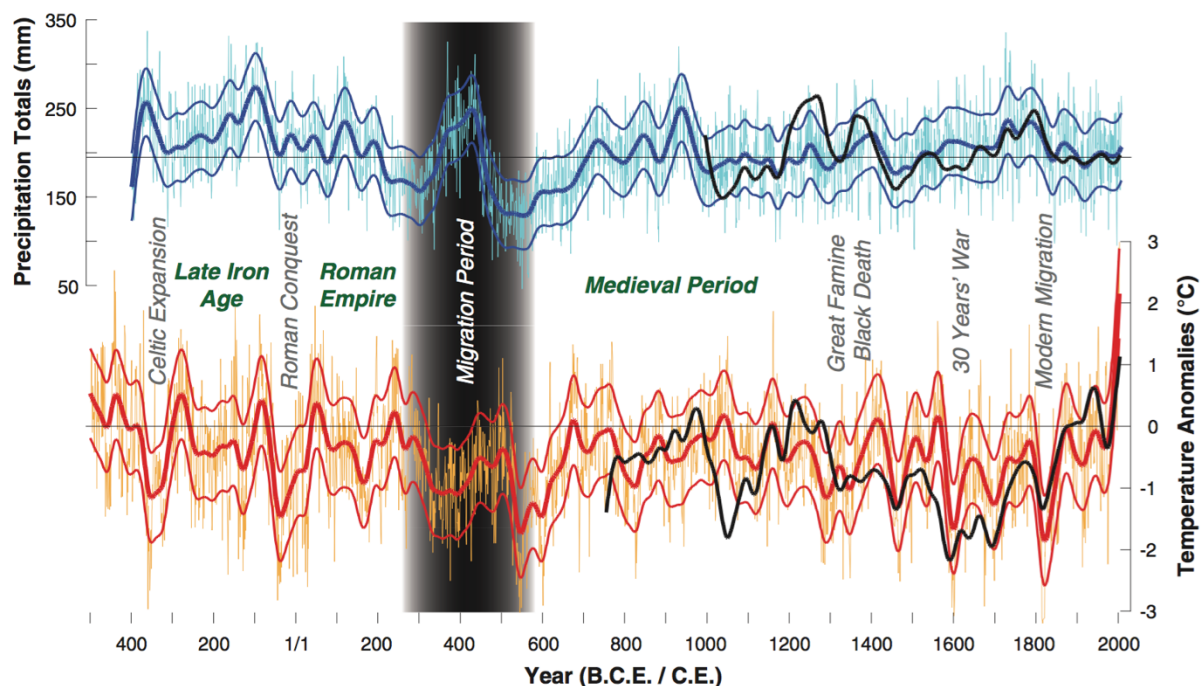


Figure 7. Tree-ring data from central Europe showing both precipitation (top) and temperature (bottom). (Büntgen *et al.* 2011, Fig. 4)

brief reductions in temperatures and precipitation around the mid-first century BC, probably caused by the eruption at 43 BC.⁴¹ The RCO ends in central Europe with notable reductions in temperatures at the midway point of the second century AD. Unfortunately, additional tree-ring data (Siberian larch) from the Russian Altai mountains in central Asia – a proxy just for temperature – only go back as far as AD 100.⁴² However, like the data from central Europe, the central Asian data show higher temperatures in the first century AD, until a sharp drop in temperatures – the largest of the Altai series – occurs in the AD 160s (Fig. 8).

⁴¹ Büntgen *et al.* 2011, 581.

⁴² Büntgen *et al.* 2016, 233.

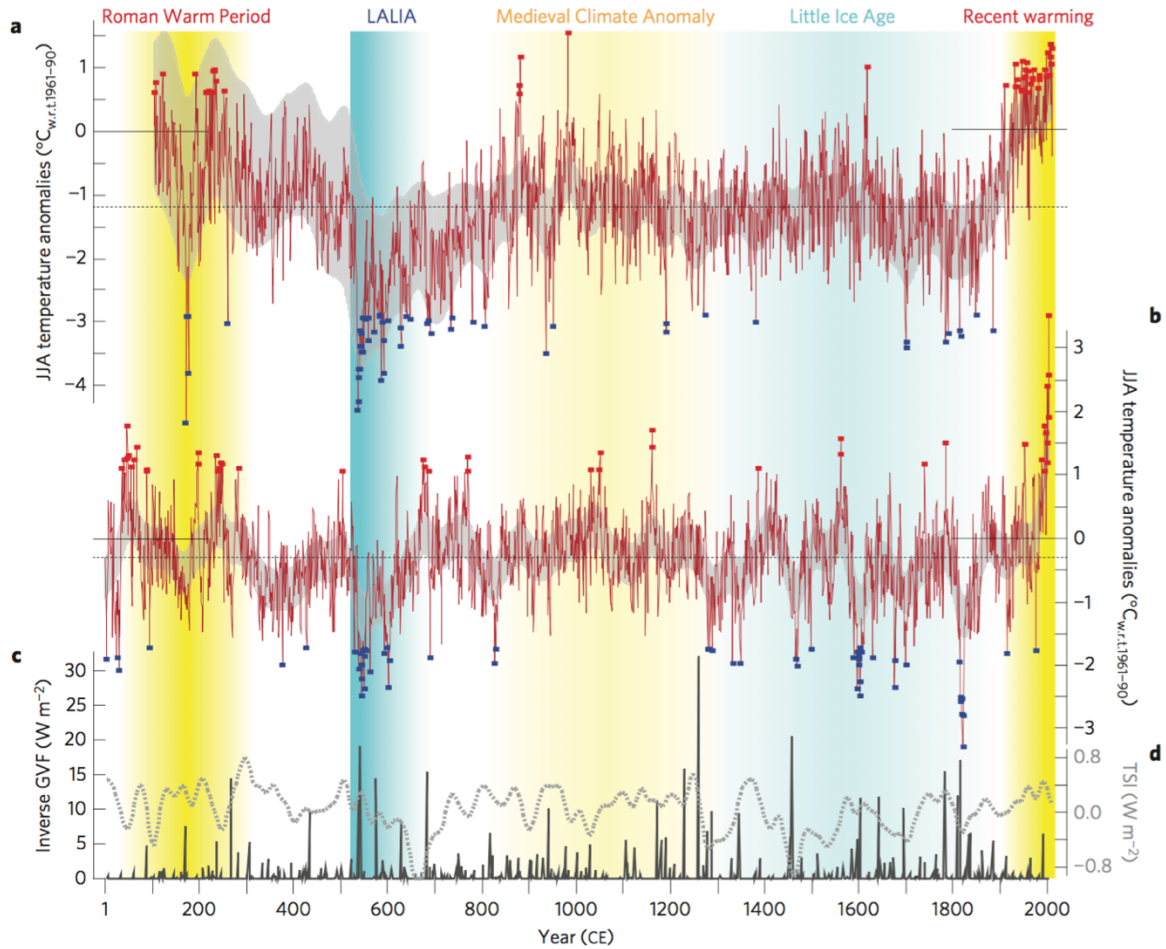


Figure 8. **a**: Tree ring-based temperature chronology from the Russian Altai in central Asia. **b**: Data from the European Alps. **c** and **d**: Show specific volcanic eruptions based on evidence from ice-core data. Note the massive drop in temperatures in the mid-second century for the Russian Altai data. (Büntgen *et al.* 2016, Fig. 2)

Tree-ring (*Cupressus dupreziana*) data representative of precipitation from the Wadi Tanezzuft in the northwest Sahara in Libya show stability from 200 BC until AD 150; however, the period between 50 BC – 1 BC/AD 1 is missing from the record (Fig. 9). ENSO-sensitive tree-ring data starting at 1 BC / AD 1 from New Zealand and New Mexico exhibit moderate warmth and wetness for the second half of the RCO (Fig. 10). These data are used to infer Eurasian and central Asian climatic trends based on two sets of data that are not from our focus region; a proxy for a proxy, so to speak. The data are in fact quite far from the focus region – New Mexico

further than New Zealand – and typically show trends initiated by the El Niño Southern Oscillation, which have been shown to be similar to trends in Eurasia.⁴³ While events like volcanic eruptions can affect the ENSO cycle, it is not guaranteed that they will, and climatic studies suggest ENSO-sensitive data are far less predictable than post-volcanic cooling observed directly at or near the source, which is in most cases visible in climatic data the year after an eruption.⁴⁴ Thus, we should keep reliability in mind when using such inferential data. Notwithstanding, the stable period exhibited from this data beginning at 1 BC / AD 1 lasts until the second half of the second century AD, when a cooler and drier period begins and lasts until the 180s.⁴⁵

Overall, the tree-ring data mostly demonstrate warmth and wetness for the Roman Climate Optimum – aside from a substantial reduction in temperatures following the 43 BC eruption, apparent in the Alps, with a relatively quick recovery. The Russian Altai and European Alps data show that the RCO ended in the latter half of the second century AD, as does the Saharan data, while the ENSO-sensitive data from New Zealand and New Mexico show loss of climatic stability earlier in the second century AD for Eurasia and central Asia.

⁴³ McCormick *et al.* 2012, 190-191.

⁴⁴ Khodri *et al.* 2017; Sigl *et al.* 2015.

⁴⁵ Fowler *et al.* 2008; Diaz, 2000 #312; Diaz and Markgraf 2000; McCormick *et al.* 2012, 193.

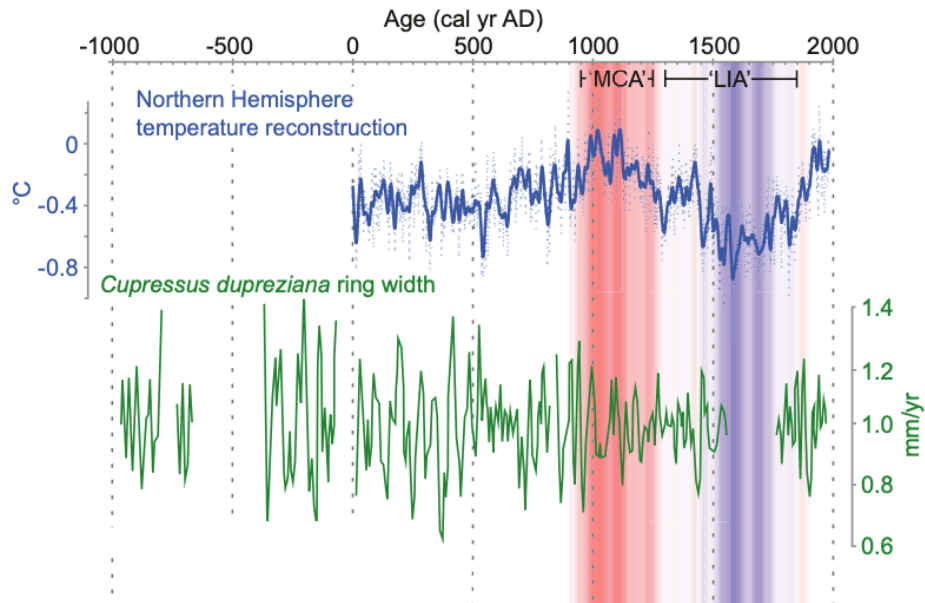


Figure 9. Comparison of Northern Hemisphere temperatures and *Cupressus dupreziana* ring-width data from the Libyan Sahara that is representative of precipitation. (Nash *et al.* 2016, Fig. 3)

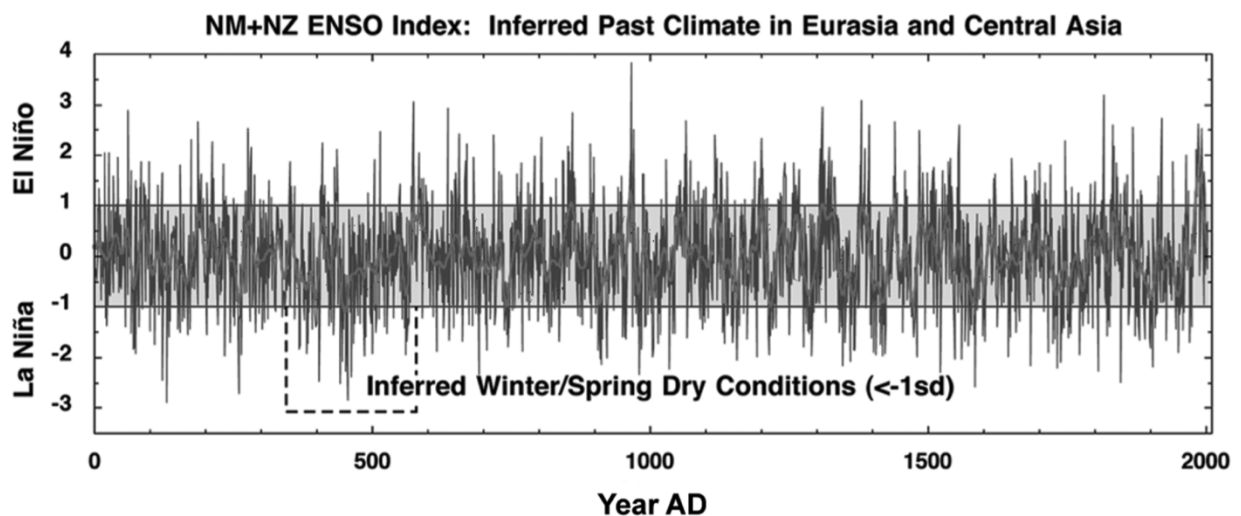


Figure 10. ENSO-sensitive tree-ring data from New Mexico and New Zealand that can be used to infer past climate in Eurasia and Central Asia. (McCormick *et al.* 2012, Fig. 9b)

Stalagmites

Stalagmites are cave formations produced over time by the infiltration and accumulation of calcium carbonate-rich cave drip waters, usually from rainfall or

snow-melt seepage.⁴⁶ By measuring stable isotopes of oxygen and carbon in stalagmite samples we can infer a region's historic precipitation and temperature trends. Fortunately, we have a number of stalagmite records that stretch back to antiquity, particularly for the so-called RCO. However, most stalagmite archives are not as highly resolved as tree-ring reconstructions, and they are only useful for paleoclimatic purposes if the complexities of the environmental parameters in the cave's surrounding region are well understood.⁴⁷

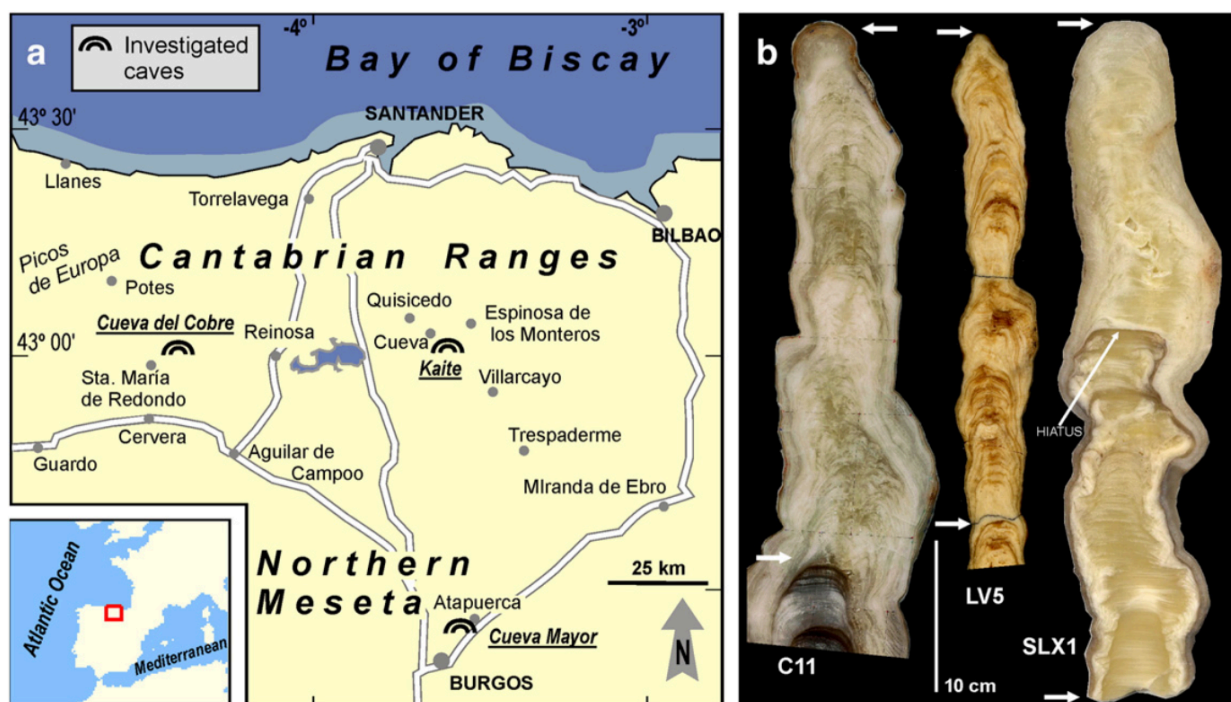


Figure 11. a) The location of the caves Cueva del Cobre, Kaite, and Cueva Mayor in Spain; b) Stalagmite samples. (Martín-Chivelet et al. 2011, Fig. 1)

Beginning in the northwest Empire, a 4000-year-long high-resolution carbon isotope ($\delta^{13}\text{C}$) record from northern Spain reveals relative warmth and stability during the Roman Climate Optimum.⁴⁸ With $\delta^{13}\text{C}$ data (in this case a proxy for land surface temperature changes) combined from three caves – Kaite, Cueva del Cobre,

⁴⁶ Göktürk 2011, 5.

⁴⁷ Harper and McCormick 2018, 27.

⁴⁸ Martín-Chivelet et al. 2011, 1-12.

and Cueva Mayor Cueva⁴⁹ (Fig. 11) – the record shows a rise in warmth at 550 BC, followed by seven centuries of stability until the early third century AD (Fig. 12). In an unrelated study, Cruz and colleagues also analysed a speleothem from the Kaite Cave, measuring the Mg/Ca of calcite from the stalagmite, which they regard as a proxy for precipitation. Their data show stable precipitation trends for much of the RCO (and into the seventh century AD), with reductions in the first century BC (Fig. 13).⁵⁰

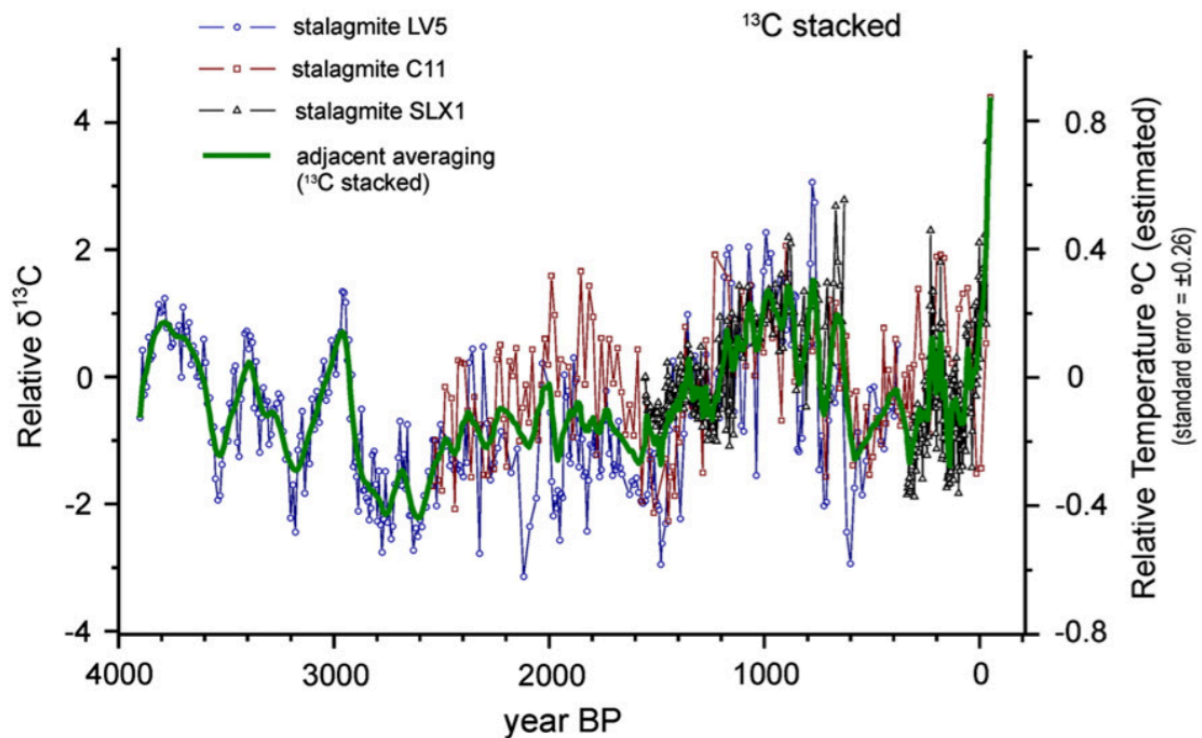


Figure 12. A $\delta^{13}\text{C}$ combined stalagmite record from Kaite (LV5), Cueva del Cobre (C11), and Cueva Mayor (SLX1) caves in northern Spain. (Martín-Chivelet *et al.* 2011, Fig. 7)

⁴⁹ The Cueva Mayor stalagmite record begins in the fourth century AD and thus should not be included in our reconstruction until then.

⁵⁰ Cruz *et al.* 2015, 7750.

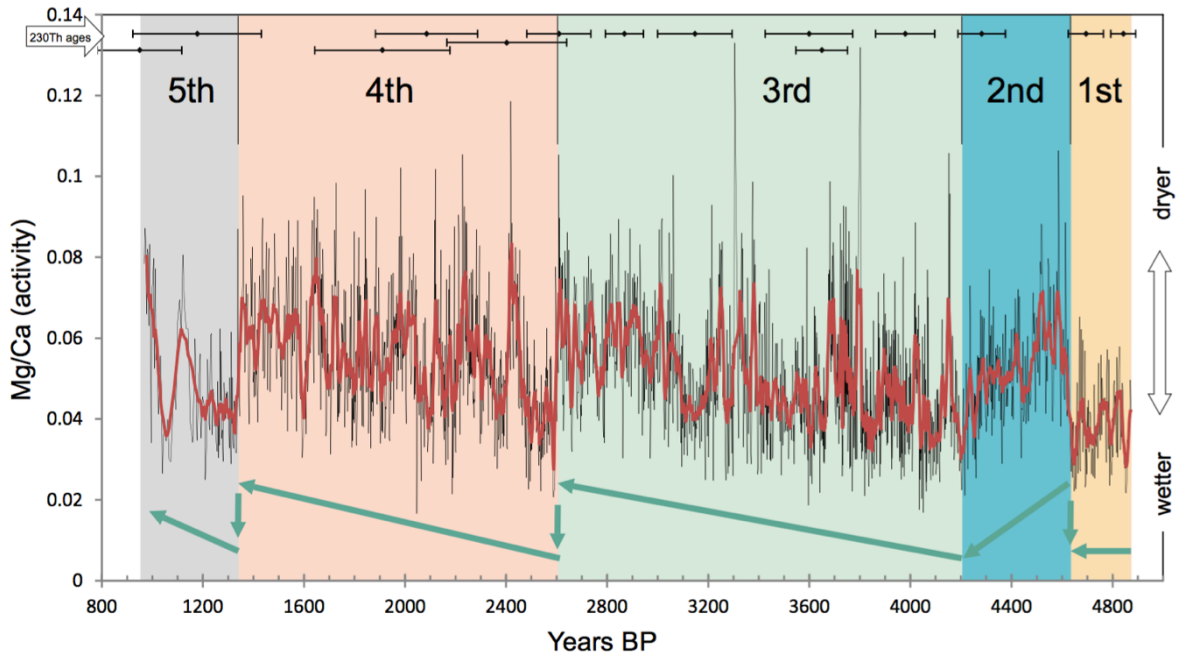


Figure 13. Stalagmite isotope data from Kaité Cave, Spain, showing moisture trends for northern Iberia. Our period of focus extends from 2150 yr BP (200 BC) to 1250 yr BP (AD 700). (Cruz *et al.* 2015, Fig. 6)

Moving northeast and slightly outside of Roman bounds, lower-resolution data from a stalagmite within the Coral Cave (Korallgrottan) in Sweden (Fig. 14) exhibit stable trends in both temperatures and precipitation from 200 BC – AD 150 (figures 15 and 16).⁵¹ Besides a rise in warmth at 200 BC, there are no other notable rises or drops in temperatures or precipitation observed from these data during the RCO. $\delta^{18}\text{O}$ isotope data from the Spannagel Cave in the Austrian Alps show a similar situation for central Europe.⁵² The Spannagel stalagmite record, demonstrating temperature trends back to 100 BC, shows few noteworthy climatic shifts from 100 BC – AD 150, aside from a rise in temperatures in the late first century BC, followed by a more gradual decline in the first century AD (Fig. 17). The Spannagel stalagmite record corresponds well with the nearby tree-ring data from the Alps (Fig. 7), in that both display stable temperature trends for much of the RCO.

⁵¹ Sundqvist *et al.* 2010, 77-86.

⁵² Mangini, Spötl and Verdes 2005, 741-751; McCormick *et al.* 2012.

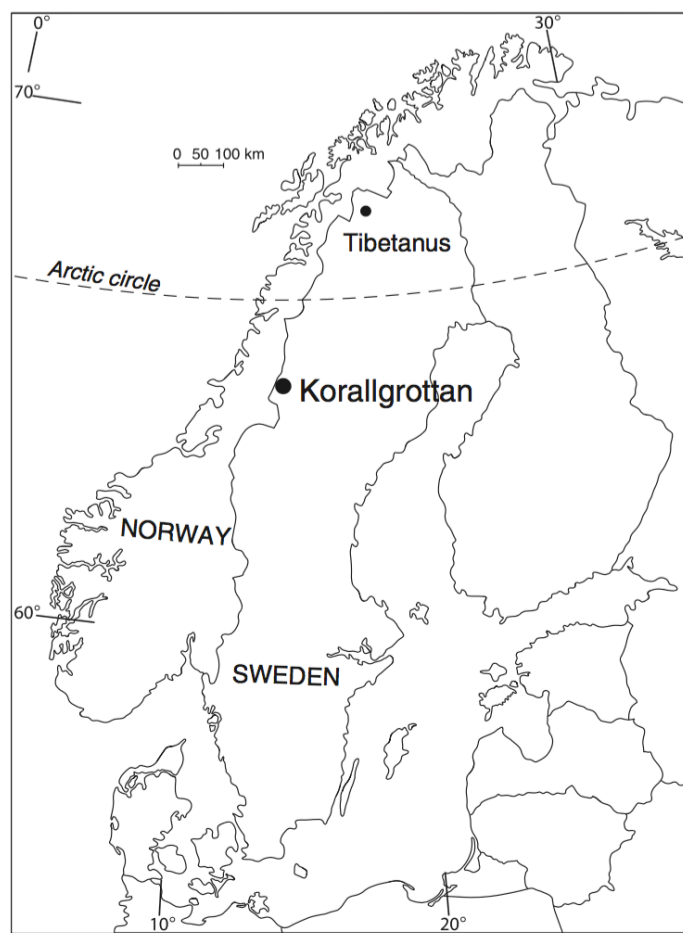


Figure 14. The location of Coral Cave (Korallgrottan). (Sunqvist et al. 2010, Fig. 1)

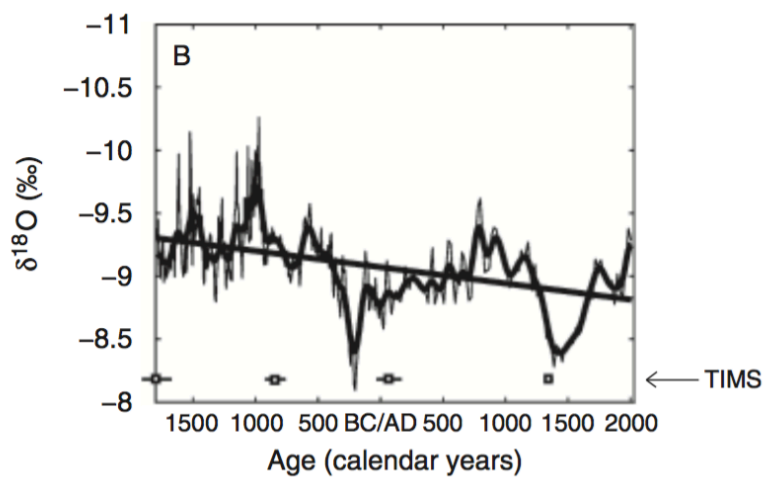


Figure 15. $\delta^{18}\text{O}$ values, a proxy for temperature in this case, from a stalagmite in Coral Cave, Sweden. (Sunqvist et al. 2010, Fig. 2b)

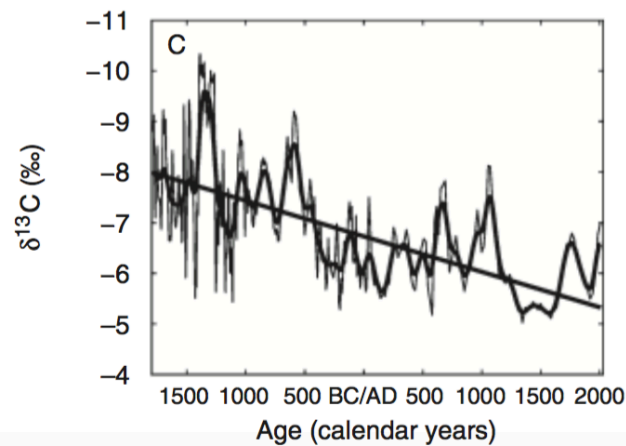


Figure 16. $\delta^{13}\text{C}$ values, a proxy for precipitation, from Coral Cave, Sweden. (Sunqvist et al. 2010, Fig. 2c)

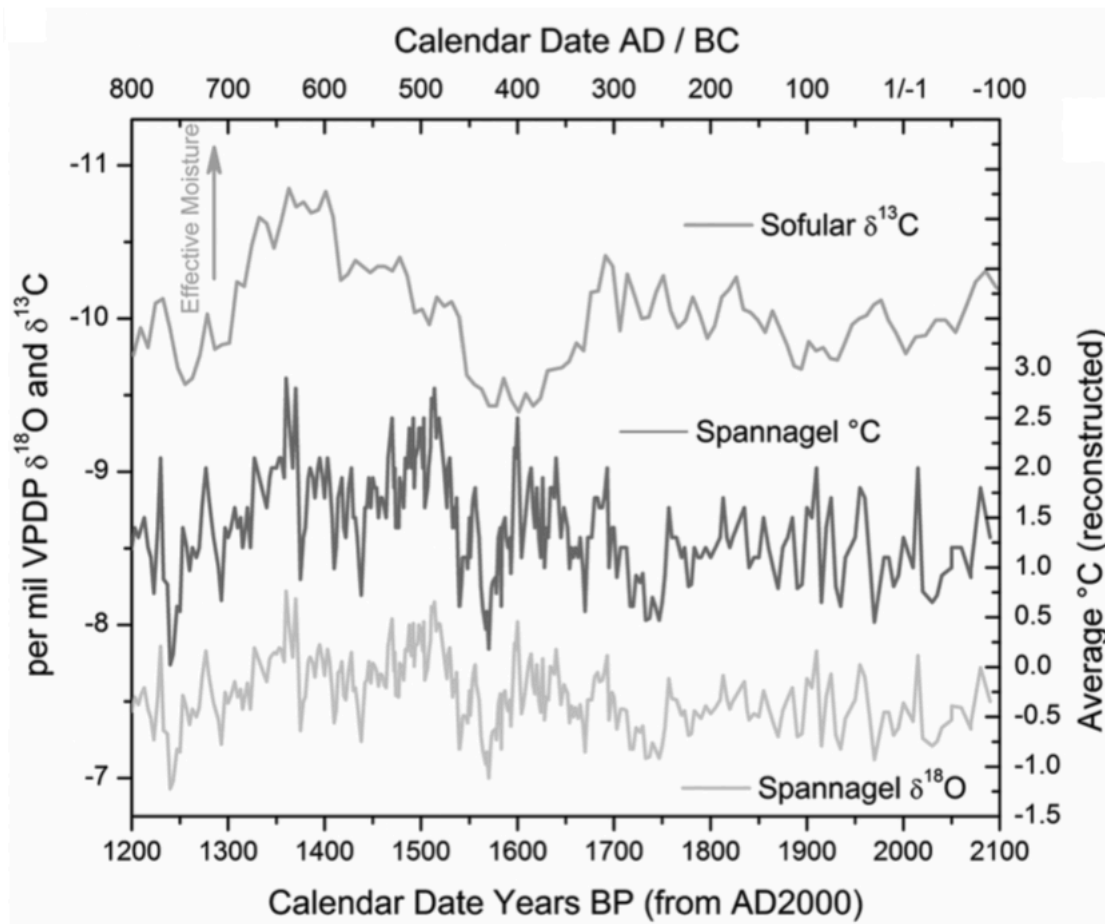


Figure 17. Stalagmite isotope data showing a reconstruction for effective moisture (i.e. precipitation) from the Sofular Cave (Turkey), and annual temperatures from the Spannagel Cave (Austria). (McCormick et al. 2012, Fig. 1b)

Moving east to the southeastern Balkan region, $\delta^{18}\text{O}$ and $\delta^{13}\text{C}$ stalagmite data from the Uzuntarla Cave in northwestern Turkey (Fig. 18) exhibit rising wetness and temperatures around the mid-to-late-second century BC (Fig. 19).⁵³ The signal for these isotopic variables is not as straightforward, as certain environmental factors blur the signals and cause complexities that are not yet well understood at Uzuntarla.⁵⁴ However, $\delta^{18}\text{O}$ and $\delta^{13}\text{C}$ correspond well with temperature/seasonal precipitation and excess water (abundant rainfall)/summer temperature respectively. The $\delta^{13}\text{C}$ stalagmite data show a rise in excess water at the start of the



Figure 18. The locations of the Uzuntarla, Kocain, and Sofular caves, as well as the Yenesu and Ovacik caves; data from the latter two caves were not used as the samples did not have the most recent portion of the Holocene. (Gokturk 2011, Fig. 1.2)

⁵³ Gökürk 2011, 75-78.

⁵⁴ Gökürk 2011, 72-75.

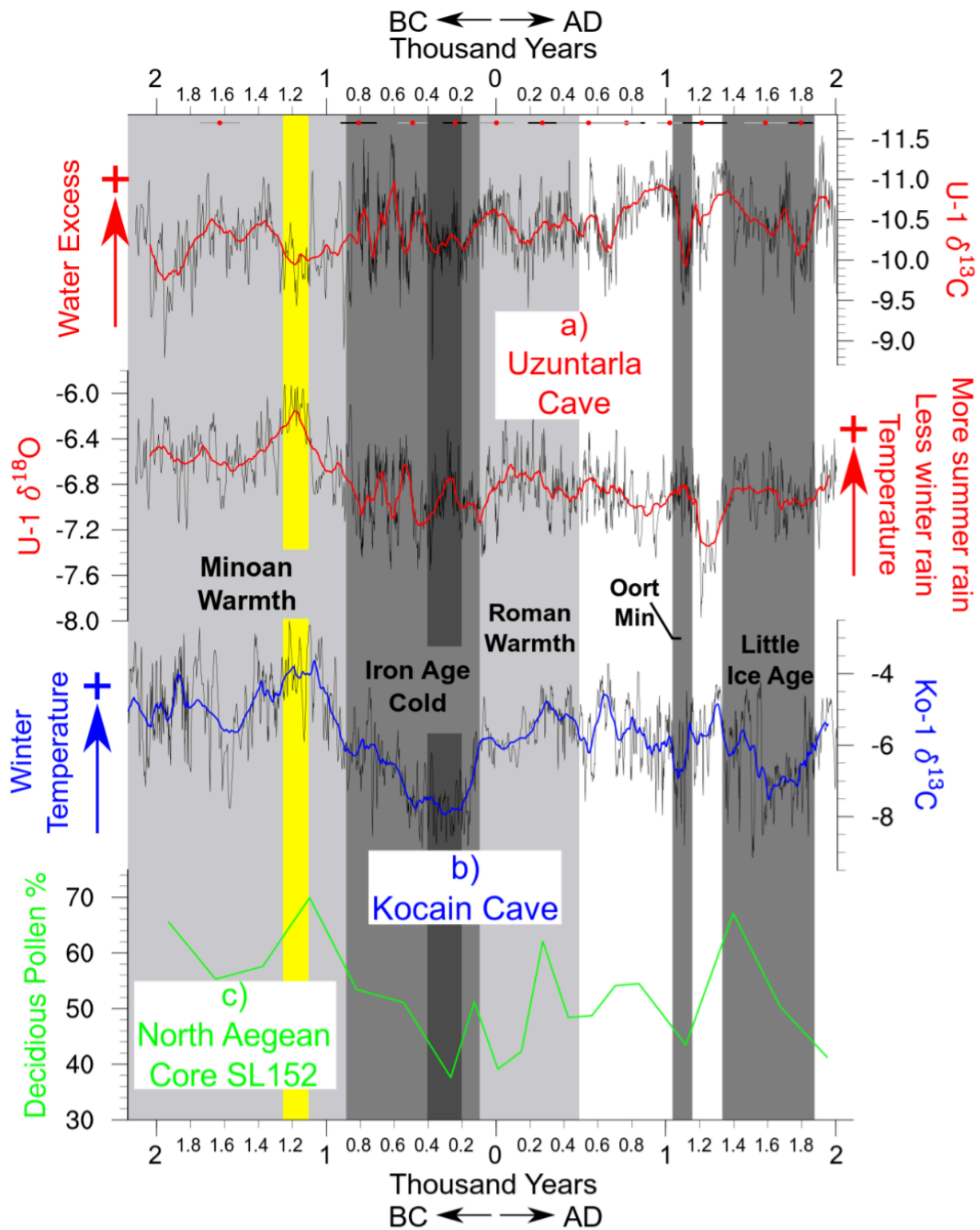


Figure 19. Stalagmite stable isotope data from Uzuntarla Cave (a) (northwestern Turkey), Kocain Cave (b), and a core from the North Aegean Sea. (Göktürk 2011, Fig. 4.5)

RCO, but reductions at the mid-first century AD that continue into the second century. Similarly, the $\delta^{18}\text{O}$ stalagmite record demonstrates a rise in temperatures

just after the RCO began, around the mid-second century BC. But unlike the $\delta^{13}\text{C}$ record, the $\delta^{18}\text{O}$ data show climatic stability until the late third century AD. Besides a decline in water excess from AD 50 – 190, the warmth and stability exhibited by the isotopic data at Uzuntarla in northwestern Turkey supports the concept of a Roman Climate Optimum.

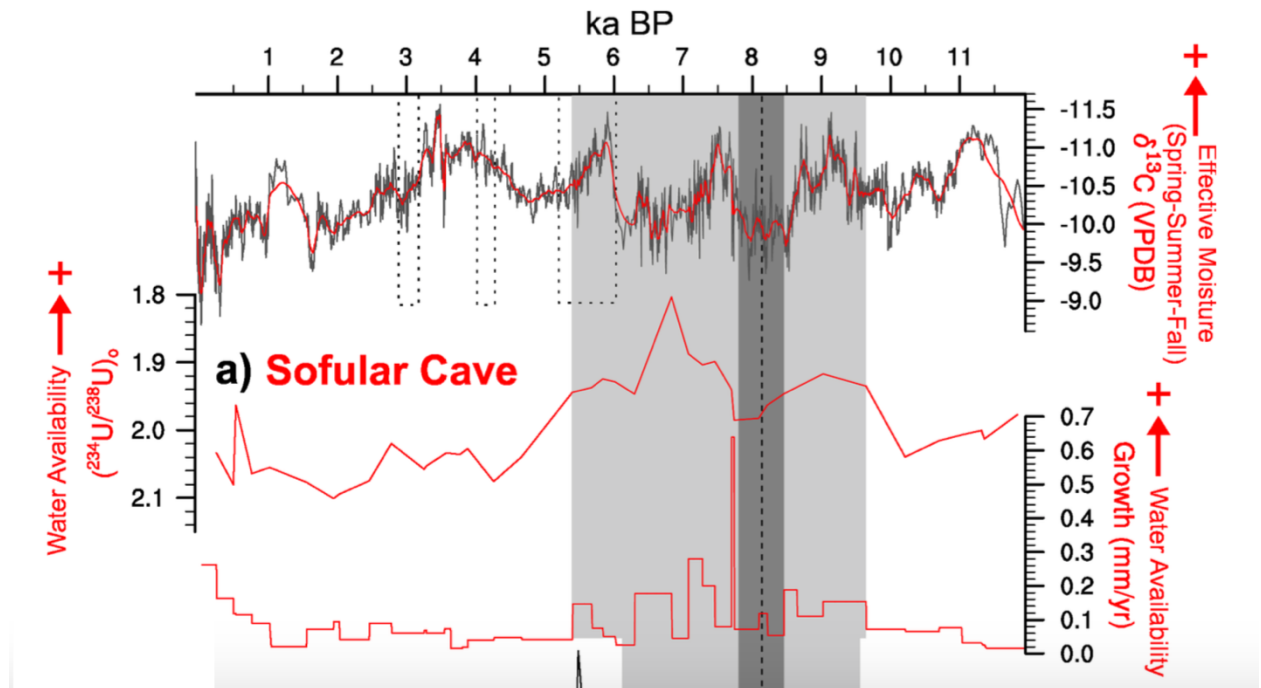


Figure 20. $\delta^{13}\text{C}$ stalagmite record for the Sofular Cave. The stable isotope values here are represent effective moisture, a proxy for both temperature and humidity. (Göktürk 2012, Fig. 2.9)

Slightly further east, the Sofular Cave on the southern Black Sea coast in Turkey also experienced a phase of relative stability during the RCO (figures 17, 18, and 20). Producing a $\delta^{13}\text{C}$ stalagmite record that measures effective moisture (a gauge for annual temperature and/or humidity), the isotopic data show a stable RCO phase similar to the Uzuntarla data (Fig. 19) and slightly more balanced than the Spannagel data (Fig. 17).⁵⁵ However, Göktürk reminds us that moisture trends, and thus the effects of climate change, at the Sofular Cave might be masked by

⁵⁵ Göktürk 2011, 28-29.

surface water excesses of the nearby Black Sea.⁵⁶ Nevertheless, the Sofular data demonstrate a few ups and downs, but, overall, a steady RCO phase around the southern Black Sea.

In southern Turkey, a $\delta^{13}\text{C}$ stalagmite record from the Kocain Cave reveals an unceasing cold and snowy phase from the eighth century BC until around the mid-second century BC, followed by a phase of warmer temperatures until around AD 500 (Fig. 21).⁵⁷ Temperatures rise at Kocain from the mid-second century BC and into the first century, but that warming is interrupted by a less-intense cold period extending from the end of first century BC until the early second century AD, possibly initiated by the 43 BC eruption. Thus, while the RCO appears to have had a later start in southern Turkey, and seems not to have been continuous, overall, the data demonstrate a phase that was substantially more stable than the 700-year period preceding it, with warming starting around 140 BC.

Just southeast of the Kocain Cave, $\delta^{18}\text{O}$ isotope data from the Soreq Cave in the Near East (Israel) demonstrate a wetter phase for most of the RCO, from 200 BC – AD 100.⁵⁸ This wet phase was followed by an arid phase that lasted for the remainder of the Roman period. At around AD 100 rainfall dropped an estimated 400mm annually, showing no recovery in the data set.⁵⁹ The stable isotope data from the Soreq Cave mostly supports the concept of an RCO for the Near East.

⁵⁶ Göktürk 2011, 34-35.

⁵⁷ Göktürk 2011, 41-58.

⁵⁸ Orland *et al.* 2009.

⁵⁹ Orland *et al.* 2009, 32.

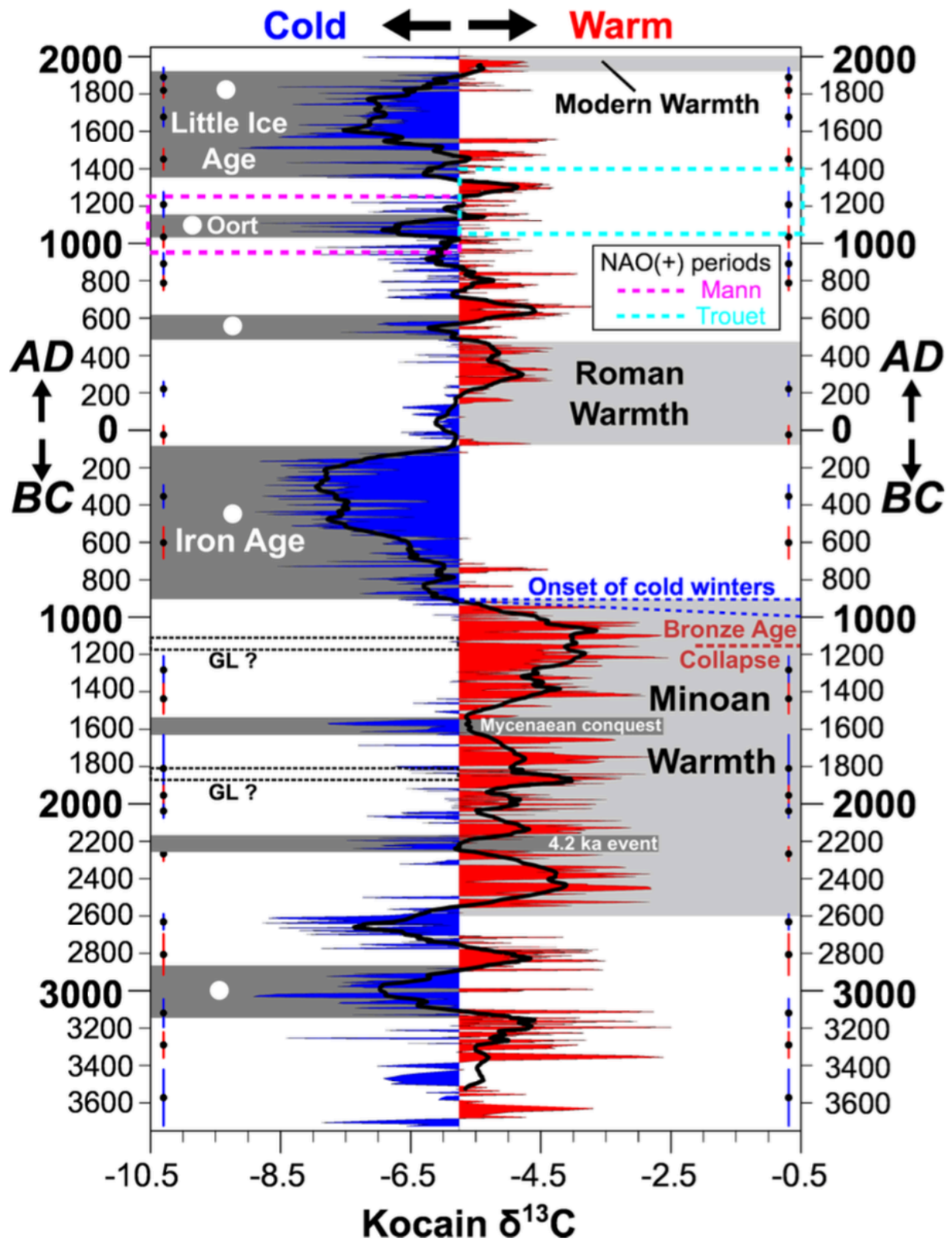


Figure 21. A $\delta^{13}\text{C}$ stalagmite record from Kocain Cave in southern Turkey.
(Göktürk 2012, Fig. 3.7)

Lake-sediment records

Lake records can give a history of regional climatic change and water balance, such as runoff and evaporation, by examining the annual layers of sedimentation for a given lake.⁶⁰ The rates of sedimentation best indicate how wet or humid regions were each year, through the preservation of stable isotopes and pollen. Lake sediments can provide high-precision paleoclimatic records, but this is not always the case. The complexities of a lake's surrounding hydrological environment, coupled with anthropogenic impact, complicate deciphering the paleoclimatic data.⁶¹ With that said, we do have lake sediment records that depict past climatic conditions fairly well for the Roman period, including for both the RCO and post-RCO phases.

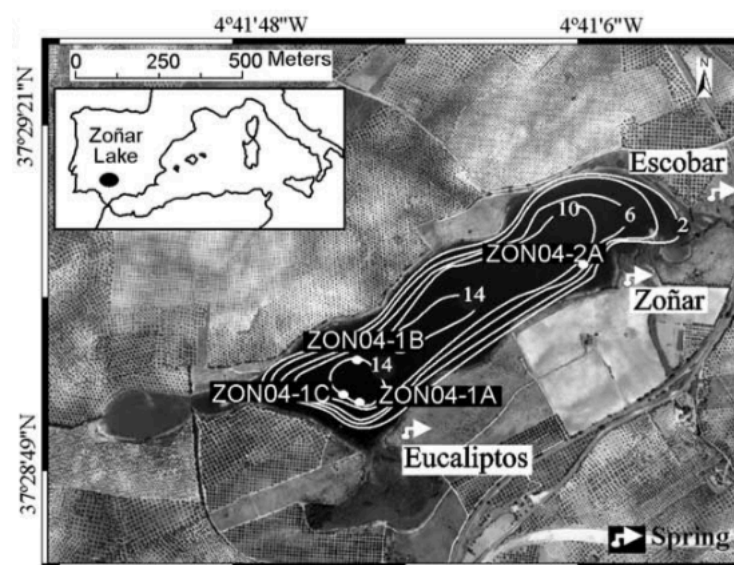


Figure 22. The location of Zonar Lake in southern Spain, and the lake catchment and bathymetric mapped on to an aerial photograph of the lake. (Martín-Puertas *et al.* 2009)

⁶⁰ Luterbacher *et al.* 2012, 112-113.

⁶¹ Luterbacher *et al.* 2012, 112-114; Harper and McCormick 2018, 30.

Analysis of sediment cores taken from Zonar Lake in southern Spain (Fig. 22) reveals an arid phase that works against the idea of an RCO for the Spanish region.⁶² Isotopic samples were taken from four sediment cores at Zonar Lake. All cores exhibited, what the study led by Martín-Puertas refer to as, “The Iberian-Roman Humid Period”, stretching from 2600 – 1600 cal yr BP (650 BC – AD 350). However, this humid phase is compared to the rest of the 4000-year core-sediment series. An evident interval of aridity occurs from 190 BC – AD 150, for nearly the entirety of the Roman Climate Optimum.⁶³ The authors of the study propose that the arid interval was largely due to natural climate forcing, but they suggest the data could be skewed by anthropogenic impact from water use at the lake.⁶⁴ Nevertheless, the improvement of conditions at AD 150 still runs counter to the theorised end of the RCO.

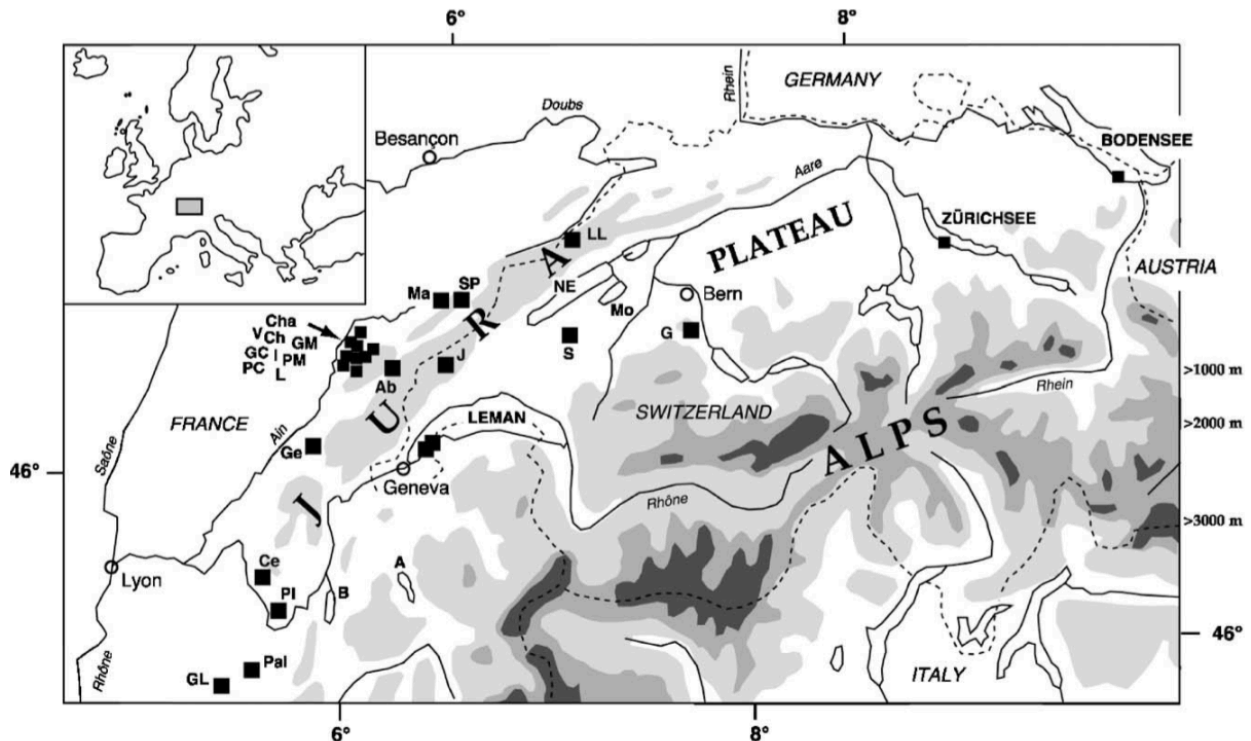


Figure 23. Geographical location of study sites in the Jura Mountains. (Magny 2004, Fig. 1)

⁶² Martín-Puertas *et al.* 2009.

⁶³ Martín-Puertas *et al.* 2009, 109-110.

⁶⁴ Martín-Puertas *et al.* 2009, 119.

Just north of the Western Alps, along the border of France and Switzerland, lake-level statuses were analysed for 26 lakes in the Jura Mountains (Fig. 23), revealing lower lake levels for all of the Roman Climate Optimum.⁶⁵ From 400 BC – AD 150, lake levels were moderate-to-low, with higher levels reported from AD 150 – 250, but a return to lower levels thereafter. These data disagree with the dendrochronological data from the nearby Alps, which show high-to-stable precipitation trends from 200 BC – AD 150 (Fig. 7). However, anthropogenic forces, such as deforestation, could have changed the hydrological landscape and thus might be blurring the paleoclimatic signals at these lakes.

Moving east, a multiproxy record from Lake Shkroda (Albania) preserves an archive of wetness for southeast Europe for the past 4,500 years.⁶⁶ The Lake Shkroda record is a prime example of a complex paleoclimatic lake-sediment signal. The study led by Zanchetta conducted stable isotope and pollen analysis to determine wetness in the Lake Shkroda region. The pollen record was not particularly useful, as the region's vegetation is not sensitive enough to changes in rainfall or hydrology to produce a reliable chronology of wetness. They found more precision with the stable isotope data ($\delta^{18}\text{O}_c$), however; in fact, much higher precision than other sediment records in neighbouring regions.⁶⁷ The data show a wet phase from 550 BC – 50 BC, followed by a dry phase 50 BC – AD 150, and a three-century period of wetness from AD 150 – 450 (Fig. 24). While the wetness observed from 200 BC – 50 BC does support a Roman Climate Optimum, the dry phase from 50 BC – AD 150 and the wet phase beginning at the supposed end of the RCO does not.

⁶⁵ Magny 1993, 77.

⁶⁶ Zanchetta *et al.* 2012.

⁶⁷ Zanchetta *et al.* 2012, 787.

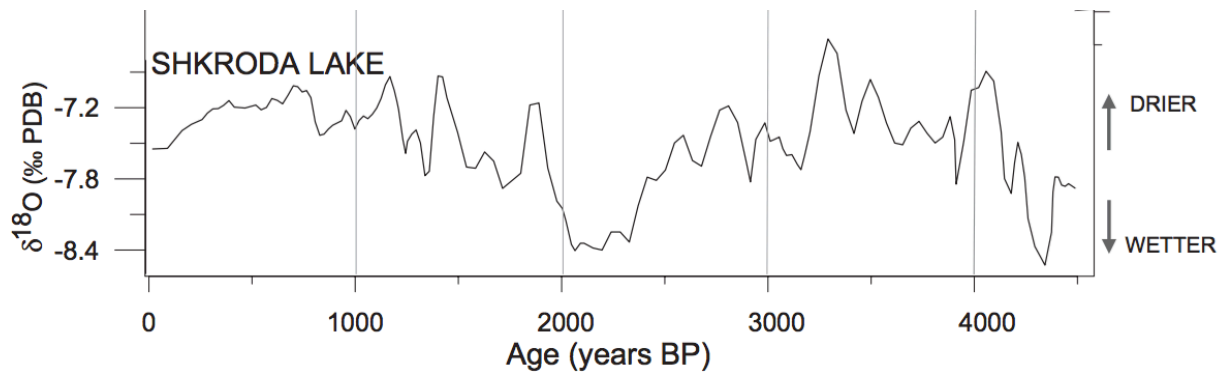


Figure 24. $\delta^{18}\text{O}$ stable isotope data from Shkodra Lake exhibiting wetness trends for the past 4500 years. (Zanchetta et al. 2012, Fig. 6)

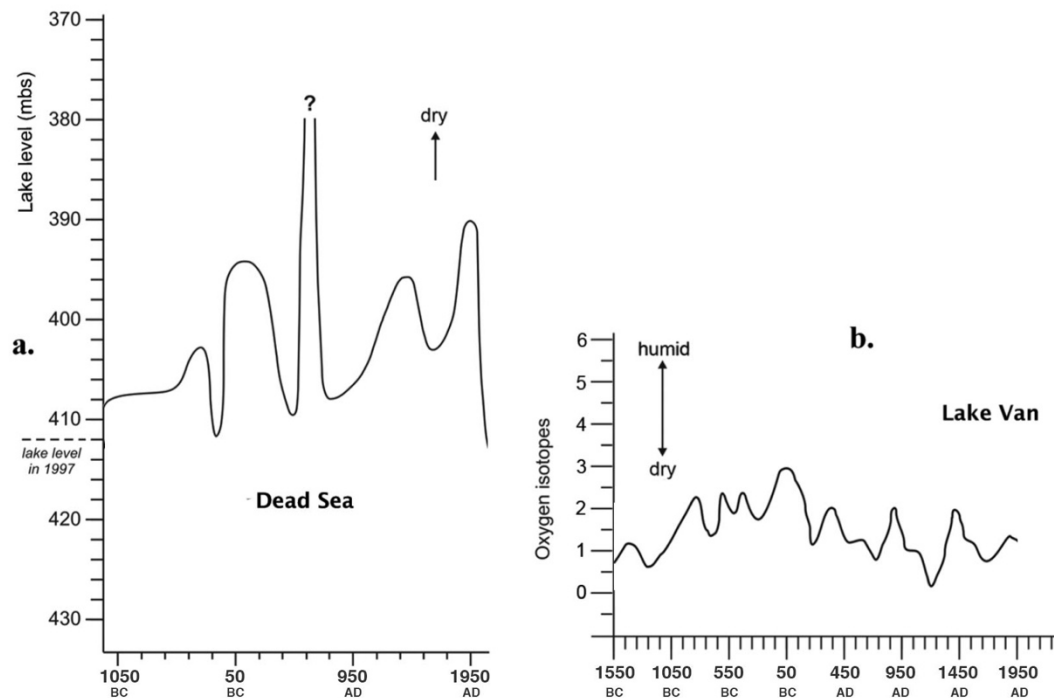


Figure 25. Lake level data for the Dead Sea (Israel/Jordan) and oxygen isotope data for Lake Van (Turkey), both representing wetness trends. (McCormick et al. 2012, Fig. 7 a and b)

Data from Lake Van in eastern Turkey reveal aridity to have already been high at the start of the RCO (the highest since 850 BC), having been on the rise since 250 BC, and peaking around 50 BC (Fig. 25).⁶⁸ Conditions improved from then, as humidity rose in line with the RCO, but peaked around AD 150, seeing a moderate

⁶⁸ Wick, Lemcke and Sturm 2003, 670.

decline from there onward until late antiquity. Thus, as a representative for eastern Turkey, the Lake Van data correspond with the second half of the Roman Climate Optimum, but not the first half.

In southwest Turkey, palynological and sedimentological data from the Bereket Basin provide a paleoclimate record for the past three millennia.⁶⁹ There is, however, a caveat. The Bereket Basin record is heavily influenced by anthropogenic forces – landscape clearance, intensive agricultural practices, and grazing pressure – that began with the Beyşehir Occupation Phase (1200 BC – AD 400 for the Bereket region), which overlaps with most of the Roman period.⁷⁰ The Bereket Basin record cannot stand on its own as a proxy for climate in southwestern Turkey. But Kaniewski and his team believe climatic variation plays a role in the paleoclimatic signals, and that the data can be juxtaposed with other proxies from the same region (e.g. Kocain Cave, Lake Van, Soreq Cave, the Dead Sea).⁷¹

Increased pollen types and $\delta^{18}\text{O}$ values point to a wet phase at Bereket from 250 BC – 40 BC, coinciding with the start of the RCO. Wetness decreased in the later first century BC, but there is a sharper increase in aridity in the second century AD, culminating in the driest episode of the Roman period at Bereket from AD 130 – 350.⁷² The Soreq Cave data and the Lake Van data correlate well with the Bereket Basin record for the RCO. Like Bereket, the Soreq Cave and Lake Van regions show wetness early on in the RCO from 200 BC – AD 100 and 200 BC – AD 50 respectively, and major aridification in the early second century AD.⁷³ Similarly, the Kocain-Cave

⁶⁹ Kaniewski *et al.* 2007.

⁷⁰ Kaniewski *et al.* 2007, 2201-02. The Beyşehir Occupation Phase is an agricultural phase in Anatolia that broadly corresponds to the period from the late Bronze Age (1200 BC) to the late Roman Empire (AD 400).

⁷¹ Kaniewski *et al.* 2007, 2212-13.

⁷² Kaniewski *et al.* 2007, 2212.

⁷³ Orland *et al.* 2009; Wick, Lemcke, Sturm 2003.

data show a phase of relative warmth and stability commencing in the mid-second century BC, but, in the case of the Kocain Cave, it lasts for 700 years.

Not far from the Bereket Basin, and also along the eastern Mediterranean, data from the Dead Sea does not agree with the consensus of data for the period defined as the Roman Climate Optimum. At the start of the RCO, around 200 BC, an arid phase begins at the Dead Sea. Lake-level data extracted from sediment cores indicate a dry period lasting for the whole of the RCO, from 200 BC until the third century AD (Fig. 25).⁷⁴ The Dead Sea data contrast with the data from Soreq Cave (just 48 kilometres west), which exhibit a wet phase from 200 BC – AD 100, demonstrating that areas not 50 kilometres can experience different climatic phases.

In Africa, lake-sediment records are the principal source of historic climatic variability (although most are low-resolution due to a largely arid climate), and there are a few important ones (to the scope of this paper) around the Ethiopian Highlands – the region from which precipitation is responsible for the flooding of the Nile – that extend back to the Roman period.⁷⁵ The majority of these records only preserve past precipitation, which explains why we have so little data on past temperature for the continent. The eastern coast of equatorial Africa receives almost all of its rain from the Indian Ocean via the East African monsoon; thus, if the East African Monsoon fluctuates, so do lake levels from Ethiopia to Malawi, as does the Nile River (below).⁷⁶ Unlike the many other regions throughout the Empire that exhibit favourable conditions during the RCO, the lakes of East Africa (Lake Turkana, Lake Tanganyika, Lake Edward, and Lake Challa) show aridity for most of

⁷⁴ Migowski 2006, 425.

⁷⁵ Nash *et al.* 2016, 2.

⁷⁶ Nash *et al.* 2016, 5.

the RCO and in some cases beyond; however, we must keep in mind that these records are low-resolution and do not exhibit even decadal chronologies.⁷⁷ The sediment record at Lake Chew Bahir (Fig. 26), however, reveals a rise to moderate levels of wetness around the start of the RCO that plateaus and lasts the entirety of

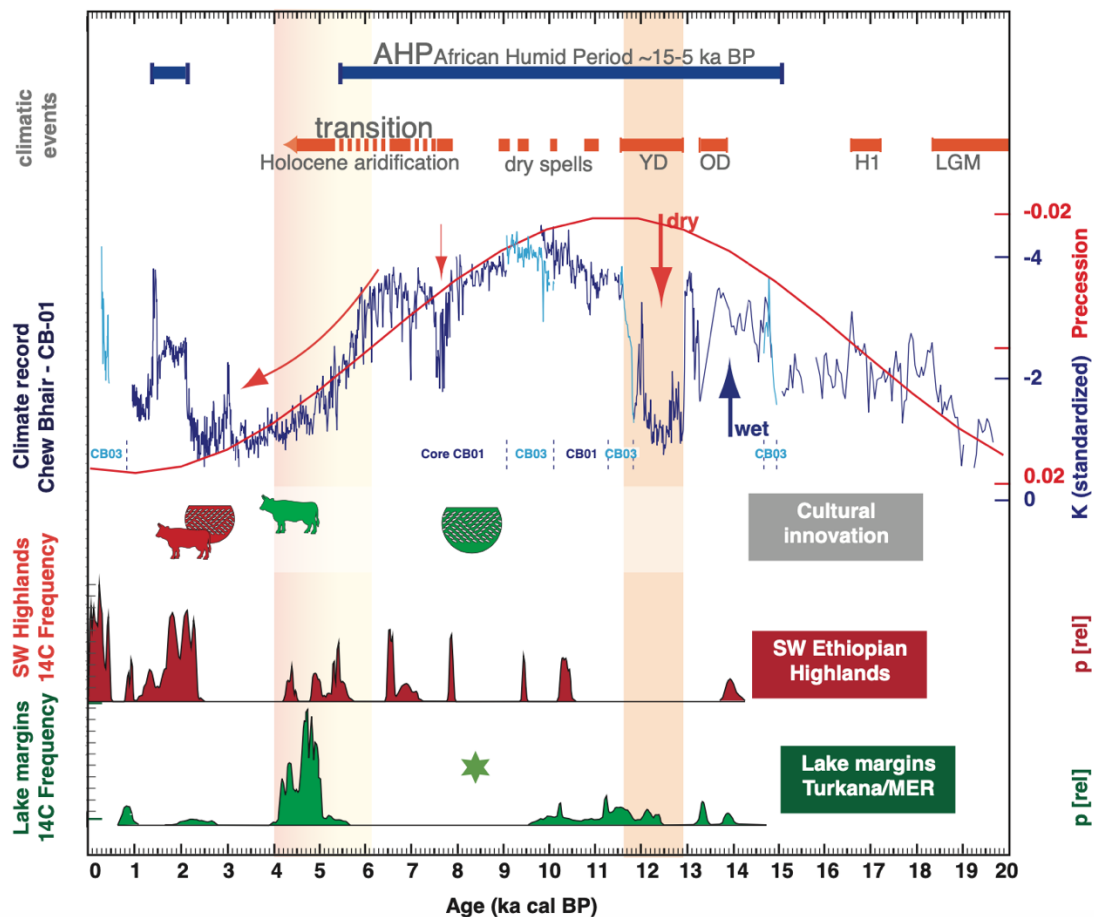


Figure 26. This figure displays a climate record for the region surrounding Lake Chew Bahir (a proxy for the Ethiopian Highlands) and settlement patterns in the Ethiopian Highlands and around lake margins of Lake Turkana and Lake Ziway-Shalla. For the purposes of this thesis, we will only focus on the climate record (blue line). (Foerster *et al.* 2015, Fig. 2)

the period. Nonetheless, four lakes, in a region where wetness is sometimes representative of the vital Nile flood, show a dry phase for the RCO, and conditions appear to worsen in some areas in the second century.

⁷⁷ Verschuren and Charman 2008, 189-231; Nash *et al.* 2016, 7; Halfman, Johnson and Finney 1994; Russell and Johnson 2005; Buckles *et al.* 2016.

Nile flooding

Documentation of Nile flooding gives us some insight into climate and environment for the Empire's most important province for agricultural production. As mentioned, lake-sediment records in East Africa, particularly around the Ethiopian Highlands, are used as a proxy for annual precipitation in the region and can sometimes be indicative of Nile-river flooding. Conversely, Nile-flood records are used as a proxy for precipitation in the Ethiopian Highlands, which assist the low-resolution lake-sediment records of East Africa.⁷⁸ Good documentation for the Nile during the Roman period starts in the first century BC, at year 88. A study in 2012 led by McCormick summarizes Danielle Bonneau's reconstruction of Nile-flood data gleaned from papyri, coin issues, and other records that were mostly based on Nilometer readings.⁷⁹ The data show that reports for good floods happened far more frequently between 88 BC and AD 156 (Fig. 27). After AD 156, in the post-RCO phase, the proportion of the two most favourable Nile flood categories dropped from 20% before AD 156 to 7%. McCormick and others also note that the most deficient flooding occurs post-156, with 31% of reports being of the three worst flood rankings, up from 21% pre-156 (more on this below).⁸⁰ We should be mindful of societal forces that might have prevented flood reporting – such as political instability, economic hardship, or disease – but the evidence we have for Nile flooding does indicate climatic eras in Egypt that support the supposed chronological boundaries of the RCO.

⁷⁸ McCormick *et al.* 2012, 188-189; Harper and McCormick 2018, 36.

⁷⁹ Nilometers are measurement pillars, wells or shafts located at various locations along the Nile that serves to indicate the height of annual Nile floods.

⁸⁰ McCormick *et al.* 2012, 189.8

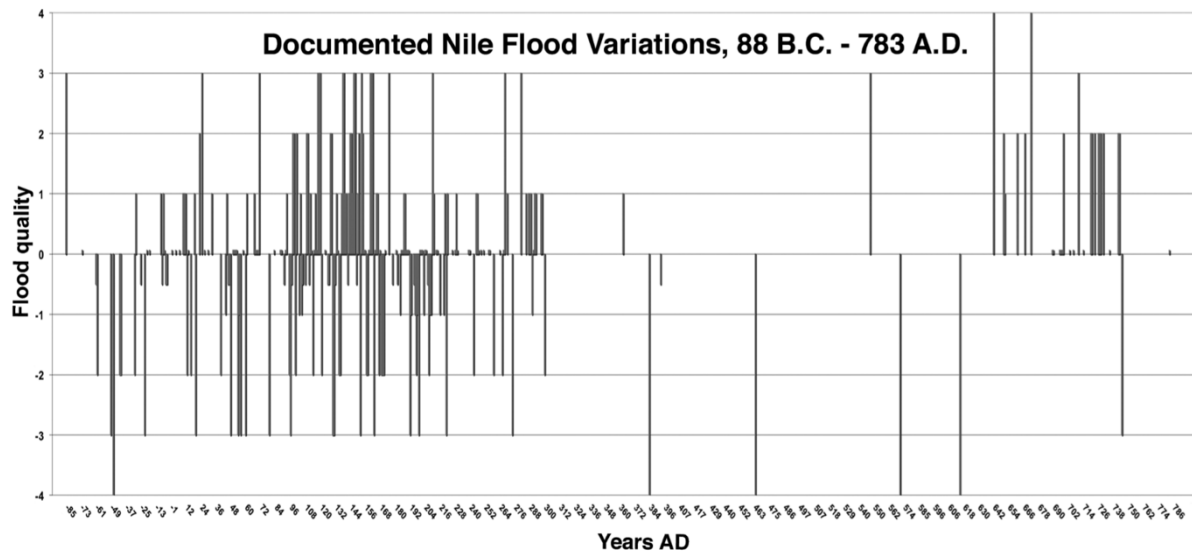


Figure 27. Documentation of Nile flooding from the first century BC until the 8th century AD. Note the apparent reduction in good Nile floods post-AD 156. (McCormick et al. 2012, Fig. 10)

Glacial evidence

Best put by Holzhauser, Magny, and Zumbühl in 2005, “The behaviour of glaciers is highly dependent upon climatic conditions and they react according to the sum of complex interactions of climatic parameters (e.g., temperature, precipitation, solar radiation).”⁸¹ Glacial advances and retreats are dated by examining treeline variations, glacial drift via glacial sediment, or through radiocarbon dating of organic matter buried by a glacier.⁸² Unfortunately, as a paleoclimatic signal, glacial data provide the lowest resolution results out of all climate proxies; glacier retreats and advances best depict climate on a centennial scale. With that said, we can still infer broad climatic reconstructions for the Roman period based on glacier fluctuations.

⁸¹ Holzhauser, Magny and Zumbühl 2005, 792.

⁸² Holzhauser, Magny and Zumbühl 2005, 790; Le Roy *et al.* 2015, 2.

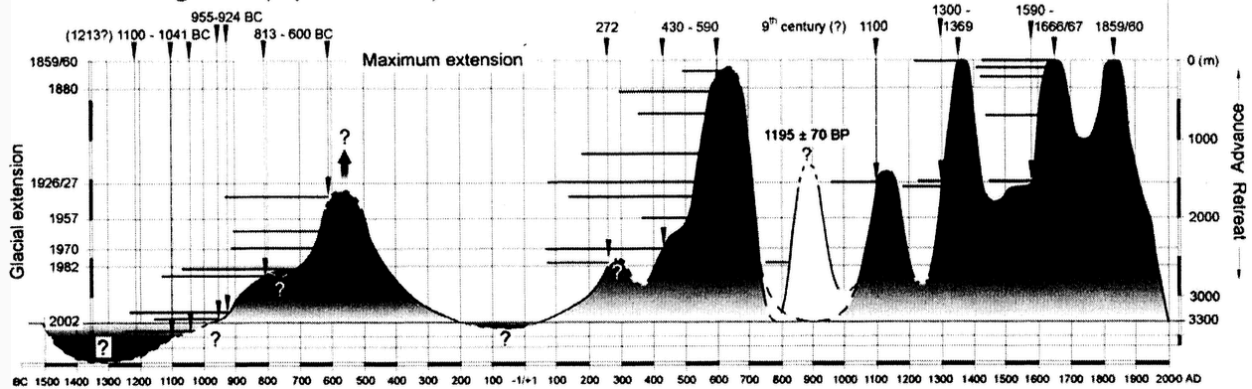
Data from the Great Aletsch, Gorner, and Lower Grindelwald glaciers, all from the Bernese Alps in Switzerland, exhibit great similarity across their 3500-year time series, and correspond with the Roman Climate Optimum (Fig. 28).⁸³ The Great Aletsch data show an advance (climate cooling) beginning in the 9th century BC, with a peak in the mid-sixth century BC (Fig. 27). From there a gradual retreat began (warming). By the early second century BC, the Great Aletsch reached 2002 levels, probably reaching its minimum around the middle of the first century BC.⁸⁴ It gradually advanced again towards the end of the first century AD and continued to grow throughout the second and third centuries. We see mostly the same fluctuations for the Gorner glacier, with lower resolution. The Gorner shows a peak advance in the mid-sixth century BC, followed by a retreat towards the RCO phase.⁸⁵ How much the Gorner glacier retreated from the fourth century BC until the fifth century AD is uncertain; the topographical dating is blurry, as is the corresponding dendrochronological dating. However, we do know that the Gorner did not grow beyond its mid-fourth century BC levels, at the very least, implying the area was warmer and wetter during the RCO, and probably until AD 400 when evidence of an advance finally appears. Data from the Lower Grindelwald are even less clear than the Gorner data (Fig. 28), showing only major advances without the contours. The data show a large advance in the sixth century BC, similar to the Great Aletsch and Gorner, but no further major advances until the sixth century AD, supporting, but not strongly implying, the idea of an RCO.

⁸³ Great Aletsch glacier: Holzhauser 1984; Gorner: Holzhauser 2001; Lower Grindelwald: Zumbühl 1976; Holzhauser, Mangy, and Zumbühl 2005, 790-791.

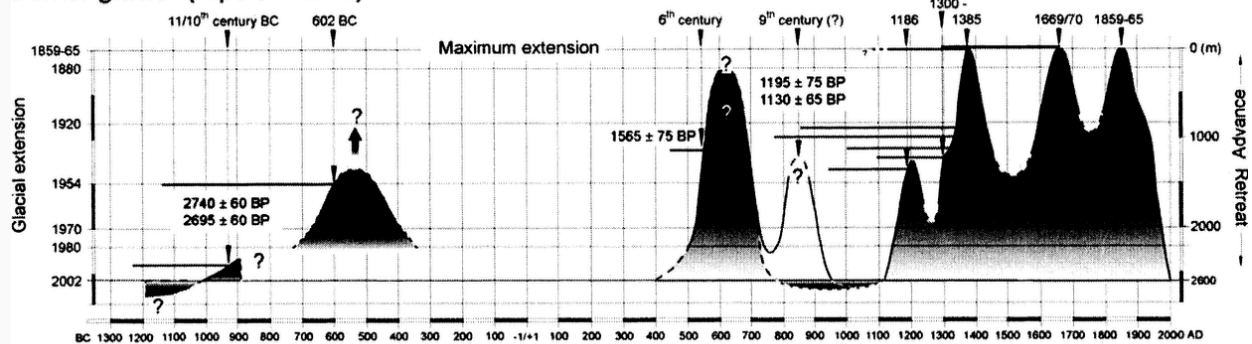
⁸⁴ Holzhauser, Mangy, and Zumbühl 2005, 791.

⁸⁵ Holzhauser, Mangy, and Zumbühl 2005, 794.

Great Aletsch glacier (Alps of Valais)



Gorner glacier (Alps of Valais)



Lower Grindelwald glacier (Bernese Oberland)

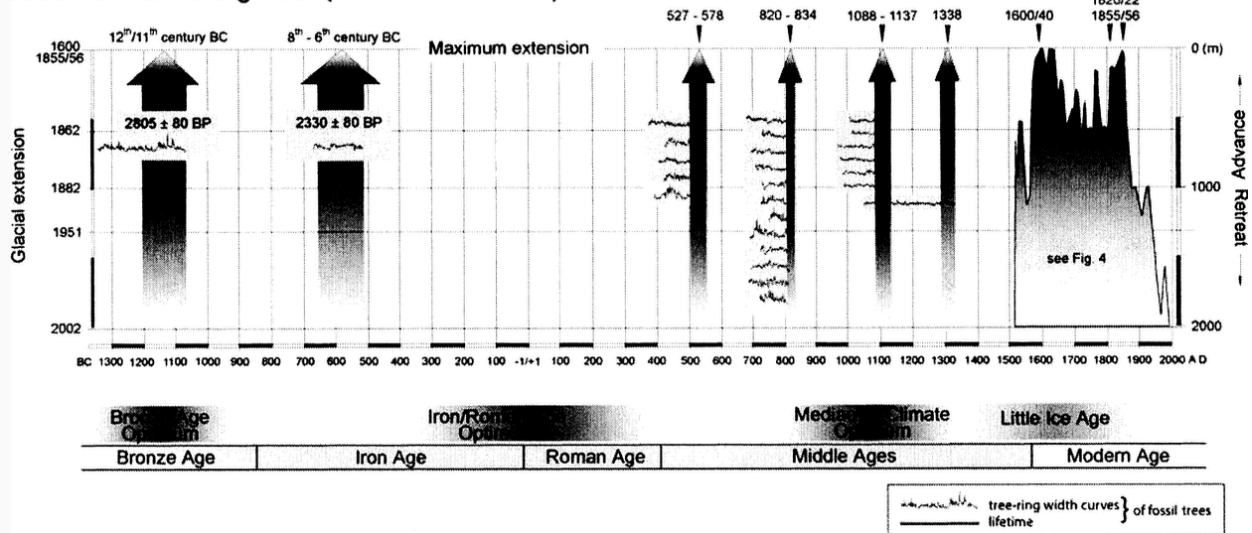


Figure 28. Glacier fluctuations for the past 3500 years, showing the Great Aletsch, Gorner, and Lower Grindelwald glaciers. (Holzhauser, Magny, and Zumbühl 2005, Fig. 2)

Data of altitudinal variations of the Mer de Glace glacier in the French Alps show growth up to around 600 BC, and no evidence of growth thereafter until the early fourth century AD. It is uncertain how much the Mer de Glace retreated

between 600 BC and AD 312, but based on the length of the retreat, Le Roy *et al.* 2015 suggest the period between the third century BC and the early-second century AD was very warm (Fig. 29).⁸⁶ For the Gepatschferner glacier in Austria, we lack data

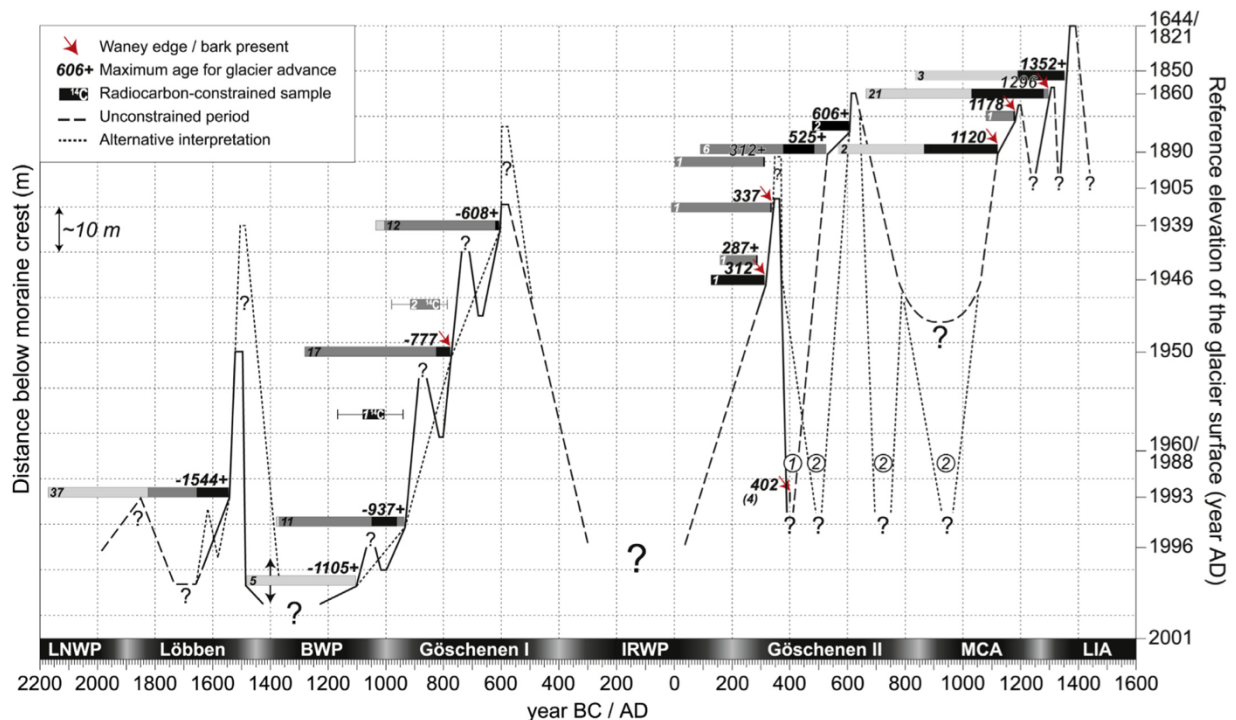


Figure 29. Altitudinal variations of the Mer de Glace glacier surface for the past 4000 years determined from subfossil wood layers. (Le Roy *et al.* 2015, Fig. 6)

before the first century BC. However, the data show an advance in the early-fourth century, and no advance before it, suggesting that, at least, it was warmer in the second half of the RCO (50 BC – AD 150) than it was in the third and fourth centuries (and probably the second century) for the Gepatschferner region of Austria.⁸⁷

The data from all five glaciers discussed suggest that these regions of central Europe were warmer during the Roman Climate Optimum. All show a cold sixth century BC, followed by a warmer period that lasted until at least the late-second century AD, and in some cases later.

⁸⁶ Le Roy *et al.* 2015, 8-10.

⁸⁷ Nicolussi and Patzelt 2000; McCormick *et al.* 2012, 186.

Discussion

Based on the paleoclimatic evidence, it does appear that there was a climate optimum for many parts of the Roman world from the second century BC until around the mid-second century AD; however, an optimum was not ubiquitous, as Spain, Albania, East Africa and Israel all experienced notable aridity during the RCO. Additionally, sharp rises in cooling and aridity occur following the eruption of the Alaskan volcano Okmok in 43 BC. Climatic deterioration registers in nearly every climatic proxy after 44 BC, but in most cases climatic balance seems to recover quickly, and the RCO continues. Overall, most of the Roman world experienced stable conditions for much of the period between 200 BC and AD 150.

The tree-ring data from central Europe show warmth and wetness for most of the RCO until the mid-second century AD, save for the short shock following 43 BC. The central Asian data (only beginning at AD 100) starts with high temperatures, but exhibit a definite end to warmth post-AD 150. Tree-ring data from the Libyan Sahara show stability throughout the RCO; however, the 50-year period before the start of the common era (that includes the massive 43-BC eruption) is missing from the record. The ENSO-sensitive data from New Zealand and New Mexico display sharp reductions in temperatures in the late-first/early-second century AD that might be linked with the significant reduction in solar activity happening around the same time. The stalagmite data from Spain display overall warmth and moisture for the RCO, and data from the Coral Cave in Sweden show relative stability for both temperature and precipitation. Similar stability is apparent from the Spannagel stalagmite data from Austria, and all of the caves in Turkey (Solufar, Uzuntarla, and Kocain) show no notable reduction in temperatures or wetness during the focus

period, besides a very cold start to the RCO at the Kocain Cave. And just south in Israel, data from Soreq Cave stalagmites indicate a wet phase from 200 BC – AD 100.

Data from the Zonar Lake in Spain show an arid phase from 190 BC – AD 150, nearly the entirety of the so-called RCO. The Shkodra Lake in Albania shows a very wet beginning to the RCO, with a notable spike in aridity at AD 50. Data from Lake Van in eastern Turkey demonstrate a late start to the climate optimum, as a rise in wetness from extreme aridity only begins in the mid-to-late-second century BC. The Dead Sea experienced a steep rise in aridity in the second century BC, which lasted until the third century AD, and while we cannot use the Bereket Basin data on their own as a reliable paleoclimatic proxy, due to anthropogenic forcing, the region does exhibit a climatically favourable span during the RCO that broadly corresponds with other nearby proxies, such as Soreq Cave.

Just outside of the Roman world, the lake-sediment data from East Africa around the Ethiopian Highlands (except for Lake Chew Bahir) show aridity for the majority of the RCO, unlike much of the Mediterranean region; but low-resolution records make it difficult to tell on a decadal scale. However, Nile-flood reports help sharpen the climatic picture in East Africa. Reporting shows much better flooding from the first century BC until AD 156 than after 156. And data on fluctuations from five glaciers in central Europe display no glacial advances during the Roman Climate Optimum, indicating that central Europe was, at least, not experiencing a cold phase. While not ubiquitous, the data above reveal that the period between 200 BC and AD 150 was warmer, wetter, and stable for most of the Roman world. But that stability was soon to end soon after AD 150.

AD 150 – 200: a dramatic start to the post-RCO phase

The majority of the climatic proxies show more turbulent activity in the second century AD, with most exhibiting a notable shift to worsening conditions (cooling and aridity) around the middle of the century. In some instances, climatic variability begins in the first half of the century, but most data show notable change after the midway point of the century, including our most chronologically precise and reliable data: tree rings. Therefore, it becomes harder to propose that the Roman Climate Optimum extended into the latter half of the century.

Tree-ring data

The tree ring data from both central Europe and central Asia show sharp drops in temperatures around the AD 150s, presenting an end to the trend of warmth experienced for most of the so-called RCO.⁸⁸ The data from the Alps show that temperatures began to decrease in central Europe around AD 150, reaching their lowest point of the century in the AD 180s, before rising again into the third century (Fig. 7).⁸⁹ At the same time, precipitation levels decreased in central Europe, but not substantially. The central Asian data show massive reductions in temperatures starting around the AD 160s (Fig. 8).⁹⁰ Like central Europe, temperatures do not recover until the late-second century. The tree-ring data from the Libyan Sahara in the Wadi Tannezuft exhibit the driest period in three centuries in the latter half of the second century (Fig. 9). And the ENSO-sensitive data from New Zealand and New Mexico, sometimes used to infer climate for Eurasia and central Asia,

⁸⁸ Büntgen *et al.* 2011, 581; Büntgen *et al.* 2016, 233.

⁸⁹ Büntgen *et al.* 2011, 581.

⁹⁰ Büntgen *et al.* 2016, 233.

demonstrate variability (cooling and aridity) in the first half of the second century, with continual fluctuations until conditions improve late in the century, similar to the central Asian and central European data.⁹¹

It is plausible that the eruption happening at Ksudach in the AD 160s (figures 5 and 6) caused the extreme drop in temperatures in central Asia and contributed to the colder, slightly more arid phase in central Europe. However, the rise in aridity in the Wadi Tanezzuft begins before the eruption at 163, so it may not have been the cause of drying there. The larger eruption happening at AD 170, which was felt globally, probably further worsened an already deteriorating climate across Eurasia.

As a whole, the tree-ring chronologies hint at a cold and dry phase stretching from central Asia to central Europe, and drying in the Libyan Sahara may indicate that most of Mediterranean region experienced the phase; but, of course, that may not necessarily be the case. We must look at the other climatic proxies to assess what may have been happening in the regions in between.

Stalagmites

The stalagmite data vary by region for AD 150-200, but, generally, the cave-data show that the Roman world was getting cooler and drier. Stalagmites from Spanish caves – Kaite and Cueva del Cobre – exhibit deteriorating climatic conditions starting around AD 150/1800 yrs BP (figures 12 and 13).⁹² North of the Empire in Sweden, low-resolution data from the Coral Cave show a drop in temperatures around the second century AD, and a larger drop in precipitation around the same

⁹¹ Fowler *et al.* 2008; Diaz and Markgraf 2010; McCormick *et al.* 2012.

⁹² Martin-Chivelet *et al.* 2011, 5-8; Cruz *et al.* 2015.

time (figures 15 and 16), while at the Spannagel Cave in Austria we see minor dips in temperature (Fig. 17).⁹³

Moving to the eastern Empire, stalagmite records from the three Turkish caves show similarities for the latter half of the second century AD, revealing that, compared to the period immediately preceding it, a colder and drier period commenced. At Uzuntarla in northwest Turkey, excess water (abundant rainfall) dropped considerably, while the temperature data show only minor reductions (Fig. 19).⁹⁴ East of Uzuntarla, the Sofular-Cave data display the start of a large drop in effective moisture (a proxy for temperature and/or humidity), suggesting that north-central Turkey began to get very cold and arid in the later second century (figures 17 and 20).⁹⁵ In southwestern Turkey, after a long and harsh cold period at the Kocain Cave from 800 BC until the later second century BC, the area got warmer (Fig. 21). However, that only lasted until the first century AD when it got cold again and stayed cold until late-second century.⁹⁶ Southeast of Kocain Cave, stalagmite data from the Soreq Cave in Israel show decreasing rainfall in the first half of the second century, sharply falling for the rest of the second century, into the third century and beyond.⁹⁷

Lake-sediment records

The lake-sediment data vary considerably between AD 150 – 200. Revisiting the data from Zonar Lake in Spain reminds us that climatic deterioration post-AD 150 was

⁹³ For the Coral Cave see Sunqvist *et al.* 2010; for Spannagel: Mangini, Spötl, and Verdes 2005; McCormick *et al.* 2012.

⁹⁴ Göktürk 2011, 72-77.

⁹⁵ Göktürk 2011, 14-35.

⁹⁶ Göktürk 2011, 54-58.

⁹⁷ Orland *et al.* 2009, 32-34.

not universal throughout the Mediterranean region. The arid phase lasting for the entirety of the so-called RCO phase in Spain ended in AD 150 and a wet/humid phase began.⁹⁸ An intense flooding episode occurred from around AD 150 – 160, followed by a shift to moderate precipitation, warmer temperatures, and overall humid conditions.⁹⁹ The conditions revealed by sediment data at Zonar Lake are in direct contrast to the Spanish stalagmite data for the period between AD 150 – 200. However, the stalagmite data is from the northernmost part of Spain, while Lake Zonar is in southern Spain, revealing the potentiality for differing climatic zones within the same region.

Like the data from Zonar Lake, the data from the 26 lakes in the Jura Mountains in Switzerland reveal higher lake levels for AD 150 – 250, suggesting wetter and more humid conditions in the Swiss region for the post-RCO era.¹⁰⁰ The data for Lake Shkodra in Albania are similar – wetter conditions that begin in AD 150 and last until 250 (Fig. 24).¹⁰¹

The lake-sediment stratigraphies so far have opposed the idea that the RCO ends at AD 150. However, upon reaching the eastern Empire, that changes. Data from Lake Van in Turkey show an arid phase starting early in the latter half of the second century and continuing into the third century and beyond (Fig 25).¹⁰² As mentioned, Bereket Basin in eastern Turkey shows extreme aridity for the period in question, part of the “driest episode during Roman times” at Bereket; however, anthropogenic disturbances may have added to the dryness recorded around the

⁹⁸ Martín-Puertas *et al.* 2009.

⁹⁹ Martín-Puertas *et al.* 2009, 116.

¹⁰⁰ Magny 2004, 77.

¹⁰¹ Zanchetta *et al.* 2012.

¹⁰² McCormick *et al.* 2012, 184.

Bereket Basin.¹⁰³ South of Turkey, evidence from the Dead Sea (along with data from Soreq Cave) infers that areas of Israel were in the midst of an arid phase, but conditions started to improve around the lake in the third century AD (Fig. 25).¹⁰⁴

Most lakes of East Africa show aridity for the second century AD. Lake Naivasha, Lake Edward, Sacred Lake, Lake Tanganyika, and Lake Challa all show drying, indicative of reduced monsoonal rainfall in this region, until at least AD 200.¹⁰⁵ At Chew Bahir, we do not see drying in the record, but a plateau of moderate wetness continuing from the RCO phase. However, the majority of proxies in the region suggest that the RCO ended in the second century in East Africa.

Failed Nile floods

As mentioned, Nile-flood data are another source that helps relate the climatic situation in Egypt and East Africa, and thus the environmental landscape along the Nile river. Based on the flood reports, conditions in Egypt in the latter half of the second century AD were not good. McCormick and company's synthesis of the flood data compiled by Bonneau in the 1970s reveal a much larger proportion of low-quality floods post-AD 156 (Fig. 27).¹⁰⁶ In addition, we have official reports (related to taxation) of failed Nile flooding (*abrochia*) on public and private land, showing that a large proportion of failed floods occurred in the AD 160s; eleven reports of the failed flood in AD 164 (Fig. 30).¹⁰⁷ Both sets of data indicate the environment was

¹⁰³ Kaniewski *et al.* 2007, 2212.

¹⁰⁴ Migowski *et al.* 2006, 423.

¹⁰⁵ Nash *et al.* 2016, 8.

¹⁰⁶ McCormick *et al.* 2012, 189-194, based on Bonneau 1971.

¹⁰⁷ Habermann 1997, 151-152; Bruun 2007, 206; Elliott 2016, 26-27.

changing in Egypt, suggesting it was a turbulent 160s, and possibly half-century, in the agriculturally-invaluable southern province.

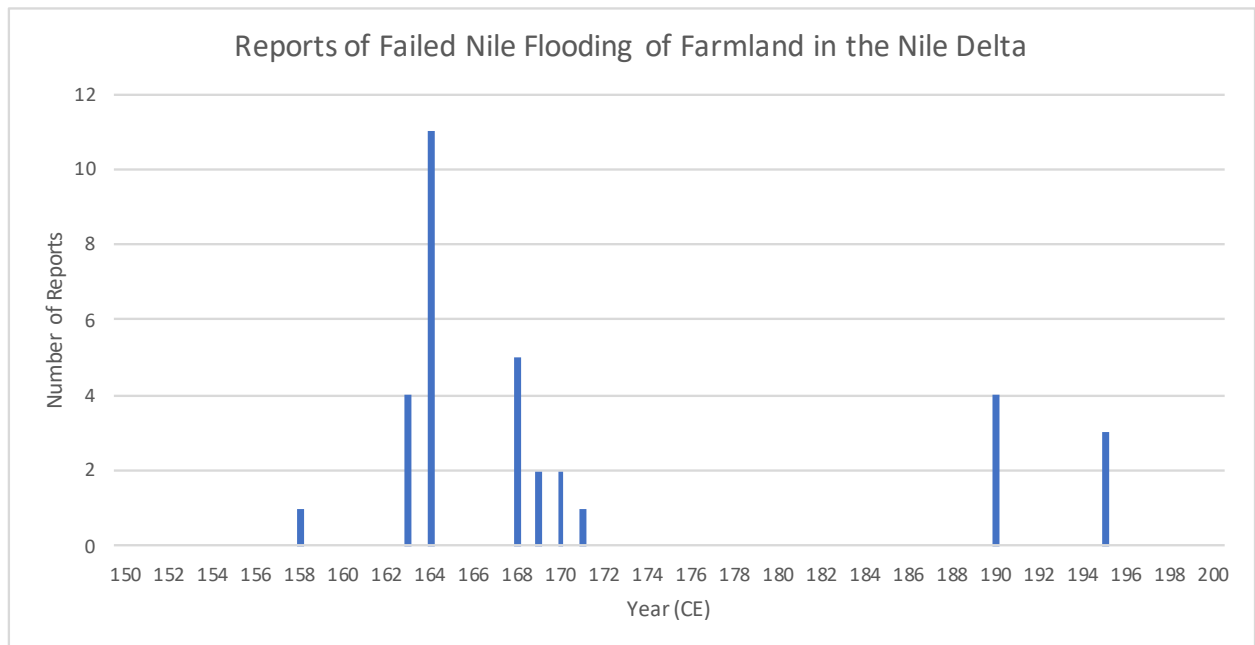


Figure 30. Reports of failed Nile flooding (abrochia) in the Delta.
(Data: Habermann 1997; my figure)

Glacial evidence

Data from the Great Aletsch glacier in Switzerland reveal the beginning of a cold phase around AD 100 (Fig. 28). The glacial advance continued through the entirety of the second century and into the third century, suggesting that there was an unceasing cold phase for this region of Europe in the second century.¹⁰⁸ However, both the Gorner and Lower Grindwald Glaciers of the same region show no signs of advance; although the data from these two glaciers produce much lower resolutions (Fig. 28).¹⁰⁹ The Mer de Glace glacier (France) also lacks the resolution for a definitive reading for AD 150 – 200, as many moraines (glacial stratigraphies) provide only

¹⁰⁸ Holzhauser 1984; Holzhauser, Magny, and Zumbühl 2005.

¹⁰⁹ Holzhauser, Magny, and Zumbühl 2005, 791.

partial evidence of advances.¹¹⁰ However, based on the near-peak of an advance in the early fourth century AD, Le Roy *et al.* suggest a glacial-advance trajectory that began in the late-first/early-second century AD, similar to the Great Aletsch (Fig. 29).¹¹¹ To the west in Austria, data from the Gepatschferner Glacier suggest an advance through all of the second century, straight on to the fourth century.¹¹²

Discussion

The tree-ring data from the Alps and the Altai mountains indicate substantial cooling and moderate aridity in central Europe, central Asia, and possibly most of Eurasia, for AD 150 – 200 – particularly from the AD 160s onward – and the data from the Libyan Sahara show a second-century arid phase that began earlier in the century with a late-century recovery. While variable in degree, the stalagmite data do mostly show climatic deterioration around the Roman world post-AD 150, with data from Spain, Turkey, and Israel standing out. The lake-sediment records are most variable, with lakes in Spain, Switzerland, Albania, and Israel all exhibiting wet conditions, while lakes in Turkey, Israel and eastern Africa show aridity. The difference in conditions between lakes in the western Empire and lakes in the eastern Empire is apparent. The Nile-flood data are striking and suggests probable aridity in eastern Africa in the latter half of the second century AD. Lastly, much of the glacier data is not definitive, but the data that is points to a second-century cold phase.

Overall, the data show that the northern and western parts of Empire were experiencing a cold phase between AD 150 – 200, but were not suffering from

¹¹⁰ Le Roy *et al.* 2015, 2.

¹¹¹ Le Roy *et al.* 2015, 9-10.

¹¹² Nicolussi and Patzelt 2001; McCormick *et al.* 2012, 181.

aridity, as wetness and humidity persist in many regions. In the eastern Empire the situation was bleaker, as virtually all of the records reviewed exhibited cooling and aridity.

The eruptions around AD 163 and 170 probably played a role in the cooling and dry spells. Some of the proxies, such as the central Asian tree-ring data and the eastern stalagmite data, show cooling and aridity soon after the 163 eruption, with further reductions following the 170 eruption. Recalling the analysis by Sigl et al. 2015, it is common to see climatic deterioration immediately following large volcanic eruptions (figures 3 and 4).¹¹³ Nowhere is this more apparent than in the Nile-flood data for the AD 160s, with eleven reports of a failed flood (*abrochia*) in year 164, the year after the eruption. Then, approximately seven years later, the second, larger eruption occurred, and cooling around the Empire only continued, not showing recovery in most of the proxies until the late-second century, or later. This cold and arid phase happening across much of the Roman world in the latter half of the second century AD could have serious implications for the Antonine Plague (AD 165 – 180s), the topic of the next chapter.

Third Century AD: ups and downs

A large portion of the climatic data show a temperature recovery in the early third century. The recovery generally lasts for at least the first half, if not all, of the third century, with data displaying reductions in temperature either around AD 250 (potentially significant for the Plague of Cyprian, AD 251- 270), in the late-third

¹¹³ Sigl et al. 2015, 546-7.

century, or in the early-fourth century. But we will focus only on the third century here to gauge the climatic situation surrounding the third-century pestilence.

The data

The tree-ring data from central Europe and central Asia show an improvement in temperatures from the end of the second century until around the mid-AD 260s (figures 7 and 8).¹¹⁴ The reductions are probably not a coincidence. Recalling the eruption occurring around AD 266 (figures 3 and 4), temperatures drop to their lowest point since the AD-160s event in the central Asian and ENSO-sensitive data (Fig 10). The tree-ring data from Libya, however, differ, showing a reduction in temperatures for the first half of the third century (Fig. 9).¹¹⁵ The stalagmite data from Spain show a continuing cold phase, and a striking drop in moisture at the Kaite Cave (figures 12 and 13).¹¹⁶ The Coral Cave in Sweden exhibits rising temperatures and precipitation, while the Spannagel-Cave data (Austria) show steady temperatures until a sharp drop in the 260s, like the tree-ring data (figures 15, 16, and 17).¹¹⁷ The Turkish stalagmite data all show improving or steady conditions (figures 17, 18, and 19) – apart from a large drop in temperatures at Uzuntarla in northwestern Turkey – and the reductions in precipitation that began at the Soreq Cave in the second century level out around AD 250, beginning to rise again in the later-third century.¹¹⁸

¹¹⁴ Büntgen *et al.* 2011, 581; Büntgen *et al.* 2016, 233; Fowler *et al.* 2008; Diaz and Markgraf 2010; McCormick *et al.* 2012.

¹¹⁵ Cremaschi, Pelfini and Santilli 2006; Nash *et al.* 2016, 6.

¹¹⁶ Martín-Chivelet *et al.* 2011; Cruz *et al.* 2015.

¹¹⁷ Sundqvist *et al.* 2010; McCormick *et al.* 2012.

¹¹⁸ Göktürk 2011; McCormick *et al.* 2012; Orland *et al.* 2009, 32.

Similar to the instability exhibited in the Spanish stalagmite data, data from Zonar Lake show a continuing phase of aridity, suggesting Spain was cold and arid for all of the third century. Lakes in the Swiss Jura Mountains were high from AD 150 – 250, dropping to moderate-to-low levels for the rest of the century, while the Albanian lake Shkodra exhibits moderate water levels for all of the third century.¹¹⁹ Aridity is apparent in the east, as data from Lake Van (eastern Turkey), the Bereket Basin (southwestern Turkey), and the Dead Sea (Israel) all display a lack of wetness for the third century (Fig. 25).¹²⁰ In contrast, from AD 200 the lakes of East Africa experience wetter conditions that result in a high-stand around AD 600 (more on this below).¹²¹

The Nile flood reports suggest flooding was moderate-to-poor in the first half of the third century, as there are more reports of low-quality floods than the opposite (Fig. 27).¹²² However, reporting becomes far less frequent in the third century, and thus the data set is less useful from the third century onward. Similarly, more than half of the glacier data – the Gorner, Lower Grindewald, and Mer de Glace – indicate no glacial growth, while data from the Great Aletsch (Switzerland) show a steepening advance in the third century.

From a macro perspective, the Empire's climate in the first half of the third century was more favourable than the second half. This is evident in nearly all of the climatic proxies, aside from those, such as in Spain, Turkey, and Libya that show cooling or aridity for most of the century, and East Africa, which shows wetness for

¹¹⁹ Martin-Puertas *et al.* 2009; Zanchetta *et al.* 2012; Magny 2004.

¹²⁰ McCormick *et al.* 2012; Kaniewski *et al.* 2007; Migowski *et al.* 2006.

¹²¹ Nash *et al.* 2016,

¹²² McCormick *et al.* 2012, 203; Bonneau 1971.

the entire century. Some of the data exhibit climatic deterioration just after AD 250, possibly assisted by the large reductions in solar activity, and the rest show conspicuous variability around the AD 260s, probably caused by the powerful mid-260s eruption. In chapter three, we will look at whether or not the climatic situation just reviewed relates to the emergence and extent of the Plague of Cyprian (AD 251 – 270).

Sixth Century AD: a tempestuous end

Emerging from a relatively quiet fifth century, the Empire experienced probably the largest climatic shock it had yet seen. A string of volcanic eruptions occurred in AD 536, 540, and 547, playing a significant role in the onset of the so-called Late Antique Little Ice Age (LALIA). In particular, the first two eruptions in AD 536 (unattributed) and 540 (Ilopango; Fig. 5) were large enough, and happened close enough in time, that by AD 541 temperatures had dropped to extraordinary lows, as evidenced by most climatic proxies.¹²³ Then the third eruption at 547 helped thwart a seeming climatic rebound. Also apparent are spikes in wetness in the arid and semi-arid climates of the Empire; in such regions this can occur immediately after dramatic cooling, such as that following large volcanic eruptions.¹²⁴ Reductions in Total Solar Irradiance in the sixth century may have also contributed to the onset and endurance

¹²³ Dull *et al.* 2019.

¹²⁴ Stenseth *et al.* 2006, 13111; McConnell *et al.* 2020.

of the LALIA, but the eruptions were the major players in this climatically-violent century.

The LALIA lasted into the seventh century, as climatic conditions in most regions took over 100 years to return to pre-AD 536 levels. Parts of central and western Asia had a particularly drawn-out LALIA, where temperatures did not fully rebound until the eighth century. This was partly caused by a late seventh-century eruption (probably the volcano Pago in Papua New Guinea), which was felt in other parts of the Roman world, but nowhere as markedly as in Asia.¹²⁵ However, here we will focus on the first half of the sixth century to determine if the climatic picture discussed below was a factor in the emergence of the Justinianic pandemic.

AD 500 – 550

For much of the Empire, the sixth century AD started off as the fifth century ended, with temperatures on the rise or at moderate levels. That changed in AD 536. The tree-ring data from the Alps show a substantial reduction to temperatures around 536, followed by a plummet around 541 (Fig. 7).¹²⁶ The precipitation data from central Europe show reductions that began in the late fifth century – which cannot have been caused by the eruptions – and remained low for most of the sixth century.

In Spain, the stalagmite data show reductions in both temperatures and wetness, especially at the Kaite and Cueva del Cobre caves (figures 12 and 13).¹²⁷ The Spanish lake Zonar continued an arid phase that began in the first century AD.¹²⁸

¹²⁵ Sigl *et al.* 2015, extended data table 4; Büntgen *et al.* 2016.

¹²⁶ Büntgen *et al.* 2011.

¹²⁷ Martín-Chivelet *et al.* 2011; Cruz *et al.* 2015.

¹²⁸ Martín-Puertas *et al.* 2009; Zanchetta *et al.* 2012.

Similarly, data from the lakes in the Jura Mountains in Switzerland have a continuation of low lake-levels until AD 650.¹²⁹ Data for every Swiss glacier – Great Aletsch, Gorner, Lower Grindewald, Mer de Glace – display massive advances after AD 536 (figures 28 and 29).¹³⁰ Just east in Austria, the Spannagel-Cave data show a steady temperature-descent at the beginning of the century, and a sharp drop in the 530s that amounted to a century low in temperatures around 550 (Fig. 17).¹³¹ 700 miles south, in Albania, wetness rises sharply after the eruptions (Fig. 24).¹³² And rounding out the European data, temperatures decrease slightly at Uzuntarla Cave (the European side of Turkey), while excess water spikes at Uzuntarla after the eruptions (Fig. 19).¹³³

In Asia, the tree-ring data from central Asia reveal the second-largest reduction in temperatures of that series, just behind that of the AD 160s, but a much slower full recovery (Fig. 8).¹³⁴ The tree-ring evidence from the Libyan Sahara exhibits a significant spike in wetness around when the eruptions occurred, dropping to moderate levels post-AD 550 (Fig. 9).¹³⁵ The ENSO-sensitive tree-ring data from New Zealand and New Mexico –used to infer climate for central Asia and Eurasia – show rises in wetness from AD 536 – 550 (Fig. 10).¹³⁶

For Turkey, the Sofular Cave reveals a virtual moisture-plateau for all of the sixth century (figures 17 and 18).¹³⁷ Lake Van exhibits moderate moisture in the sixth

¹²⁹ Magny 2004, 77.

¹³⁰ Zumbühl 1980; Holzhauser 1984; Holzhauser 2001; Holzhauser, Magny, and Zumbühl 2005; Le Roy *et al.* 2015.

¹³¹ Mangini, Spötl, Verdes 2005; McCormick *et al.* 2012.

¹³² Zanchetta *et al.* 2012

¹³³ Göktürk 2011, 76.

¹³⁴ Büntgen *et al.* 2016.

¹³⁵ Nash *et al.* 2016, 6.

¹³⁶ Fowler *et al.* 2008; Diaz and Markgraf 2010; McCormick *et al.* 2012.

¹³⁷ Göktürk 2011, 28.

century (Fig. 25), and sediment deposits and local marsh vegetation at Bereket Basin suggest wetter conditions.¹³⁸ In southwestern Turkey, data from the Kocain Cave show a sudden spike in cooling around AD 540, lasting until the later sixth century (figures 19 and 21).¹³⁹ And to the south in Israel, Dead Sea lake-levels plummeted at some point around the mid-sixth century, but it is not clear exactly when (Fig. 25), and the stalagmite data from Soreq Cave show a 200mm reduction in precipitation early in the sixth century with no increase until centuries later.¹⁴⁰ In East Africa, most lakes around the Ethiopian Highlands (Lake Naivasha, Lake Tanganyika, Lake Ashenge, Lake Turkana, and Lake Chew Bahir) experienced highstands until around AD 550-600, indicating that high levels of precipitation most likely followed the eruptions; this was particularly apparent at Lake Chew Bahir, the data for which show a significant rise in wetness just after the 540 eruption (Fig.26).¹⁴¹ The moisture trends in East Africa may have implications for the first outbreaks of the Justinianic Plague (Chapter 4).

Climatically-speaking, AD 500-550 might be the most homogenously unstable half-century in the history of the Roman Empire. Nearly every data set reveals climatic perturbation around AD 540, an apparent result of the two massive eruptions, which might also have been aided by decreases in solar activity. At around AD 541, one of the coldest years in Roman history, the Justinianic Plague begins. In Chapter 4, we will focus on the emergence of the Justinianic Plague, its impact on the Empire, and the epidemiology of the disease, assessing whether the

¹³⁸ McCormick *et al.* 2012; Kaniewski *et al.* 2007, 2213.

¹³⁹ Göktürk 2011, 54.

¹⁴⁰ Migowski *et al.* 2006; McCormick *et al.* 2012, 197.

¹⁴¹ Nash *et al.* 2016, 8; Foerster *et al.* 2015, 336.

severe climatic events and environmental responses in the sixth century helped give rise to “the great plague”. We will also look at the impact this climatic phase had on Roman society and consider whether observable downturn should be attributed to climate change or the plague.

Conclusions

It does seem accurate that the Romans were in the midst of a “climate optimum” during their transition from republic to empire. Whether this span of beneficial weather played a role in the progression of the Empire remains to be systematically proven; but it certainly did not hurt them. The agricultural economy was the Empire’s lifeline, and a favourable climate was most necessary for prosperity. This seems to have been put at risk for a short period in the mid-first century BC, in the latter half of the second century AD, in the later third century AD, and in the years between AD 536 – 550. However, climate not only may have affected the Empire’s ecology via environmental deterioration, it also may have instigated disease. The three major plagues of Roman times happened (at least partly) during climatically unstable periods. The goal of the subsequent chapters is to examine these pandemics – through epidemiological studies of the diseases thought to be responsible – and to assess the likelihood of whether or not the phases of climatic change we have just reviewed played a role in their rise and spread. In addition, the succeeding chapters include discussions on the influence of the pestilences on the ancient world. In some cases, it is difficult to disentangle the effects of significant climate change (as well as other issues in the case of the third century AD) from the impact of pandemic disease. Further complicating that assessment, the changing climate may have given

rise to two of the pandemics, and it is implicated in the question of impact that way. Nevertheless, those sections work to home in on the effects of the pandemics themselves. However, it becomes apparent that in two of the three cases climate change may have played a direct role in the economic decline we observe occurring at the same time as the pandemics.

The Antonine Plague

Introduction

Written evidence has led scholars to accept that a major epidemic raged through the Roman Empire from around AD 165 until the late second century, which has become known in the modern literature as ‘the Antonine Plague’. Scholars continue hotly to debate the severity of its effects on the Roman empire – estimates range from devastating (c. 30% population loss or more) to minimal (c. 1% population loss); but the fact of its existence is not in dispute.

Galen, Aelius Aristides, Lucian/Crepereius Calpurnianus, Herodian, Cassius Dio, and some papyrological documents are the main sources for the Antonine Plague.¹⁴² These are typically cited as the central sources, as all are contemporary with the pandemic. They offer some insight into the nature of the disease, as well as its symptoms (Galen) and the devastating effects the disease had on towns and the Empire as a whole (Herodian and Cassius Dio); one author even contracted the plague (Aristides). Later sources, like the *Historia Augusta*, also mention the pandemic’s impact. Galen, the physician, surgeon, and philosopher from Pergamon, is most useful for relating the immediate effects suffered by a person who contracted the disease. Galen had first-hand knowledge of the disease, since he was in Rome when it arrived in AD 166, and in Aquileia (northern Italy) in AD 168-169 where the

¹⁴² Galen, *Method of Medicine*, 5.12, Loeb edition trans. Ian Johnston and G.H.R. Horsley; Cassius Dio, *Roman History*, LXXIII.14.3-4, Loeb edition trans. by Earnest Cary; Herodian, *Roman History*, I.12.1, trans. by C.R. Whitaker; Aelius Aristides, *Orations* 48.38-44, translated by C.A. Behr; Lucian, *How to Write History*, 15, trans. by A.M. Harmon, K. Kilburn, and M.D. Macleod.

plague swept through the legions stationed there.¹⁴³ His description is viewed by most scholars as crucial in diagnosing the disease as smallpox (*Variola major* or *Variola minor*).¹⁴⁴ Since we lack aDNA evidence from second century AD-human remains that confirms the etiological agent of the disease, our diagnosis is mostly based on Galen's description of the symptomology, leading most scholars to believe the Antonine Plague was smallpox (the *variola virus*) – a diagnosis first proposed by Heinrich Häser in 1881 and robustly argued for by Littman and Littman in 1973.¹⁴⁵ However, if smallpox, the Antonine Plague may have been a more basal form of the disease, but was most likely comparable biologically if the symptoms were similar. In contrast to Galen, Aristides relates how he himself contracted the plague in Smyrna (western Turkey) in AD 165 – 166, while Cassius Dio and Herodian write of the pestilence's larger effect on the people of Rome and the Empire towards the pandemic's end in the AD 180s, stressing the disease's demographic harshness rather than the specificity of its symptoms.¹⁴⁶ Lucian, a rhetorician and satirist, also alludes to the Antonine Plague, remarking how the historian of the Parthian War, Crepereius Calpurnianus, writes about the pandemic reaching Nisibis in AD 165, which could be the earliest mention of the plague in the Roman Empire. The pestilence is said to have originated in the *Historia Augusta* in Babylonia – brought back to the Empire by the army of Lucius Verus in AD 166 – but this work is

¹⁴³ Galen, *Method of Medicine*, 5.12, Loeb edition trans. by K. Kilburn; Littman and Littman 1973, 244; R. P. Duncan-Jones 1996, 124.

¹⁴⁴ Littman and Littman 1973, 243-255; Lo Cascio 2012.

¹⁴⁵ Häser 1881, 24-33. The most recent historians subscribing to the theory that the Antonine Plague was smallpox are Harper 2017, 102; and Elliot 2016, 5; the numerous contributors of the volume edited by Lo Cascio in 2012 also suspect this plague was smallpox; see additionally Littman and Littman 1973, 243-255; Zelener 2012; Cunha and Cunha 2008, 12; Sallares 2007a, 37; Scheidel 2002, 95.

¹⁴⁶ Cassius Dio, *Roman History*, LXXIII.14.3-4; Herodian, *Roman History*, I.12.1.

notoriously unreliable.¹⁴⁷ One papyrological document suggests that the Antonine Plague reached and probably had a considerable effect in the Nile Delta, while a papyrus letter often cited as evidence of the Antonine Plague in Egypt is more likely from the period of the Plague of Cyprian.¹⁴⁸

Galen's account of the Antonine Plague stands out as the most important and influential piece of literary evidence we have for identifying the epidemic of the later second century AD. While Littman and Littman believe Galen's description is in some ways 'sketchy', they feel it offers enough pertinent information to identify the disease as smallpox; however, their diagnosis has recently been called into question.¹⁴⁹ Other than Galen's description, historians have little additional written evidence for the Antonine Plague that would either help make a diagnosis or suggest the extent to which the plague affected the Empire. Nor do we have much evidence that the plague reached certain parts of the empire which historians seem to think were ravaged by it; in particular, there is surprisingly little unambiguous evidence for the plague from the comparatively well-documented Roman province of Egypt.

The main goal of the chapter is to examine whether second-century climate change may have been a factor linked with the Antonine Plague, and if so, how. In particular, I examine how the climatic cooling and aridity we observed in Chapter 1 disposed certain regions from Europe to Asia to conditions that were prime for the transmission and persistence of smallpox; as we will see below, smallpox perseveres and spreads better in more moderate climates.

¹⁴⁷ *Historia Augusta*, Lucius Verus, 8.1-8.5.

¹⁴⁸ *P. Thmouis* 1.104; Blouin 2014, 64-65; *P. Oxy* 14.1666, trans. by Grenfell and Hunt 1914, 121-123

¹⁴⁹ Littman and Littman 1973, 245; Flemming 2019, 219-244.

As we saw in Chapter 1, the scientific data on climate change during the Roman period show that in the AD 160s average temperatures in central Europe dropped over 1°C.¹⁵⁰ Sigl and colleagues' adjusted Greenland and Antarctic ice-core chronologies indicate that two volcanic eruptions, happening around AD 163 and AD 170, caused dramatic shifts in the global climate, explaining the drop in temperatures of 1°C-4°C attested in central Europe and central Asia, and probably elsewhere.¹⁵¹ The beginning of this cool period correlates well with the outbreak of the Antonine Plague. I shall argue that mid-second-century-AD climate change brought on by large volcanic eruptions lowered regional temperatures throughout the Mediterranean region and Asia by between 1°C-4°C, allowing the disease-causing agent of the Antonine Plague to reach the Roman Empire from central Asia.¹⁵²

The first section of the chapter summarizes the evolution of the modern historiography of the Antonine Plague. In the second section, I discuss the ancient written evidence for the second-century pandemic. The third section examines the epidemiology of smallpox and why most scholars believe the Antonine Plague was the *variola virus* or something closely related to it. In the fourth section, I explore the origin and spread of the Antonine pandemic, considering both epidemiological and geographical origins of the disease, and whether or not the pestilence had a devastating effect in Africa through the lens of second-century-AD Roman Egypt. In the fifth section, I consider an explanation for the origin of the virus' spread, looking

¹⁵⁰ Büntgen *et al.* 2011, 581.

¹⁵¹ Sigl *et al.* 2015, 544; original ice core chronologies were inaccurate and skewed, see Larsen *et al.* 2008. For discussion, see Chapter 1.

¹⁵² Regional changes in temperature during the second century is explained by Büntgen *et al.* 2011, 578-582; Sigl *et al.* 2015, 543-549; and Büntgen *et al.* 2016, 1-7.

at the effects of climate on the epidemiology of the disease to determine if climate change of the second century AD provoked a pandemic. And in the sixth and final section, I consider the impact of the Antonine Plague through archaeological and proxy evidence. Using science as the principal lens with which to view the latter half of the second century AD, I hope to firmly add climate change as a major factor affecting the Empire in the second century; one that appears to be linked directly to the extent of the Antonine pandemic.

Historiography

The historiography of this pandemic has oscillated between views of opposing extremes regarding the severity of its impact.

Gibbon paid little attention to the Antonine Plague in *Decline and Fall*, demonstrating that he did not view it as a transformative event.¹⁵³ Barthold Georg Niebuhr and Otto Seeck were the proponents of the idea that the Antonine Plague was central to the disintegration of the Empire.¹⁵⁴ Seeck asserted that over half of the Roman Empire succumbed to the disease, and nearly forty years later Niebuhr claimed, “This pestilence must have raged with incredible fury; and it carried off innumerable victims...The ancient world never recovered from the blow inflicted upon it by the plague.”¹⁵⁵ By contrast, James Frank Gilliam was one of the first to question this view, in 1961. Gilliam argues that contemporary historians such as H.M.D. Parker, A.E.R. Boak, and even the *Cambridge Ancient History*, ascribed too

¹⁵³ Gibbon 1806.

¹⁵⁴ Gilliam 1961, 225-226.

¹⁵⁵ Seeck 1911, 398-405; Niebuhr 1849, 251; Gilliam 1961, 225-226.

much significance to the plague as a factor in the decline of the Empire.¹⁵⁶ While Gilliam concedes that there was a great epidemic during the reign of Marcus Aurelius, and that it probably caused more deaths than other epidemics before the third century, he shows that much of the ancient literature illustrates the effects of the disease as “highly colored and extravagant,” cautioning scholars to question the accounts of its impact related by the ancient literature.¹⁵⁷ Rostovtzeff, similar to Gibbon, had also assigned less importance to the plague as a factor in the decline of the Empire. Gilliam, however, is one of the first to systematically formulate a plausible theory arguing that the plague was not as destructive as historians like Niebuhr and Seeck have suggested.¹⁵⁸

In 1996, Richard Duncan-Jones attempted to put the argument on a better evidential footing. Alongside the written evidence, Duncan-Jones examines papyrological evidence that show signs of agricultural decline in Egypt at the time of the Antonine pandemic. He also quantifies time-series proxy data showing that there are gaps in documentation, as well as a decline in building, inscriptions, and minting in the later second century, all of which he intimates were caused by the pandemic.¹⁵⁹ However, much of the evidence is broad, is not uniform throughout the Empire, and we see similar gaps in other periods well before the Antonine Plague.

Walter Scheidel’s article in the *Journal of Roman Archaeology* in 2002 builds on Duncan-Jones’ analysis, presenting a model that predicts the effects of the Antonine Plague on Roman Egypt. He uses evidence for land rents and prices, commodity

¹⁵⁶ Gilliam 1961, 226; Parker 1958, 20; Boak 1974; Bowman, Garnsey and Rathbone 1982, 348-349.

¹⁵⁷ Gilliam 1961, 250.

¹⁵⁸ Gilliam 1961, 226.

¹⁵⁹ Duncan-Jones 1996, 107-136.

pricing, and rural wages to argue for a period of economic decline at the time of and just after the pestilence.¹⁶⁰ Scheidel is not explicit in saying that the decline experienced in Egypt was the case throughout the Empire, but the idea is hard to avoid when reflecting on his analysis. In the same volume, an article written by Roger Bagnall follows Scheidel's, and questions the practicality of Scheidel's model.¹⁶¹ However, Bagnall's article serves more as a caution for modelling based on papyrological data than it does to disprove the message Scheidel's model suggests.

The following year, two separate articles were published in the *Journal of Roman Archaeology*, contesting the evidence presented by Duncan-Jones and Scheidel. These articles, by James Greenberg and Christer Bruun, reconsider the impact of the Antonine Plague, essentially suggesting that the evidence presented by Duncan-Jones and Scheidel is flawed. Greenberg questions each of the proxies used by Duncan-Jones' and Scheidel, arguing that the sample sizes are too small, that we observe similar downturn earlier in the second century, and that decline could just as easily be attributed to war.¹⁶² Bruun argues that Duncan-Jones' series of building inscriptions is incomplete, and that decline is not apparent in Ostia after the Antonine Plague, which would be expected if Rome was hit hard by the pandemic.¹⁶³ Greenberg and Bruun are not suggesting the plague did not cause decline throughout the empire, but they are asserting that we cannot infer its impact using the evidence provided by Duncan-Jones and Scheidel.

¹⁶⁰ Scheidel 2002, 97-114.

¹⁶¹ Bagnall 2002, 114-120.

¹⁶² Greenberg 2003, 413-425

¹⁶³ Bruun 2003, 426-434.

In 2007, Rossignol and Durost published their study “Volcanism global et variations climatiques de courte durée dans l’histoire romaine”, in which they investigate volcanic eruptions in the mid-second century AD, and how environment and agriculture were suffering during the pandemic.¹⁶⁴ In addition, they detail evidence for disease and decline in Han China – first pointed out by McNeil in 1976 – questioning whether it is purely coincidence that these two major world-powers suffered similar turmoil in the second century AD.¹⁶⁵ They consider the idea that climate variation of the AD 160s might have encouraged the westward spread of the disease, noting that smallpox seems to spread more easily in cooler months.¹⁶⁶ While they do ponder whether colder conditions might have allowed a disease to spread from China to Roman boundaries, this is as far as their investigation goes.

In 2012, Elio Lo Cascio published a volume titled, *L’Impatto della Peste Antonina*, which contained a number of articles from scholars working on the Antonine Plague.¹⁶⁷ The papers examine a range of different topics concerning the plague during the reign of Marcus Aurelius, including: a review of the papyrological evidence, a re-reading of the literary sources, the effect of the plague on the Roman army, climate and famine in the context of the plague, epidemiological modelling, the rate of mortality before and during the plague, and economic change brought on by the plague.¹⁶⁸ Rossignol’s article in that volume again touches on Han China being the possible point of origin of the Antonine Plague, as well as suggesting that

¹⁶⁴ Rossignol and Durost 2007, 414-415.

¹⁶⁵ Rossignol and Durost 2007, 420-421; McNeill 1998, appendix.

¹⁶⁶ Rossignol and Durost 2007, 420-421.

¹⁶⁷ With important articles from E. Lo Cascio, W. Scheidel, W.V. Harris, C. Bruun, W. Eck, B. Rossignol, and Y. Zelener.

¹⁶⁸ Lo Cascio 2012.

climate change might have been the stimulus behind the rise and spread of the plague.¹⁶⁹ However, again, he investigates no further.

The most novel article in Lo Cascio's volume is by Yan Zelener, in which he examines the genetic history of smallpox, pointing out that smallpox, which may have been the cause of the Antonine Plague, is more closely related to camelpox than to cowpox (the strain originally thought to be the origin of transmission to humans).¹⁷⁰ Zelener's study is significant because it constrains identification of the virus' geographical origin, since camels are found in far fewer parts of the world (Africa and Asia) than are bovine animals.

In 2013, Andrew Wilson challenged the traditional historiography of the Antonine Plague's effect in Egypt, doubting the notion that economic troubles and depopulation in Egypt were caused by the pandemic, and emphasising that the turning point identified by Duncan-Jones could be explained by the failed Nile floods presented in McCormick et al. 2012 (Chapter 1).¹⁷¹ Wilson questions: whether (1) it was a changing climate that affected the Nile flood regime, creating weaker harvests, which combined with the plague to cause economic downturn and depopulation; or (2) was the plague not factor in at all, and instead the troubles in Egypt were solely provoked by poor floods that lead to bad harvests, both a result of climate change?

Colin Elliot's 2016 article, "The Antonine Plague, Climate Change and Local Violence in Roman Egypt," questions models of economic and demographic decline based solely on literary sources and scant papyrological data, similar to Bagnall's

¹⁶⁹ Rossignol 2012, 87-122.

¹⁷⁰ Zelener 2012, 167-177.

¹⁷¹ McCormick et al. 2012, 194; Andrew Wilson 2013, 259-267.

caveat. However, Elliot's doubt comes from another place. Elliot's entire article is essentially a response to the questions raised by Wilson in 2013. Elliot suggests rising Egyptian wages, rising prices, and tax and census documents showing population loss, do not definitively prove that the Antonine Plague severely affected Egyptian society.¹⁷² Elliot responds to Wilson's prompt, providing Nile-flood data and evidence of drought to propose that climate change – possibly provoked by volcanic eruptions – and not a pandemic, was the reason for economic downturn and disruption in Egypt. He also uses evidence of revolts in Egypt to suggest that drought led to uprisings, and proposes that flight due to crop failure or land seizure was the main reason for depopulation.¹⁷³ In Elliot's view, climate change played a larger role in the disruption of second-century-AD Roman Egypt than did the Antonine Plague, and it is hard to disagree. Elliot's article calls into question the nature of plague-impact modelling, using climate-dependent Egypt as an example. Roman Egypt at the time of the Antonine Plague will be discussed in further detail below. While climate change does enter Elliot's discussion about the Antonine Plague, he does not explore its potential impact on the rise or spread of the pestilence.

In 2017, Kyle Harper published his book *The Fate of Rome: Climate, Disease, and the End of an Empire*, proposing that ecological shocks were the impetus behind the Empire's disintegration.¹⁷⁴ His book suggests that climate change and diseases –

¹⁷² Elliot 2016, 3-5; for rising prices in Egypt see Rathbone 1997. For declining supply in labor leading to higher wages: Scheidel 2012, 265-295; Scheidel in De Callatay 2014, 209-218; for information on tax and census documents for Egypt see Blouin 2014, 246-247: which Elliot uses to show "death is explicitly cited as the reason for declines, but 'flight' and ruination' are by far the most common causes listed by officials." A most recent article published by Elliot adds to his analysis from 2016, reaffirming that the Antonine Plague was not as severe in Roman Egypt as most scholars think: Elliott 2020, 297-326.

¹⁷³ Elliot 2016, 27, 28-31; Alston 1999, 129-154; Blouin 2014, 266-270.

¹⁷⁴ Harper 2017.

with the Empire's superior connectivity and penchant for commerce which helped spread the diseases – were the reason for Rome's undoing. Harper dedicates a chapter to the Antonine Plague, discussing the biology, epidemiology, and impact of the pandemic; and while he does dedicate a considerable portion of his book to the effects of climate change on the Empire, he does not examine the effect it may have had on the disease responsible for the Antonine pandemic.

In 2018, shortly after Harper's book came out, a study led by ice-core scientist Joe McConnell measured ancient lead pollution in Greenland ice, indicating that lead mining in western Europe decreased sharply and considerably with the start of the Antonine Plague in AD 165-166 (Fig. 3).¹⁷⁵ While the drop in mining here most certainly represents economic downturn at the time of the plague, it also suggests substantial population loss, since the decline is so dramatic around AD 166. This proxy evidence indicates that the Antonine Plague was substantial in intensity and scope.

Discussion

The current consensus is that the Antonine Plague had an important effect on the Empire. The evidence suggests that most regions of the Empire suffered economic and demographic losses, but in light of Elliot's article from 2016, the plague's impact in Egypt needs to be more thoroughly scrutinised. A goal of this chapter is to expand on Elliot's theory, using new and updated ice-core and paleoclimate records discussed in the previous chapter to argue that climate change may have been the main disrupter of Egyptian civilization in the second century AD.

¹⁷⁵ McConnell *et al.* 2018, 3.

The possible link between climatic events of the 160s – the volcanic eruptions followed by cooling and aridity (Chapter 1) – and the Antonine Plague needs to be properly investigated. As discussed above, there is an exploration of ‘impact’ – all considering the impact of the Antonine pandemic and some also assessing the effect of contemporaneous climate change – but nowhere is a connection between climatic variability and the suspected etiological agent responsible for the pestilence examined. The closest we have come is Rossignol and Durost’s 2007 article, and Rossignol’s chapter in Lo Cascio’s volume from 2012; both of which imply that there may be a link, without thorough analysis. The goal of this chapter is to approach a better understanding of this unexplored question, while also reflecting on ‘impact’ of the pandemic.

Written Evidence

Scholars are still debating the effects of the Antonine Plague today because of the vagueness of the accounts by ancient writers, who indicate the existence of a disease ravaging parts of the Empire but in some cases the references to scope or impact are ambiguous. The lack of physical evidence for the Antonine Plague contrasts with say, the Justinianic Plague, which affected the Empire in the AD 540s – not fully passing until probably the eighth century – and which in 2013 was confirmed as bubonic plague, based on aDNA evidence from skeletons found in sixth-century-AD burials.¹⁷⁶ Since we currently lack usable archaeological remains that reveal aDNA evidence to define the etiological agent of the Antonine Plague, the literary sources

¹⁷⁶ Harbeck *et al.* 2013, 1-8.

remain most important for saying that there was a pandemic, as well as for helping to identify it. In addition to clarifying the symptomology and societal disruption caused by the pestilence, the written evidence helps chronologically trace the geographic spread of the pandemic in the Empire. The accounts tell us, at the very least, when the Antonine Plague reached their region, as well as providing clues concerning the extent of the pandemic throughout the Mediterranean region.

Galen

The least contested and best ancient source on the Antonine Plague is Galen. Galen leads us to believe it had a devastating effect on Rome, as he left the capital in haste soon after the pestilence arrived, for his home city of Pergamon.¹⁷⁸ He also records an outbreak amongst the Roman army stationed at Aquileia in 168, after he was summoned there by the Emperors.¹⁷⁹ It was probably here, in treating infected legionaries during the first Marcomannic War (166 – 175 CE), that Galen became familiar with the symptomology of the disease. Littman and Littman believed that Galen had appreciable experience with the virus, given his specificity in relating its symptoms, and his apparent confidence in suggesting treatment.¹⁸⁰ Therefore, with Galen's medical knowledge, and at least two direct experiences with the virus, we must consider his account of the disease as crucial evidence. However, Littman and Littman correctly indicate that Galen's references to the plague are 'scattered and brief,' since his documentation was focused on treating the disease, not write its

¹⁷⁸ For opinions on why Galen left Rome in haste see: Nutton 1973, 158-161; Mattern 2008, 193-194, Gilliam 1961.

¹⁷⁹ Galen, *On My Own Books*, 19.16-17.

¹⁸⁰ Littman and Littman 1973, 244.

history.¹⁸¹ Nevertheless, overall, Galen is our best source for making a definitive diagnosis.

In his vivid description of throat ulcers related to contraction of the disease, Galen describes the symptoms, their effects, and the duration with which they affect a victim, as well as his treatment of the affliction:

“...those ulcers that occur in the rough arteries (trachea and bronchial tree) in relation to their inner wall, and especially those near the larynx, or even in it, are treatable, and I have cured quite a few patients so affected. In particular, I discovered the cure of these ulcers here at the time of the great plague (would that it will at some point cease), which first came upon us. At that time, a young man broke out in sores over his whole body on the ninth day, just as did almost all the others who were saved. On that day there was also a slight cough. On the following day, immediately after he washed himself, he coughed more violently and brought up with the cough what they call a scab...I also opened his mouth and examined the pharynx in case the sore might be somewhere in this...he felt an irritation in that region to the point of exciting coughing. I then advised him to persevere as far as possible and not to cough, so he did this... applying some of the drying medications externally while lying the patient on his back. Then, having given him a moist medication from among those made for such sores, and having required him to retain this in his mouth, I relied on it flowing into the rough artery (trachea) gradually.”¹⁸²

Galen gives a number of detailed accounts of plague-symptoms similar to the one above, such as descriptions of fever, exanthema (a skin rash that accompanies a disease), bowel movements, vomiting, and violent coughing.¹⁸³ However, he does not consider where the disease came from nor why it was affecting the population in such great numbers. In fact, Galen does not focus much on larger significant themes with regard to the plague. There are just two passages, pointed out by Duncan-Jones, in which Galen alludes to the plague's larger impact: (1) “After I had passed another three years in Rome, when the great plague began, I swiftly returned to [Pergamum] my native city”; and (2), mentioned above, “When I reached Aquileia [in A.D. 168],

¹⁸¹ Littman and Littman 1973, 244.

¹⁸² Galen, *Method of Medicine*, 5.12, trans. by I. Johnston and G.H.R. Horsley.

¹⁸³ Galen, *Method of Medicine*, 5.12, trans. by I. Johnston and G.H.R. Horsley.; Littman and Littman 1973, 246-248; Kühn 1821, 5.115, 10.360, 10.367, 17.1.885, 17.1.709, 17.2.683, 17.1.709.

the plague grew fiercer than ever, so much so that the Emperors immediately went back to Rome with a few soldiers, while we the majority had difficulty in surviving. Most of us died, not merely from the plague, but because the epidemic was happening in the depths of winter.”¹⁸⁴ While he is here not attempting to assess the impact of the plague, we can sense his fear and urgency to avoid the pestilence at all cost, implying its intensity. Galen affords us the most explicit account of the signs and symptoms of the Antonine Plague, faithfully accepted based on his medical expertise and apparent first-hand encounters with the disease, which all gives greater weight to his two brief illustrations of the plague’s larger impact.

Crepereius Calpurnianus in Lucian’s How to Write History

In his *How to Write History*, Lucian discusses how Crepereius Calpurnianus mentions that the Antonine Plague struck Nisibis (Asia Minor) in his now-lost chronicle of the Roman-Parthian war (AD 161 – 166). On the surface, it seems Calpurnianus had knowledge that the plague also struck Roman Egypt, as Lucian quotes him saying, the plague “descended into Egypt” from Ethiopia, on its way to Asia Minor. However, since Calpurnianus was following the Roman-Parthian war from around 162 – 166, and probably witnessed the pestilence not far from Parthia (Nisibis: southeastern Turkey), but claims that it started in Ethiopia, one should question Calpurnianus as a source for the pandemic reaching Egypt. If chronicling the Roman-Parthian war in Asia Minor right around the suspected beginning of the Antonine Plague (AD 165), and close to the region from which most believe the disease was transferred to the Roman Empire (Asia), what would persuade him to

¹⁸⁴ Galen, *Method of Medicine*, 4.78, trans. by I. Johnston and G.H.R. Horsley; Duncan-Jones 1996, 118.

believe the plague began in Ethiopia? Lucian notes the striking similarities between Calpurnianus' narrative and Thucydides' account of the Plague of Athens.¹⁸⁵ He points out identical sentences and phrases in Thucydides, including that the disease "descended into Egypt" from Ethiopia.¹⁸⁶ Lucian says that, while Calpurnianus probably witnessed an outbreak at Nisibis, he plagiarised its presentation, including the epidemic's origin. Ethiopia as a point of origin for plagues was a literary trope of the time, and Calpurnianus assumed the plague had gone through Egypt if it reached Asia Minor. It is probable Calpurnianus invented that part of the story, not knowing where the pestilence he witnessed at Nisibis came from, and thus assumed it originated in Ethiopia.

While Calpurnianus' story on the origin of the Antonine Plague is questionable, it is hard to believe he completely concocted the idea of a plague in Asia Minor – and indeed not even Lucian suggests that he did; especially since we have additional sources like Aelius Aristides and the *Historia Augusta* – both dated after Calpurnianus' work – describing an epidemic in Asia Minor in the mid-to-late-160s AD. However, the dating of Calpurnianus' account as contemporary with the Antonine Plague has recently been disputed. Elliot asserts that because Lucian's work was composed in late 165/early 166, Calpurnianus' work – a chronicle of the Roman-Parthian war of AD 162 – 166 – is too early for him to be describing the Antonine Plague, since, as he claims, 'no scholar dates [the Antonine Plague] from before AD 165', so Calpurnianus must have witnessed an earlier disease.¹⁸⁷ But if Calpurnianus was chronicling the war until 165, is it not possible that he witnessed

¹⁸⁵ Lucian, *How to Write History*, 15, trans. by A.M. Harmon, K. Kilburn, and M.D. Macleod.

¹⁸⁶ Thucydides, *History of the Peloponnesian War*, 2.48.1; Lucian, *How to Write History*, 15.

¹⁸⁷ For the work's date see Wheeler 2010, 14. Elliot 2016, 11.

an early case of the Antonine Plague in the last year of the war? If Aristides contracted the virus at around that time (AD 165-166), why is it that Calpurnianus could not have experienced or learned of the plague too? Thus, Elliot's claim that Calpurnians must have been speaking of an earlier pestilence is not compelling.¹⁸⁸ Currently we do not have evidence of another epidemic in the mid-160s AD. Therefore, it is probable that Calpurnianus' remark was about the Antonine Plague. It is plausible that Calpurnianus' mention is our earliest written evidence of the disease amongst Romans.

Nonetheless, while it appears Calpurnianus might be one of the earliest accounts of the pandemic within Roman boundaries, Calpurnianus via Lucian is not good evidence for the Antonine Plague in Egypt, since Calpurnianus's account of the plague passing through Egypt on its way from Ethiopia to Asia Minor appears simply to be copied from Thucydides, and there is no independent evidence that it started in Africa.

Aelius Aristides

Unlike the medical detail of Galen, Aristides, a sophist from Smyrna, and a lifelong hypochondriac, focuses mainly on his own health in his reference to the Antonine Plague, since he himself contracted the virus.¹⁸⁹ In his *Orations*, Aristides says:

"A plague infected nearly all my neighbours. First two or three of my servants grew sick, then one after another. Then all were in bed, both the younger and the older. I was last to be attacked. Doctors came from the city and we used their attendants as servants...The livestock too became sick. And if anyone tried to move, he immediately lay dead before the front door. There was also terrible sickness in the city...I was attacked by the terrible burning of a bilious mixture...The doctors gave up...and announced that I would die immediately ...little by little with trouble and

¹⁸⁸ Elliot 2016, 11.

¹⁸⁹ Behr 1968; Duncan-Jones 1996, 118.

*difficulty I recovered. The fever did not leave me completely until the most valued of my foster children died.”*¹⁹⁰

In contrast to Galen, Aristides’ account of the plague relates less about characteristic pestilence-symptoms, and more about the extent to which the plague affected his town; probably not much different from the range of infection in other towns throughout the Empire. Additionally, this excerpt relates that the disease at one point affected Smyrna (modern day Izmir, Turkey), along the Ionian Coast. Based on Calpurnianus’s account of plague reaching the army at Nisibis (southern Turkey) and Seleucia (central Iraq) in the summer of AD 165, Behr surmises that the disease must have reached Smyrna soon afterwards in AD 165. Duncan-Jones agrees, and there is currently no reason to believe otherwise.¹⁹¹

Cassius Dio and Herodian

Cassius Dio was 25 years younger than Galen at the time of the pandemic, and he probably wrote his narrative after the pestilence vanished. We have no evidence that Cassius Dio had first-hand experience of the pandemic. Therefore, it is quite possible that, unlike Galen and Aristides, he knew little to nothing about its symptoms. On the other hand, Cassius Dio does lead scholars to believe the pandemic lasted well into Commodus’ reign, as well as suggesting the pestilence was connected with homicidal activity:

¹⁹⁰ Aelius Aristides, *Orations*, 48.38-44, trans. by C.A. Behr.

¹⁹¹ Lucian, *How to Write History*, 15; Behr 1968, 96 and n. 8; Duncan-Jones 1996, 118.

“Moreover, a pestilence occurred, the greatest of any of which I have knowledge; for two thousand persons often died in Rome in a single day. Then, too, many others, not alone in the City, but throughout almost the entire empire, perished at the hands of criminals who smeared some deadly drugs on tiny needles and for pay infected people with the poison by means of these instruments. The same thing happened before in the reign of Domitian. Now the death of these victims passed unheeded; for Commodus was a greater curse to the Romans than any pestilence or any crime.”¹⁹²

Bizarre accounts of murder aside, Dio’s account is important because it extends the length of the pandemic by nearly a decade after its commonly-cited end (around AD 180).¹⁹³ If we are to trust Cary’s notations in his translation of Dio’s *Roman History*, then the above quote is meant to represent a period in AD 189 or 190.¹⁹⁴ In addition, it is difficult to believe there was a new epidemic affecting the Empire not yet a decade after the Antonine Plague that was wholly different from, and more severe than, the great plague under Marcus Aurelius. The most probable hypothesis here is that the plague was still affecting the Empire throughout Commodus’ reign, possibly subsiding around AD 190, and Cassius Dio’s dramatic language (“the greatest of any of which I have knowledge”) was considering the entire extent of the plague (from AD 165 – AD 190), as well as conveniently complementing his feelings about Commodus.

Like Dio, Herodian wrote his history after the pandemic had ended, and he might not have had first-hand knowledge of the disease or its symptomology, since he was quite young (born in AD 170) even at the end of the pestilence. His account, like Dio’s, is thought to relate events in the late-AD 180s, during Commodus’ reign:

“Just at this time a plague struck Italy, but it was most severe in Rome, which, apart from being normally overcrowded, was still

¹⁹² Cassius Dio, *Roman History*, 73.14.3-4, trans. by E. Cary.

¹⁹³ Duncan-Jones 1996, 116-117; recently the AD 189 end-date for the plague is suggested by Elliot 2016, 8.

¹⁹⁴ Cassius Dio, *Roman History*, 73.14.3-4, trans. by E. Cary.

getting immigrants from all over the world. The result was a tremendous toll of life among men and beasts of burden... On the advice of his doctors, Commodus retired for the time to Laurentum, a cooler spot...the doctors thought this place was safe because it was reputed to be immune from infectious diseases in the atmosphere by virtue of the redolent fragrance of the laurels and the pleasant shade of the trees.”¹⁹⁵

Herodian’s narrative also reveals that the plague was affecting Rome, and other unspecified areas of Italy, well into Commodus’ reign. One cannot be sure if Herodian was borrowing information from Cassius Dio, since they are the only two to report that the pestilence was still surging through, or ferociously reappeared in, Rome in the late-AD 180s. The language in each account seems distinct enough from one another – with Cassius Dio’s report of homicide, and Herodian’s narrative of fragrance-filled Laurentum as a safe-haven from the epidemic – to make a case for the Antonine Plague remaining as a sore on the Empire until the late-AD 180s.

The Historia Augusta and Ammianus Marcellinus

Both the *Historia Augusta* and the works of Ammianus Marcellinus were written centuries after the Antonine pandemic – both composed in the fourth century – but they do reveal how the second-century pestilence was perceived then; however, caution is of course necessary when reading these narratives, especially the *Historia Augusta*.

The *Historia Augusta*, a notoriously controversial work, explains that the Antonine Plague first struck co-emperor Lucius Verus’ army around AD 165 at Seleucia.¹⁹⁶ It was from here, the work claims, that Verus and his army brought the

¹⁹⁵ Herodian, *Roman History*, 1.12.1.

¹⁹⁶ *Historia Augusta*, *Lucius Verus*, 8.1-5. For the work’s date see: Cameron 2011, 772; Gilliam 1961, 232.

disease back to Rome. It could be argued that the account is a complete fabrication, as some excerpts from the *Historia Augusta* have been. However, Ammianus Marcellinus gives a similar account, claiming that, from Seleucia, Verus and the army “polluted” the frontiers of Persia and spread the disease to the Rhine and to Gaul, the latter portion of which the *Historia Augusta* does not mention. Both accounts claim that the pestilence arose when Verus’ soldiers disturbed the temple of Apollo at Babylonia; undoubtedly a fantastical tale. While the imaginative details within the two accounts may render them dubious to some, they do cohere with Calpurnianus’ mention that the Roman army was infected with the disease at the siege of Nisibis (about 700km from Babylonia) in 165; and like the evidence of Aelius Aristides, point to western Asia around 165 as the place and time where the epidemic first struck the Empire.

The Papyrological Evidence

P.Thmouis 1.104

P.Thmouis 1 is a group of reports from the Egyptian village of Thmuis in the Delta that detail Roman administration within the Mendesian Nome.¹⁹⁷ The reports, written by the nome’s *basilikos grammateus* (imperial minister) between AD 170-173, document the debts owed by taxpayers in Mendes, Thmuis, and their surrounding villages in AD 168-169 (Figures 31 and 32).¹⁹⁸ In section 104, the *basilikos grammateus* accounts for the abandonment of the village Kerkenouphis, thought to have been located within the toparchy of Psanitēs or Ptempathiō (Fig. 32).¹⁹⁹

¹⁹⁷ *P. Thmouis* 1, 104; Kambitsis 1985, 98-99; Blouin 2014, 64-65.

¹⁹⁸ Blouin 2014, 64-65; *P. Thmouis* 1, 104.

¹⁹⁹ Blouin 2014, 271.

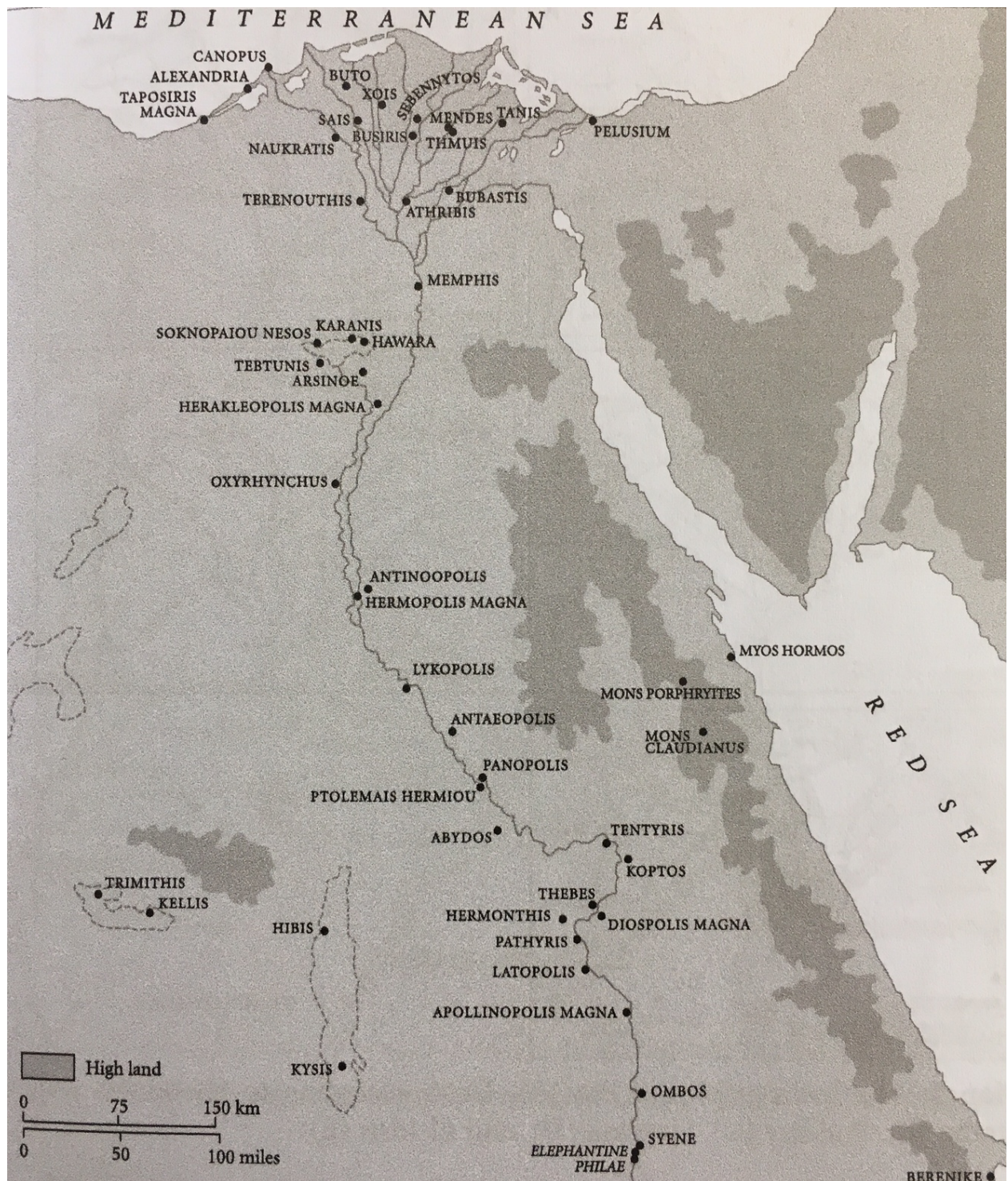


Figure 31. The Nile River of Roman Egypt. The village of Kerkenouphis was located up in the Delta near Mendes. (Blouin 2014)

The *basilikos grammateus* explains that the village was destroyed by what seems to be a rebel nomad tribe: ‘most of the men were killed by the godless Nikochites who attacked and burnt the village. The remainder died from the pestilential situation (τῷ

λοιμικῷ καταστήματι) and only a small number fled.’²⁰⁰ The phrase *to loimikon katastēma* translates to ‘the pestilential situation,’ or ‘the pestilential condition,’ with *loimikon* meaning ‘plague’ or ‘pestilential,’ and *katastēma* referring to a state, situation, or condition related to the body.²⁰¹ Blouin asserts that the phrase

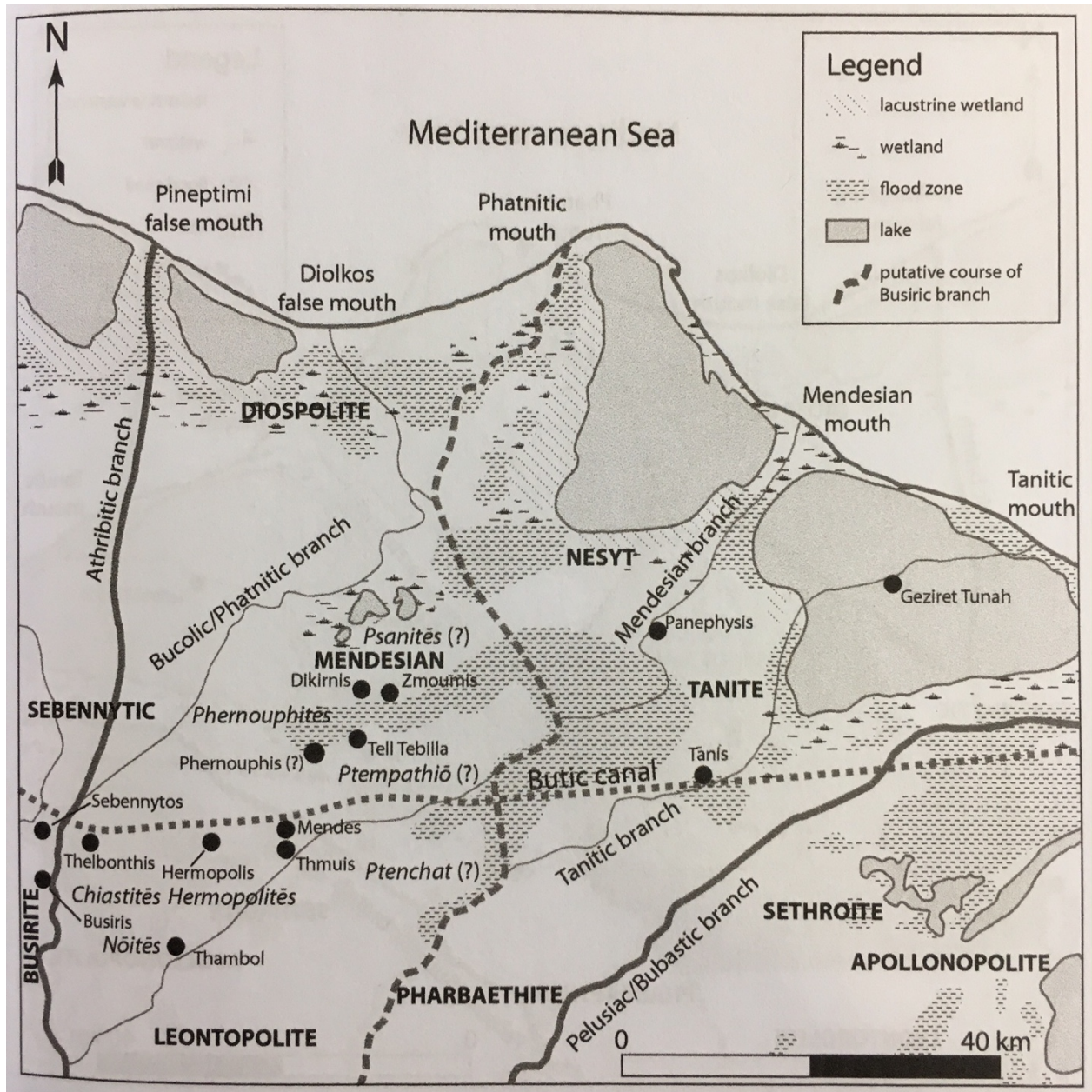


Figure 32. The Mendesian Nome in Roman Times. Note Ptempathiō and Psanitēs; it is between these two sites that the village of Kerkenouphis is thought to have been. (Blouin 2014)

²⁰⁰ P. Thmouis 1, 104.12-18.

²⁰¹ LSJ.

means “literally ‘the pestilential situation’”, and it is hard to disagree.²⁰² The use of *loimikon*, an adjective (in the accusative here) commonly used to refer literally to diseases, suggests that the *basilikos grammateus* was illustrating a rampant state of disease in Kerkenouphis; not just a situation of conflict or economic hardship.²⁰³ And the use of *katastēma*, or “situation,” implies the phenomenon is well-known and not an independent event. The papyrus is thus typically read as denoting an epidemic striking the village of Kerkenouphis.²⁰⁴

This one small section of the *P. Thmouis 1* papyrus is the only direct evidence we have for the plague having reached Roman Egypt. With that said, *to loimikon katastēma* seems to have been an actual and virulent disease affecting Kerkenouphis, enough so that the imperial minister listed it as one of the reasons for the town’s depopulation and decimation in AD 168-169. Thus, we must accept the strong probability that the epidemic wiping out Kerkenouphis, which occurred at the same time the Antonine Plague was affecting other parts of the Empire, was the same disease. This does not definitively prove that most of the Nile Delta, or even most of the Mendesian Nome, was suffering from the pandemic, and there is no evidence for pestilence in the Nile Valley further south, other than Calpurnianus’ questionable remark about the plague originating in Ethiopia. Use of the word “situation” does suggest the disease was known outside of Kerkenouphis. However, the royal minister might have heard that the pestilence was affecting other parts of the Empire. We can assume that at the very least a single path from either the

²⁰² Blouin 2014, 255.

²⁰³ For instances of *loimikos* or *loimos* being used to refer specifically to disease or plague see: Thucydides, II.47 and 54; Polybius, *Histories*, I.19.1; Philo II.102; Lucian *How to Write History*, 15; *Papyrus Oxyrhynchus*, 14.1666.

²⁰⁴ *P. Thmouis 1*, 104; Kambitsis 1985, 98-99; Blouin 2014, 255-6; Elliot 2016, 10.

Mediterranean Sea or the Red Sea to Kerkenouphis was affected by the plague (Fig. 31). It is also safe to assume that the villages nearest to Kerkenouphis (the surrounding areas of either the toparchy of Psanitēs or Ptempathiō) had some of their population infected by the epidemic; if the epidemic helped wipe out one village, it is hard to believe neighbouring villages did not experience the disease (Fig. 32). However, I find it impossible to subscribe to the idea that the Antonine Plague was running rampant through Roman Egypt, since there was much documentation in the province, but no mention of a severe pestilence aside from the mention in *P.Thmouis 1*. What we glean from *P.Thmouis 1* is the pandemic did at least reach and potentially affect portions of the Nile Delta, but there is still no good evidence for disease in the Nile Valley or elsewhere in Roman Egypt at the time of the Antonine Plague. I return to a possible explanation for this below.

The Disease Responsible for the Antonine Plague

This section examines the epidemiology of smallpox in connection with Galen's symptomology, seeking to confirm whether or not we can maintain that the Antonine Plague was the *variola virus*.

Symptoms: confirming the Antonine Plague was probably smallpox

Galen, in his account of the symptomology of the Antonine Plague, mentions that on the ninth day of infection a person's entire body is covered by exanthema (rash and spotting).²⁰⁵ If Galen's 'ninth day' indicates a period from the start of initial

²⁰⁵ Galen, *Method of Medicine*, 5.12 (trans. G.H.R. Horsely and Ian Johnston, Loeb); Littman and Littman 1973, 246.

symptoms, it is probable that the 9 days include the onset prodromal period (2 – 4 days), and the following period in which the vesicles transform to pustules (another 4-5 days); Galen refers to the bumps as *helkos* (ἐλκεσιν: ‘sores’ or ‘ulcers’), denoting an open wound and not just a fluid-filled bump, or vesicle (figures 33 and 34). In addition to his specific account of the rash, he also describes the fever, at one point referring to it as a ‘fever plague’, as well as internal ulcerations, vomiting occurred in some cases, and an upset stomach in all cases.²⁰⁶

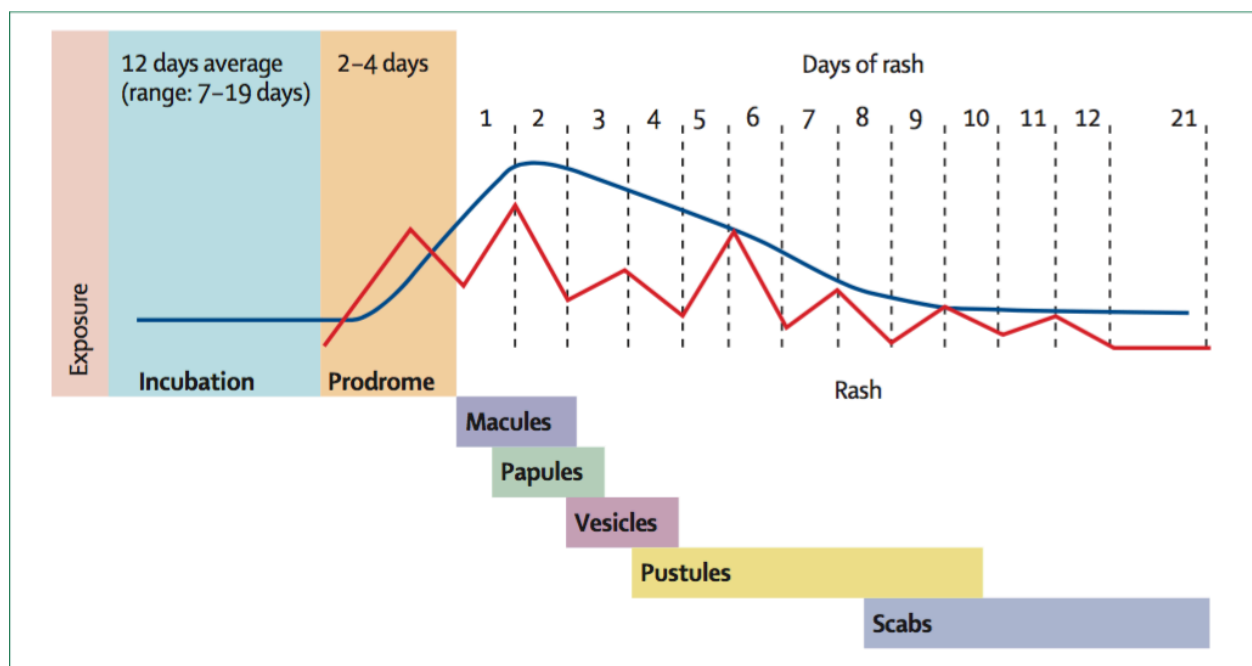


Figure 33. The timeline for a person infected with smallpox. The red line indicates a person's temperature/fever, and the blue lines represents respiratory viral shedding. (Moore, Seward, and Lane 2003)

The symptoms illustrated by Galen have similarities with the symptoms reported in smallpox studies, including fever, vomiting, upset stomach, internal ulcerations, and a post-fever rash of pustules that covered the entire body.²⁰⁷ R.

²⁰⁶ For fever, see Galen, *Hippocratis Libr. 6 Epidemiorum Aph.* 29 and 57 (trans. Littman and Littman 1973, 247); for vomiting and upset stomach, see, *Method of Medicine*, 5.12 (trans. by G.H.R. Horsely and Ian Johnston); for internal ulcerations see *Method of Medicine*, 5.12 (trans. by G.H.R. Horsely and Ian Johnston); Littman and Littman 246-248.

²⁰⁷ Fenner *et al.* 1988, 9–12; Moore, Seward and Lane 2006, 425-435.

Littman, a classics scholar, and his brother M. L Littman, a medical doctor, explain that although there are several exanthematous diseases – one of which (typhus)



Figure 34. An example of the smallpox rash on 'Day 9'. (Fenner et al. 1988)

even has symptoms similar to smallpox – Galen's detailed descriptions of the skin rash combined with the additional symptoms are enough to distinguish it from other

diseases.²⁰⁸ The fact that Galen relates that the Antonine Plague rash spread relatively evenly across the entire body, differentiates the disease from bubonic plague (Chapter 4), in which the infected experience skin abnormalities that are more localised.²⁰⁹ Littman and Littman point out that Galen describes the rash as generally black due to remnants of blood putrefied in the blisters (haemorrhagic), corresponding to the haemorrhagic phase of smallpox, happening when the ‘pustule’ and ‘scab’ stages tend to overlap in the timeline-average (Fig. 33).²¹⁰ While these symptoms do help distinguish smallpox from most diseases that exhibit exanthema, it does not set it apart from typhus rashes.²¹¹ However, Littman and Littman note that a most deciding feature of the rash described by Galen is the pustular lesions (*helkos*), happening on Galen’s ninth day. They explain how the lesions of typhus rash are never pustular, whereas in smallpox they are.²¹² As mentioned above, recent studies on smallpox confirm this pustular stage.²¹³ Thus, the diagnosis made by Littman and Littman in 1973 that the Antonine Plague was probably smallpox rather than typhus is compelling and their conclusions have been accepted by most scholars.²¹⁴

Although most ancient historians over the past half-century see smallpox as the probable cause of the second-century pestilence, Rebecca Flemming reopened the debate in 2019, re-examining the literary evidence with help from a newer

²⁰⁸ Littman and Littman 1973, 248-249.

²⁰⁹ Littman and Littman 1973, 249.

²¹⁰ Moore, Seward, Lane 2006, 426; Littman and Littman 1973, 249.

²¹¹ Ricketts 1910, 109; Littman and Littman 1973, 249.

²¹² Littman and Littman 1973, 249-250.

²¹³ Moore, Seward, Lane 2006, 426-430; Fenner *et al.* 1988, 5-22.

²¹⁴ Harper 2017, 102; Elliot 2016, 5; many contributors to Lo Cascio (ed.) 2012; Zelener 2003; Cunha and Cunha 2008, 12; Sallares 2007, 37; Scheidel 2001, 95; Mattern 2008.

discovery: Galen's *περὶ ἀλυπίας* (*De indolentia*).²¹⁵ For the purposes of this chapter, Flemming makes two important points: (1) that two ancient writers (Aristides and Herodian) remark how livestock were also inflicted by disease at the time of the Antonine Plague, which works against a diagnosis as smallpox since (in our current understanding of the disease as a human-obligate virus) it can only infect humans; and (2) that there is no mention by Galen or other contemporary writers of the facial scarring that we associate with modern smallpox survivors.²¹⁶ On the first point, Flemming cites a phylogenetic study on basal strains of poxviruses conducted by Smithson, Imbrey, and Upton (2017) to show that ancestors of the virus have been around for 3,000 years.²¹⁷ However, Flemming does not make the connection that the study from 2017 suggests that the most recent common ancestor of smallpox emerged after the Antonine Plague.²¹⁸ Monica Green raises the possibility, that if Smithson, Imbrey, and Upton's study is correct, a human-specific and scar-producing poxvirus might have emerged nearly a century after the Antonine Plague, and before that there may have been a "generalist" poxvirus strain floating around infecting multiple species; could this not be the explanation for the infected livestock mentioned by Aristides and Herodian?²¹⁹ It is certainly possible, but as Danielle Gourevitch proposes, the mention of livestock could be for dramatic effect; a literary trope used by previous ancient writers.²²⁰ Flemming goes on to itemise examples of

²¹⁵ Flemming 2019, 219-244. While Vivian Nutton provided the new translation for the recently discovered (2005) *περὶ ἀλυπίας* in Singer 2013, it was Flemming who introduced its larger importance as a source for the Antonine Plague.

²¹⁶ The second point is a detail first raised as troublesome for a smallpox diagnosis by Danielle Gourevitch in 2005: Gourevitch 2005, 65.

²¹⁷ Smithson, Imbrey, Upton 2017.

²¹⁸ Flemming 2019, 234.

²¹⁹ Green 2019: Monica Green's analysis here is from a post on the international listserv message board MEDMED-L, which is devoted to premodern medicine, health, and disease.

²²⁰ Gourevitch 2013, 74-75.

dying animals used for dramatic emphasis, but, ultimately, concludes that the casualness and straightforward manner of Aristides' and Herodian's remarks about disease-ridden livestock indicate their veracity, compared to the metaphorical nature of her examples.²²¹

If we are to accept that livestock were in fact disease-ridden in the later second century AD, how can we be sure that these epizootic outbreaks were caused by the same disease as the Antonine Plague? Neither Aristides nor Herodian describe anything regarding symptoms suffered by the livestock, just that they were sick or dying.²²² That livestock were infected with the same disease as second-century Romans *is* still a possibility, but it is equally possible that cattle were sick with a cattle-specific disease. Can we yet propose with confidence that there was a "generalist" poxvirus making its way through the Roman Empire in the latter half of the second century CE?

A most recent paper from 2020 inches us closer to answering that question. Led by Barbara Mühlemann, aDNA analysis of skeletal remains uncovered 13 individuals from the Middle Ages that tested positive for VARV, 11 of which were from the Viking Age.²²³ Upon analysis of the smallpox genome with the new evidence, Mühlemann and colleagues found that two distinct lineages of smallpox – mVARV (an early strain of modern smallpox) and aVARV (a previously unknown ancient strain) – diverged from an ancient strain of poxvirus between 180-500 CE.²²⁴ This indicates that an ancient strain of poxvirus existed from around 1500-500 BCE

²²¹ Fleming 2019, 234.

²²² Aristides, *Sacred Tales* 48.37-45; Herodian 1.12.1-2.

²²³ Mühlemann *et al.* 2020.

²²⁴ Mühlemann *et al.* 2020, 3-5.

(when the ancient strain diverged from *its* common ancestor) until the divergence of the more recent poxvirus strains mVARV and aVARV.²²⁵ Further, the researchers note that ancestral forms of smallpox may have had a wider host range.²²⁶ Genes that were found to be active in aVARV were inactive in later strains of VARV, demonstrating that aVARV and its common ancestor may have been able to infect multiple mammals, only evolving to a human-obligate virus with the emergence of mVARV. Along with the literary evidence of infected livestock, this new aDNA evidence suggests, as Green speculated in 2019, that an ancient generalist strain of poxvirus may have existed at the time of the Antonine Plague and could be the disease responsible. Additionally, a divergence from the ancient form of poxvirus to the more recent mVARV and aVARV strains hints that an active period for the ancient strain which prompted evolution, such as a pandemic, preceded the divergences. Given the period when the divergence occurred (180-500 CE), the Antonine Plague would fit as that event. Further evidence and research are needed to solidify this hypothesis, but, as of now, the aDNA evidence is consistent with an ancient strain of poxvirus as the disease responsible for the second-century pandemic.

Returning to Gourevitch's and Flemming's point about the absence of smallpox scarring in the ancient sources, it is an important question, but as far as diagnosis goes, it is worth recalling Littman and Littman's assertion that "one cannot argue out of hand that a sign or symptom was not present, simply because Galen does not describe it, unless that symptom would be an integral part of the specific

²²⁵ Mühlemann et al. 2020; Babkin and Babkina 2015; Smithson, Imbrey, Upton 2017.

²²⁶ Mühlemann et al. 2020, 5.

item Galen is describing"; especially since scarring is not an affecting symptom, but an aftereffect that need not be mentioned for an effective history or symptomology of the afflicted – i.e. the principal reasons for mentioning the pestilence.²²⁷

What remains as our strongest evidence for identifying this pandemic is the symptomology related by Galen, and more than anything else it points to a smallpox-like virus as the cause. Until additional, stronger evidence suggesting otherwise surfaces, an ancient strain of smallpox – a closely related ancestor of modern smallpox – is the best hypothesis for the agent responsible for the second-century pandemic.

Basic pathology of smallpox

As shown in Figure 3, the incubation period for those infected with smallpox is typically 10 – 14 days (in rare cases as short as 7 days or as long as 19 days), followed by a prodromal period of 2 – 4 days, and finally a 20-day rash, at the end of which the pustules of the infected turn into scabs.²²⁸ Before the appearance of the rash, the virus causes high fever of approximately 38.9°C – 40.6°C during the prodromal period, as well as vomiting, abdominal pain, headache, and chills.²²⁹ Lesions begin to appear following the prodromal period, usually forming on all parts of the body (including the face, mouth, tongue), which in most cases begin to scab nearly three weeks after their initial appearance. Smallpox deaths were generally caused by

²²⁷ Littman and Littman 1973, 246: It should be noted that Littman and Littman were writing before the discovery of Galen's *De indolentia*, but I am not convinced that their diagnosis would change in light of this new evidence; the brief references do not outweigh Galen's more vital and specific descriptions from *Method of Medicine*.

²²⁸ Moore, Seward, Lane 2006, 426-427.

²²⁹ Fenner *et al.* 1988, 10-11; Moore, Seward, Lane 2006, 426-427.

bacterial infections in the blood which led to organ failure and pneumonia.²³⁰ The modern-day mortality rate of those infected with *variola major* (the more severe strain of the virus) was around 30%.²³¹

As a poxvirus, the *variola virus* is part of the largest family of viruses that infect people and cause disease to human beings.²³² At present, it seems that most poxviruses are restricted to specific animal hosts. However, as I will discuss in the next section, it is possible for some poxviruses to infect an animal other than its typical host. What makes the *variola virus* so dangerous is its ability to evade the immune response of the host, by encoding proteins that manipulate and harm the immune system.²³³ Since smallpox was eradicated in 1980 before the arrival of modern molecular technology, some of our information about its pathogenesis is gleaned from non-variola orthopoxviruses.²³⁴ Data from past research and animal-based models show that the most common route for transmission of smallpox is through the respiratory tract, by the inhalation of infected airborne droplets; however, transfer through the skin is still possible.²³⁵

Some epidemiology: transmitting smallpox

In the monumental study led by Frank Fenner in 1988, *Smallpox and its Eradication*, the authors claim that there are three main routes of viral infection, “corresponding to the three principal surfaces of the body: the respiratory tract, the alimentary tract,

²³⁰ Fenner *et al.* 1988, 47-49; Moore, Seward, Lane 2006, 426-427.

²³¹ Fenner *et al.* 1988, 175-178; Moore, Seward, Lane 2006, 426.

²³² Moore, Seward, Lane 2006, 425.

²³³ Moore, Seward, Lane 2006, 425.

²³⁴ Moore, Seward, Lane 2006, 425-426.

²³⁵ Fenner *et al.* 1988 117-118; Moore, Seward, Lane 2006, 426; Sarkar *et al.* 1973, 517-522; Downie *et al.* 1961, 49-53; Mitra *et al.* 1974, 106-107; Kitamura *et al.* 1977, 229-239.

and the skin.”²³⁶ They maintain that there is no evidence whatever that the *variola virus* can be transmitted through the alimentary tract (via food), nor is there any evidence that arthropods, such as mosquitos or flies, can assist transmitting the virus from human to human.²³⁷ The authors emphasise that the main routes of infection are through the oropharynx or nasopharynx (through the mouth or nose) of the upper respiratory tract, with the lower respiratory tract and skin as less common routes via, and the least common, but still viable route, through the conjunctiva (the mucous membrane that covers the front of the eye and lines the inside of the eyelids).²³⁸

During the 12 – 14 day incubation period there is no evidence of viral shedding – the release of the virus during viral reproduction, both internally to different parts of the body and externally into the environment – meaning the virus is unable to transmit from human to human for nearly two weeks after initial infection.²³⁹ However, things change after the fever begins (Fig. 33). Once the smallpox fever commences, any one person’s infectivity increases, as the virus at this point can break through the mucous membrane of the upper respiratory tract.²⁴⁰ Both studies led by Downie in 1961 and Sarker in 1973 found that smallpox was in most cases able to be transmitted on the third or fourth day following the onset of fever.²⁴¹ In testing oropharyngeal secretions from lesions in the mouths of patients, both studies found that, after the onset of fever, a large portion of patients tested positive for smallpox from day 3 until day 14 (Table 2). Based on the data, the virus

²³⁶ Fenner *et al.* 1988, 182.

²³⁷ Fenner *et al.* 1988, 183.

²³⁸ Fenner *et al.* 1988, 183.

²³⁹ For viral shedding see Dimmock, Easton and Leppard 2016, 31-36; Fenner *et al.* 1988, 183.

²⁴⁰ Upham 1986, 116-117.

²⁴¹ Sarker *et al.* 1973, 517-522; AW Downie *et al.* 1961, 49-53.

was in most cases discharged from the mouth or nose on days 3 through 9 following onset fever.²⁴² Particles carrying the virus would exit the mouth of the infected typically by exhalation, and enter a non-infected person by either inhalation or implanting the virus into the mouth or nasal cavity via hands which had touched contaminated surfaces, bedding, or clothing.²⁴³ Transmitting smallpox through infected sores was still a viable, albeit less likely, route of transmission. Studies published by Mitra and colleagues in 1974 and Kitamura and colleagues in 1977 found that on approximately day 15 of the rash, or day 18 following onset fever, sores would fully scab and subsequently fall off.²⁴⁴ While sores and scabs were highly infectious, epidemiological assessment by Fenner and colleagues in 1988,

Day of disease (from onset of fever)	Oropharyngeal secretions				Urine			
	Downie et al. (1961a)		Sarkar et al. (1973a)		Downie et al. (1965a)		Sarkar et al. (1973a)	
	Number tested	Number positive	Number tested	Number positive	Number tested	Number positive	Number tested	Number positive
1	1	0	-	-	-	-	-	-
2	4	0	2	2	-	-	-	-
3	8	4	10	10	-	-	5	5
4	20	8	22	22	-	-	9	9
5	39	18	20	20	1	0	16	15
6	49	32	22	22	3	1	14	12
7	51	33	22	21	5	3	10	6
8	39	24	18	14	4	2	9	6
9	34	22	18	11	3	2	8	6
10	10	0	13	7	4	2	5	4
11	15	4	10	6	3	2	5	4
12	12	3	8	4	5	2	3	3
13	12	1	9	4	1	1	4	4
14	5	1	5	4	2	1	-	-
15	3	0	2	0	1	0	3	3
16	1	0	1	0	1	1	1	0
17	2	0	-	-	-	-	2	1
18	4	0	-	-	-	-	1	0
19	-	-	-	-	-	-	2	1
20	-	-	-	-	-	-	1	0

Table 2. Testing the oropharyngeal secretions and urine of the smallpox patients. Note the high number of patients testing positive on days 3-9 after onset fever. (Fenner *et al.* 1988)

²⁴² Sarkar *et al.* 1973, 517-522; Downie *et al.* 1961, 49-53; Fenner *et al.* 1988, 183-187.

²⁴³ Fenner *et al.* 1988, 186.

²⁴⁴ Moore, Seward, and Lane 2006, 425-436; Mitra, Sarkar and Mukherjee 1974, 106-107; Kitamura *et al.* 1977, 232-236; Fenner *et al.* 1988, 184-185.

which combined data from four studies, confirmed that transmission of the virus through face-to-face contact during week 1 was more likely than infection via touching contaminated sores or expelled scabs.²⁴⁵ They admit that the reason for the difference in frequency of transmission is difficult to ascertain.²⁴⁶ Fenner and company consider that oropharyngeal secretions could be expelled from an infected person and reach a non-infected person's vicinity (via inhalation, or respiratory droplets being exhaled onto another's clothing or bedding) more easily than infection via contact with sores.²⁴⁷ We must also consider, simply, the likelihood of non-infected people coming in contact with the infected on day 3 or 4 of the fever, when the smallpox sickness is not yet apparent. We should expect that this, along with sharing contaminated space, would have accounted for many new infections in Roman times. That an infected Roman shed the virus onto common surfaces, through exhalation, open sores, or scabs, is highly likely. Given limited understanding of viral transmission 1900 years ago, it is reasonable to assume that the virus transmitted more widely. Due to the lack of modern medical practice and awareness, the mortality rate of those infected during the Antonine pandemic was probably much higher than the modern-day rate of 30% related by Fenner and colleagues.

²⁴⁵ Fenner *et al.* 1988, 185.

²⁴⁶ Fenner *et al.* 1988, 185.

²⁴⁷ Fenner *et al.* 1988, 185.

Origin and Spread

This section will begin by investigating the epidemiological origin of the Antonine Plague which, when coupled with the literary evidence, can help us pinpoint its geographical origin.

Epidemiological and geographical origin

As mentioned, phylogenetic analysis shows that smallpox, camelpox (poxvirus of camels), and taterapox (poxvirus of naked-soled gerbils) are the most closely related poxviruses.²⁴⁸ In 2012, Yan Zelener's chapter in *L'Impatto della Peste Antonina* examined the genetic evidence and epidemiology of the *variola virus*, bringing the similarities between the strains of poxvirus to the attention of historians and archaeologists.²⁴⁹ Citing a study led by McLysaght in 2003, he pointed out that the closest genetic relative to smallpox is camelpox, noting that camelpox and smallpox "are separated by genetic distances of 30 and 31 gene gain/loss from their common ancestor", as compared to a genetic distance of 60 gain/loss observed between smallpox and cowpox, the strain originally thought to be the closest ancestor of smallpox.²⁵⁰ Research conducted by both Afonso in 2002 and Gubser and Smith in the same year confirms this, indicating that both camelpox and smallpox share all genes involved in replicative activity.²⁵¹ McLysaght's study reveals significant characteristics of poxvirus evolution, including, as Zelener mentioned, confirmation that camelpox is the closest relative of the *variola virus*, as well as phylogenetic

²⁴⁸ Babkin and Babkina 2015, 1100-1112; McLysaght, Baldi and Gaut 2003; Zelener 2012, 168-169.

²⁴⁹ Zelener 2012, 168-169.

²⁵⁰ McLysaght *et al.* 2003, 15655-15660; Zelener 2012, 168-169.

²⁵¹ Afonso *et al.* 2002, 783-790; Gubser and Smith 2002, 863; Bera *et al.* 2011, 30.

evidence demonstrating some of these poxviruses may have switched hosts in their history.²⁵²

In his book, *The Fate of Rome*, Kyle Harper argues that the probable geographic origin of the Antonine Plague is Africa.²⁵³ His theory stems from an article published in 2015, in which Babkin and Babkina present phylogenetic evidence that suggests new strains of orthopox virus, such as smallpox and camelpox, might have come about with the introduction of the camel to Africa over 3,000 years ago.²⁵⁴ After analysing the evolutionary history of smallpox, based on the conserved central regions of orthopoxvirus genomes, Babkin and Babkina propose that smallpox, camelpox, and taterapox diverged from the same ancestor approximately 3,400 years ago.²⁵⁵ They claim that the introduction of the camel to Africa 3,500 years ago, which would have been a new potential host with unique antibodies, could have caused the evolution of a cowpox-like ancestral virus and its further separation into three highly specialised species – camelpox, taterapox, and smallpox.²⁵⁶ Babkin and Babkina also suggest, that because central Africa is the only known habitat of the naked-soled Gerbil, it must be the geographic region of the virus' divergence.

On the surface this theory seems plausible, especially since the DNA evidence points to the three poxviruses originating at around the same time. However, there are two problems with this hypothesis. First, TATV has been isolated only once, in 1975, from a naked-soled gerbil, and there has been very little investigation as to whether it is found in, or can infect similar gerbil species.²⁵⁷ For instance, it is

²⁵² McLysaght *et al.* 2003, 15657.

²⁵³ Harper 2017, 91-94.

²⁵⁴ Babkin and Babkina 2015, 1100-1112.

²⁵⁵ Babkin and Babkina 2015, 1103-1108.

²⁵⁶ Babkin and Babkina 2015, 1108.

²⁵⁷ Parker *et al.* 2017, 13; Andrew Wilson, pers. comm.

unknown if taterapox exists or can infect *tatera indica* (Indian gerbil), a species found in various parts of Asia, particularly the Middle East.²⁵⁸ Thus, it is far from clear whether TATV first emerged in naked-soled gerbils.

Second, the evidence they present for the camel's introduction to Africa 3,500 years ago is unconvincing and comes from hypothetical conclusions drawn by Richard Bulliet in his 1975 book, *The Camel and the Wheel*.²⁵⁹ In this publication, Bulliet sees technological parallels between camel saddles in Somalia and the island of Socotra, which he views as the spread of the camel from Arabia to Somalia.²⁶⁰ His presumption is based mainly on Socotran rock drawings, dated to the 10th century BC, which in my opinion is not enough to mark it as the camel's introduction to Africa. E.W. Bovill thinks the Romans were responsible for the introduction of the camel to Africa. He believes the very earliest possible date that the camel was introduced to Africa was by the Persians in the sixth century BC.²⁶¹ However, Bovill cites Caesar's capture of twenty-two camels at Thapsus (Tunisia) in 46 BC, and its documentation and publication, as evidence for the rarity of camels in Africa at that time.²⁶² Could Caesar's reference to camels be his surprise at seeing the animals outside of their usual habitat centuries later than the introduction proposed by Bulliet?

Since the history and geographical origin of TATV, as well as the camel's introduction to Africa, remain unclear, we cannot propose with confidence that the three strains of poxvirus – smallpox, camelpox, and taterapox – diverged in Africa;

²⁵⁸ Harrison 1956, 442; Feeroz, Hasan and Khan 2015, 162; Andrew Wilson, pers. comm.

²⁵⁹ Bulliet 1990, 7-28.

²⁶⁰ Bulliet 1990, 38-56.

²⁶¹ Bovill 1956, 19-21.

²⁶² *De Bello Africo*, LXVIII.4; Bovill 1956, 20.

but it cannot be ruled out as a possibility. It is also plausible that the viruses emerged in Asia, given that Asia is the habitat of similar gerbil species of the same genus (*tatera indica*), as well as a main habitat of camels.²⁶³ Or it may be that interaction amongst the species connected to these orthopoxviruses was not the cause of the divergence. It is also possible that significant changes in climate, as mentioned by Babkin and Babkina to historically be a cause of viral divergences, was the culprit.²⁶⁴ The point is that there is not enough evidence at this stage to claim a specific origin, either epidemiologically or geographically. However, what we do know for certain is that an ancient version of the smallpox virus existed around 1000 BC – AD 300, and it was most closely related to poxviruses that infect camels and gerbils, so it probably originated somewhere in the broad region of Asia and Africa, the only place geographically that these animals exist together. Nevertheless, it matters less where the virus originated centuries before the Antonine pandemic, but more where its reservoirs were in the second century AD before the start of the pestilence. And the written evidence points to central or western Asia as the reservoir region from which the virus transferred to the Roman Empire.

Pestilence in central Asia?

Additional support for the geographic origin being central Asia or the Middle East is evidence of pestilence in Han China. In his 1996 article, Duncan-Jones shows evidence of epidemics from the Chinese records.²⁶⁵ Citing epidemics recorded in Chinese records, originally used by McNeill in 1989, Duncan-Jones explains that

²⁶³ R. T. Wilson 1998.

²⁶⁴ Babkin and Babkina 2015, 1107.

²⁶⁵ Duncan-Jones 1996, 117.

there is evidence of two epidemics in the AD 160s, and evidence of three in the AD 170s.²⁶⁶ The pestilence of AD 173 in Han-ruled China was mentioned by the Song dynasty historian Sima Guang (AD 1019-1086).²⁶⁷ While Guang is nearly nine centuries removed from the pestilence he speaks of, and we should not rely on this account alone for the origin of the Antonine Plague, it cannot be denied that his reference to a pestilence in AD 173 correlates with the period of the Antonine Plague, which at this point was having a serious effect on the Mediterranean region. While it seems the two bouts of pestilence do conveniently appear at the same time, there is no evidence besides timing that suggests the pestilence in China was the same disease. But it is certainly possible that the Han China pestilence was the same disease, and if we consider that the disease mentioned by Guang was a smallpox-like pestilence, along with the literary evidence for epidemics in western Asia, central Asia seems a most likely place for the pandemic to have originated geographically, as it is situated between both the Roman Empire and Han China.

Rossignol and Durost discuss, on the basis of Han dynasty court documents, how two Roman ambassadors arrived in the royal courts of Han China (AD 166) at around the time of the Antonine Plague.²⁶⁸ They suggest that the Roman ambassadors might have been exposed to a pestilence while in China, bringing the disease back to infect the people of the Mediterranean region.²⁶⁹ This is highly unlikely. First, since there is evidence of outbreaks in Asia Minor one year earlier (the accounts of Calpurnianus and Aristides in AD 165), it nullifies the notion

²⁶⁶ Duncan-Jones 1996, 117; McNeill 1989, appendix.

²⁶⁷ de Crespigny and Suma 1989, (trans. by) 54.1757 A (Y4) and n.02; Rossignol and Durost 2007, 418.

²⁶⁸ Rossignol and Durost 2007, 420; Raschke 1978, 645 and 850.

²⁶⁹ Rossignol and Durost 2007, 420.

straightaway. The theory is additionally ineffective because the chances that this lone encounter involving only a few individuals, which happens to be on record, was responsible for bringing the Antonine Plague to the Roman world, are low in comparison to the idea that multiple groups of people were forced to leave their ethnic region, due to war or economic turmoil, and inadvertently exposed the virus to virgin populations. Moreover, if the ambassadors were infected, they would have probably died from the disease well before reaching Rome, and, if the disease was an ancient strain of poxvirus, they almost certainly were not travelling with a group large enough to allow the virus to persist over that great a distance.

It is more probable that groups from central Asia migrated due to the climatic disturbances mentioned in Chapter 1. The drop in temperatures in central Asia probably caused extensive crop-failure, and, in hopes of seeking better opportunity in a neighbouring region, people probably migrated to regions that were unexposed to the virus. In considering this perspective, the notion that two Roman ambassadors began the Antonine Plague is less likely.

In the same category of plague-transmission through mass-migration, it is quite possible that the Roman army brought the plague back to the Empire from Persia. This idea is supported by the outbreak at Nisibis mentioned by Calpurnianus (information which probably came from Roman soldiers since he was chronicling the Roman-Parthian war), the excerpt in the *Historia Augusta* telling how Lucius Verus and his army brought plague back to the Empire, and Galen's mention of a legion infected with plague at Aquileia.²⁷⁰ Since an infectious disease like smallpox is

²⁷⁰ Lucian, *Quomodo historia conscribenda sit*, 15; Galen, *Methodus menendi*, 5.12; *Historia Augusta*, *Lucius Verus*, VIII.7.

reliant upon large concentrations of people for its spread (e.g. urban centres; armies on the march: a Roman legion was typically 3,000-6,000 soldiers), the following seem the two most likely scenarios for the *variola virus* reaching the Empire: (1) climate-driven mass migration of tribes from central Asia caused infected nomads to come in contact with Parthians, who then, during the war, passed the virus on to the Roman army; and the virus then persisted within the legions (much like Galen experienced with the Roman legion stationed in Aquileia) until they returned to Mediterranean cities from which the rest of the Empire was infected; or (2), a moving settlement of central Asian nomads transmitted the virus directly to a Roman legion somewhere in Parthia.²⁷¹

Smallpox and Climate: the sensitivity of the variola virus to temperature and other climatic factors

Research both before and after its eradication in 1979 shows that smallpox is sensitive to temperature, relative humidity, and in some cases precipitation. Field studies conducted by Leonard Rodgers in 1926 and 1928 on smallpox incidence in India and the U.K. respectively, found that smallpox cases increased in the winter.²⁷² Despite the U.K. and India having different climates, Rodgers discovered smallpox incidence rose when it was colder in these regions compared to the rest of the year, affirming that “a cold winter and spring are likely to result in increased smallpox”.²⁷³ With Rodgers’ studies in mind, in 1929 Russell and Sundararajan calculated coefficients of correlation between smallpox mortality and four climatic factors —

²⁷¹ For the requirement for a large, dense population to enable the spread of smallpox, see Eichner 2003, 118-128.

²⁷² Rogers 1926, 42; Rogers 1928, 300-302.

²⁷³ Rogers 1928, 302.

rainfall, relative humidity, temperature, and pressure – finding that lower rainfall, lower relative humidity, and lower temperature are inversely correlated with smallpox, and facilitate its spread.²⁷⁴

Smallpox is a disease transmitted between people (not via insect vectors) typically by the transfer of respiratory droplets (e.g. coughing, sneezing, dripping, exhaling) either directly or through contact with a contaminated common space.²⁷⁵ Because the virus can survive and remain infective for a period of time outside of the body, lab studies were conducted to test the virus' viability under certain conditions. In 1947, Downie and Dumbell performed a study that tested the survival of *variola virus* in infected crusts (scabs) at room temperature in the sunlight and in darkness. Their results showed that smallpox survived fifty days longer at room temperature in darkness than at room temperature in sunlight.²⁷⁶ Although they made no tests higher or lower than room temperature, their results led them to conclude, "it seems a priori highly probable that the period of survival at lower temperature would be longer than at room temperature." MacCallum and McDonald conducted similar analysis in 1957, but for the virus' survivability in raw cotton at a specific temperature and relative humidity.²⁷⁷ They found the virus to survive far longer at lower relative humidity. Then in 1976, Farida Huq, like Downie and Dumbell, performed lab tests on crusts, but under different temperatures and relative humidity. Overall, she observed that the virus' survival was "considerably prolonged" at lower temperatures and relative humidity.²⁷⁸

²⁷⁴ Russell and Sundararajan 1929, 590-94

²⁷⁵ Fenner *et al.* 1988.

²⁷⁶ Downie and Dumbell 1947, 552.

²⁷⁷ MacCallum and McDonald 1957, 247.

²⁷⁸ Huq 1976, 710.

The monumental study led in 1988 by Frank Fenner, “Smallpox and its eradication”, which both investigated the virus in a laboratory setting and synthesized a wealth of information on smallpox, also noted the negative relationship between smallpox incidence and both temperature and relative humidity. The study indicated that there is strong correlation of higher incidence with low temperature and humidity, citing studies from the former Soviet Union, Brazil, western and central Africa, India, and Bangladesh.²⁷⁹ The authors assert that “the effect of environmental conditions on the viability of variola virus was probably an important factor in determining the seasonal incidence of smallpox.” In a more recent review of the historical data, Nishiura and Kashiwagi’s 2009 paper, “Smallpox and Season,” which reanalyses seven historical datasets, clearly shows annual peaks in smallpox incidence every January for regions in the Northern Hemisphere (winter).²⁸⁰ Additionally, while they did not find a notable association with rainfall, they did find a negative association with relative humidity, concluding that smallpox is dependent on season and “would most likely be enhanced by dry weather.” They also note that the virus immediately loses infectivity above 30°C and 55% relative humidity.²⁸¹

Based on the scientific evidence and research data, there is a clear association between smallpox incidence and low temperature and humidity. The data show that smallpox is more likely to survive longer and spread better when the climate is cooler and drier. If the disease responsible for the Antonine Plague was smallpox, or an ancient ancestor of smallpox which shared smallpox’s sensitivity to cool and dry

²⁷⁹ Fenner *et al.* 1988, 180.

²⁸⁰ Nishiura and Kashiwagi 2009, 1-10.

²⁸¹ Nishiura and Kashiwagi 2009, 5.

conditions, then climatic conditions in the second century might have had an important effect on the spread of the disease.

Discussion

In reconsidering the first reported cases of the Antonine Plague, all, aside from Aristides' account, make direct mention of the Roman army.²⁸² The army was involved in a bitter war with Parthia from AD 162 – 166 in the region that included Armenia and Upper Mesopotamia.²⁸³ At the time, the Parthian Empire controlled territory from western central Asia in the east to Mesopotamia in the west. If we recall the drop in temperatures in this region in the 160s, recorded in the tree-ring data from the Russian Altai Mountains (Chapter 1, Fig. 8), most likely caused by the eruption in Far Eastern Russia AD 163, as well as the notable rise in aridity, as evidenced by the paleoclimatic data from western Asia, it is plausible that there was a rise in incidence of smallpox (or a closely related ancestor) in central Asia near the Roman-Parthian war, similar to the rising smallpox incidence in colder conditions documented in the studies mentioned above. A susceptible Roman army probably contracted the virus near a reservoir region during the period of heightened incidence, afterwards carrying the disease back to an equally susceptible Empire. Climatic conditions were further worsened after the eruption around AD 169/170, as temperatures and moisture were lowered further in parts of Europe and Asia and remained low until the late-180s (in some cases later). The second eruption and subsequent cooling may have helped the virus spread better in the 170s, but it seems

²⁸² Aristides' account is generally thought to be associated with Verus' army: Gilliam 1961, 230.

²⁸³ Birley 2000, 160-5.

that the main actors instigating the Antonine Plague were the AD 163 volcanic eruption and the Roman army. Collaborative multidisciplinary research will be necessary to flesh out this hypothesis, but it is most likely that climate change contributed to the Antonine Plague.

Impact: Evidence for the Antonine Pandemic?

Most scholars today believe the Antonine Plague probably had a significant impact on the Empire both demographically and economically.²⁸⁴ However, as Elliot has pointed out, it is mostly assumed that evidence of decline (at least in Egypt) between AD 165 – 180 is from the plague.²⁸⁵ This section will start by looking at examples of decline from the northern half of the Empire and discern whether the evidence is indicative of pandemic disease, climate, or something else. Lastly, we will return to the case of Roman Egypt to assess what evidence of downturn can actually be attributed to the Antonine Plague with the little direct evidence we have.

The Northern Empire

Decline in silver mining

In 2018, Joe McConnell led a study that measured ancient European lead pollution in Greenland ice. Lead pollution is a proxy for lead smelting activity associated with lead mining, and by extension with silver mining since much lead occurs in lead/silver ores. The team's research indicated that lead/silver mining across Europe decreased sharply and considerably around AD 166, just after the start of the

²⁸⁴ For those subscribing to a 'severe' impact: Harper 2017; Duncan-Jones 1996; Scheidel 2002; Sallares 2007; Jongman 2007; Rathbone 1997; Giardina 2007; and most of the contributors to Lo Cascio (ed.) 2012.

²⁸⁵ Elliot 2016; Elliot 2020.

Antonine Plague (Fig. 35).²⁸⁶ While this clear reduction in mining undoubtedly represents economic downturn at the time of the plague, more precisely it reveals substantial population loss.

Since the drop in mining at the start of the plague is so dramatic, it probably signifies demographic decline and disruption at mining sites, rather than loss induced by, for example, climate change. If the changing climate we observed for the mid-160s had affected agriculture, its effect on the economy would be more gradual. However, given the sudden nature of the decline, it almost certainly indicates population loss. The Marcommanic Wars (AD 166 – 180) could be partly responsible for this loss – from either abandonment or bloodshed – but that does not explain mining stoppages for the entirety of Gaul, or stoppages in Spain and Britain. The Antonine Plague seems to be the most likely culprit.

We still need to be cautious in applying these data to confirm the impact of the Antonine Plague given it is still proxy evidence. But, as of now, it is the best proxy evidence we have, and it suggests that in Europe the second-century pandemic was substantial in intensity and scope.

²⁸⁶ McConnell *et al.* 2018, 5726-5731.

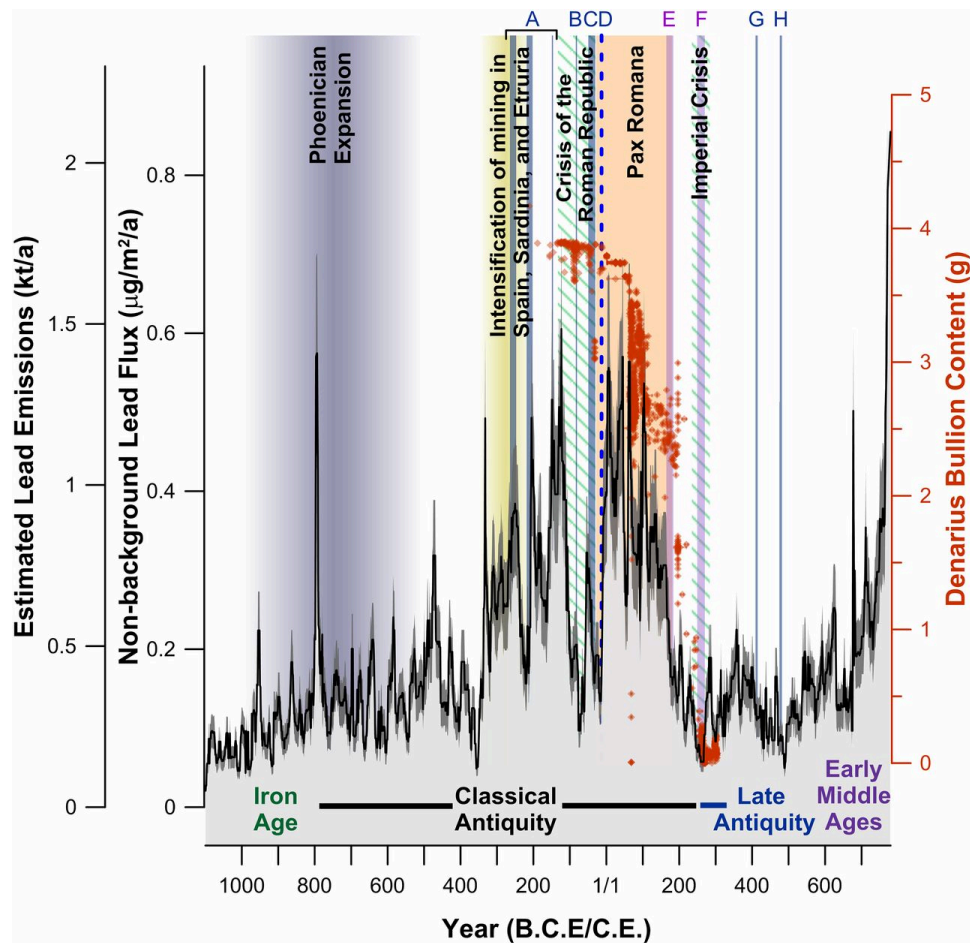


Figure 35. Measurements of lead deposition in Greenland ice, showing European lead emissions and denarius coinage (silver) bullion content. “E” in purple shading represents **the Antonine Plague**. Note the sudden reduction in silver mining at the start of the plague. (McConnell *et al.* 2018, Fig. 3)

Alburnus Maior / Roşia Montană

In the northern Empire, discoveries at the Roman gold mining site of Alburnus Maior (most likely beneath the modern village of Roşia Montană in western Romania) in the 18th and 19th centuries uncovered evidence that might bear on the Antonine Plague. Thirty-three legible writing tablets were discovered in at least five different mining galleries, dating from the AD 130s – 160s. The tablets document various activities, from loan contracts, to labour contracts, to the sale of slaves.²⁸⁷

²⁸⁷ Wilson, Mattingly, and Dawson 2010.

One tablet is of particular interest and might be indicative of an epidemic. A tablet dated to February 9th, AD 167, explains how the funerary collegium of Alburnus Maior had recently been dissolved.²⁸⁸ The funerary collegium, or burial club – which typically organises funerals, and/or burials for recently deceased members – seems to have dissolved for a few reasons: (1) it had just 17 of its original 54 members left in 167; (2) its co-magistrate (co-president of the club) had fled the community; and (3) most of the collegium's members were no longer attending meetings or paying their dues.²⁸⁹

This is another clear case of demographic dislocation, rather than decline caused by climate or environmental change. It is possible that the mining community of Alburnus Maior was feeling the effects of the Marcomannic War, which was at the time just beginning. The abandonment in the late 160s of several different mining galleries that were seemingly in full swing does suggest war or invasion, and the writing tablets cease after May 29th, 167. However, the tablet explains that the remaining magistrate and the treasurers had returned money to present members. This suggests the association did not have sufficient members, and thus the funds to run the collegium. The tablets do not indicate the fate of absent members, but it seems residents were gradually fleeing Alburnus Maior. This case of flight might demonstrate community members avoiding the Antonine Plague, and not war, as two other tablets were found dating from 167, both later than the funerary collegium tablet. These tablets record a *societas* agreement dated March 28th,

²⁸⁸ *Tabulae Ceratae Daciae* I = CIL III, pp. 924–7, I.

²⁸⁹ Duncan-Jones 1996, 121; Wilson, Mattingly, and Dawson 2010; Liu 2017, 208-209; Duncan-Jones 2018, 43.

and a loan repayment dated May 29th.²⁹⁰ They demonstrate that war in the region had not yet severed business at the mines, even after the dissolution of the burial club. We can assume the war did finally reach the mines after May 29th, 167, the date of the latest tablet, and that it was during these invasions that several concession owners hid their documents in the mines. But, like Duncan-Jones and Wilson, I am inclined to attribute the earlier dissolution of the funerary collegium to the Antonine Plague.

The Catacombs of Marcellinus and Peter (Rome)

Multi-burial tombs containing more than 1,300 individuals were found at the catacombs of Marcellinus and Peter on the Via Labicana south-east of Rome in the early 2000s; however, only rooms/tombs X80/T16, X82/T18, and X78/T15 have been fully excavated, while just the upper respective layers of rooms X83 and X84 were excavated, and rooms X80 and X81 were not excavated at all aside from two small soundings in X81 (Fig. 38).²⁹¹ The investigating archaeologists propose that an epidemic was the cause of death for at least the burials in T16 and T18, mainly based on the number of multiple burials, the disposition of the bodies, and the lack of traumatic lesions on the remains. They date the burials to between the end of the first century AD and the first half of the third century AD, based on material finds (three items of jewellery), numismatic evidence (four coins), and radiocarbon dates, with which they hypothesise a connection to the Antonine Plague for the fully

²⁹⁰ Wilson pers. comm; *Tabulae Ceratae Daciae* XIV = CIL III, p. 950–1, XIII; *Tabulae Ceratae Daciae* XIII = CIL III, p. 949, XII.

²⁹¹ Blanchard *et al.* 2007, 989-998.

excavated rooms.²⁹² The burials in T16 and T18 were in most cases well managed, aligned head-to-toe to maximise the space of each tomb or room (Fig. 36), except for certain layers exhibiting erratic organisation that indicate groups of bodies were thrown into the tomb from above (Fig. 37) – conditions typical of an epidemic mass grave.²⁹³ In addition, the archaeologists note that the tombs were initially intended as a funeral space (at least for T16 and T18: Fig. 38); the tombs seem well planned and most layers showed traces of plaster and textiles from corpse preservation (remnants indicating burial methods that were most likely similar to mummification), demonstrating that the deceased were handled with care and probably in accordance with community funeral practices.²⁹⁴ However, Blanchard and colleagues think that because the burial spaces show evidence both of meticulously management and of haphazard arrangement of bodies that the haphazard burials reflect a breakdown in normal practice, demonstrating a community experiencing a pandemic.

Using Tomb 16 as an example, they propose that the meticulous management discovered for levels 1 – 3, followed by the disorganisation of levels 4 and 5, and ending with a return to careful arrangement in levels 6 – 9, might indicate the phases of an epidemic – the typical gradual start, an eventual peak in deaths, and finally the waning of cases – explaining that the disorderly nature of levels 4 and 5 could be the chaotic peak of a pestilence. However, they admit that establishing a chronology for

²⁹² Blanchard *et al.* 2007, 996; Blanchard *et al.* 2015, 204.

²⁹³ Blanchard *et al.* 2015, 203-204.

²⁹⁴ Blanchard *et al.* 2015, 203: they discuss similar funeral practices in the East and North Africa, specifically Tunisia, hypothesising that the community using the catacomb probably derived their burial methods from the latter.

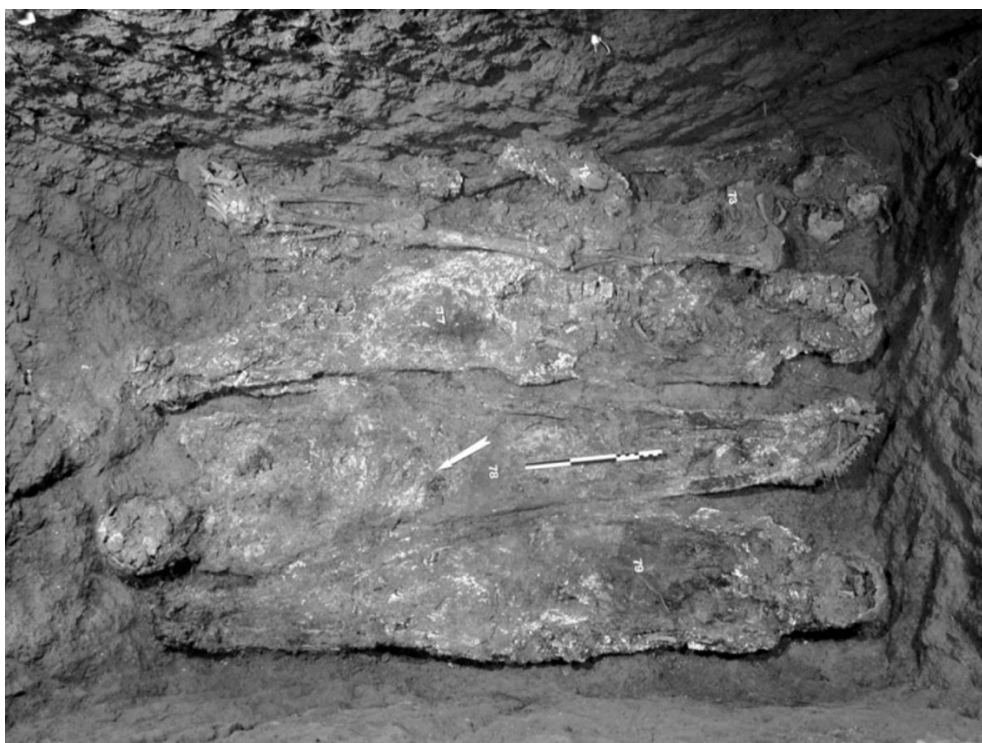


Figure 36. Tightly aligned burials in Tomb 16 of the Catacombs of Marcellinus and Peter.
(Blanchard *et al.* 2007)

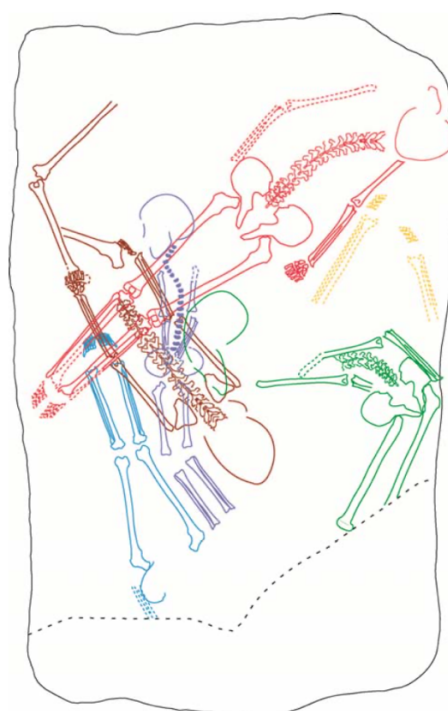


Figure 37. Levels 4 and 5 of T16. Note the haphazard distribution of the bodies compared to figure 3.
(Blanchard *et al.* 2015)

the levels is difficult, as, in many cases, thin layers of sediment separate remains, and it is evident that decomposition needed to have taken place to fit the 76 and 78 bodies in tombs 16 and 18 respectively.²⁹⁵ Thus, the team proposes a mixed-burial conclusion: several multi-burial deposits and some individual burials (probably from both natural causes and disease) occurring for longer than a single event; over decades, or centuries, and possibly through multiple epidemic episodes.²⁹⁶

For the partially-excavated rooms (X83 and X84), Blanchard admits it is difficult to hypothesise, since only 0.20 m of the 1 m thick deposit has been excavated. Based on the limited excavation, he noted that the management of bodies in T16 and T18 was better than in X83 and X84 – probably due to limited space in the former – but all of the bodies examined appeared to have been shown care and attention, and that, unlike T16 and T18, no bodies were hastily deposited. Also, unlike T16 and T18, there was no sediment between the upper phases of deposition in X83 and X84.²⁹⁷ The absence of sediment between phases might be indicative of one mass deposit from a single event over a short period of time, such as the first wave of a devastating epidemic. However, because the bodies were meticulously managed in these spacious rooms (uncharacteristic of epidemic mortality), and with so much still to excavate, Blanchard was hesitant to speculate that an epidemic was the cause.²⁹⁸ For the two unexcavated rooms, X80 was left completely untouched, and only two small soundings were done by the Vatican in room X81 in their preparation for the installation of new arches to stabilise the catacomb.²⁹⁹ These

²⁹⁵ Blanchard *et al.* 2015, 201: the team had modelling work conducted by Géraldine Sachau (Sachau 2012) for tombs 16 and 18, exhibiting “l’impossibilité de déposer autant de cadavres dans des espaces aussi restreints.”

²⁹⁶ Blanchard *et al.* 2015, 204-205.

²⁹⁷ Philippe Blanchard, personal communication; Blanchard *et al.* 2015, 200.

²⁹⁸ Philippe Blanchard, personal communication.

²⁹⁹ Blanchard *et al.* 2015, 201-202.

soundings uncovered coins of Faustina (141 – 161) and Gordian (238). Aside from the coins for X81 and that the rooms contain burials, nothing else is known about X80 and X81.

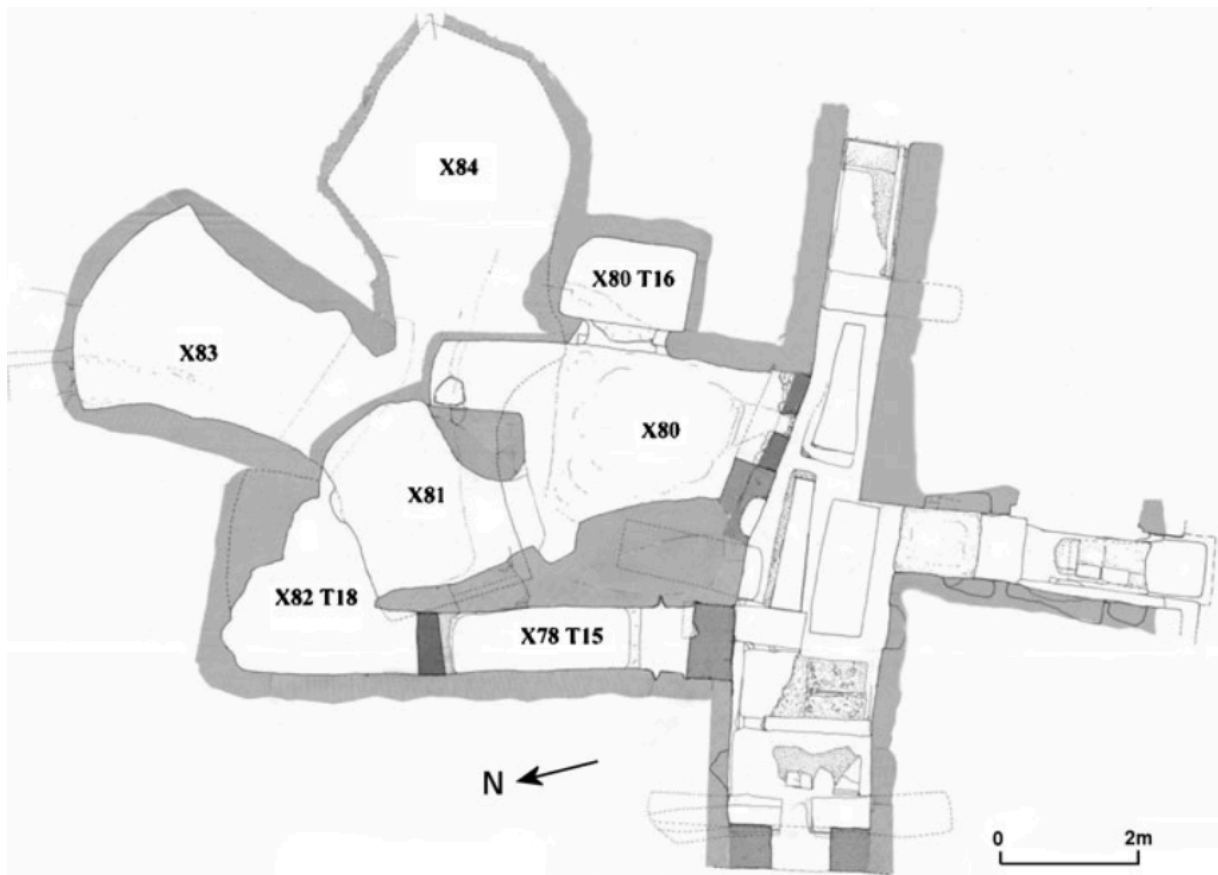


Figure 38. The central portion of the Catacombs of Marcellinus and Peter.

Establishing a chronology for the central portion of the Catacombs of Marcellinus and Peter (Fig. 38) is complicated; as is determining the cause of mortality for each room and burial. The fully excavated rooms (T15, T16, and T18) are the most definitive – the material evidence (coins of Titus [AD 79 – 91] and Marcus Aurelius [AD 161 – 180]; and jewellery: Fig. 39) indicates a *terminus post quem* between the late first century AD to the end of the second century, and the disposition of the bodies and the skeletal characteristics (the lack of traumatic

lesions) suggest epidemic mortality; making clear why the archaeologists hypothesise a connection with the Antonine Plague. However, the other deposits are less obvious. We know next to nothing about the deposits in rooms X80 and X81, aside from the coins uncovered from the soundings in X81; the coin of Gordian (AD 238) suggests a mid-third-century date for that deposit, too late for the Antonine Plague. For rooms X83 and X84, as mentioned, only the top layers were excavated, revealing the careful management of bodies, but no sediment between deposits. Radiocarbon dating of human bone from room X83 returned a chronological range of cal.AD 140 – 380, pointing to the mid third century.³⁰⁰ In X84, a bone hairpin and a black ring (jet stone from modern-day Whitby in northern England: Fig. 39) were found in contact with skeletal remains; both items probably date to the third century.³⁰¹ Additionally, the archaeologists noted that tombs T15, T16, and T18 preceded rooms X80, X81, X83, and X84 in use. This all suggests that



Figure 39. Gold earrings (1st-3rd century AD) and a black ring (3rd century AD) found in Tomb 18 and room 84, respectively, in the Catacombs of Marcellinus and Peter. (Blanchard *et al.* 2015, 213)

³⁰⁰ Calibrated at 2 sigma (95.4% confidence).

³⁰¹ Blanchard *et al.* 2015, 201.

the burials in T15, T16, and T18 may be from the Antonine Plague (AD 165 – 190), and that those in X80, X81, X83, and X84 may be from the Plague of Cyprian. Alternatively, it is possible that all burials are from the Plague of Cyprian, given the *terminus post quem* for each area.

Harper thinks these burials are connected to the Plague of Cyprian, based on the coin of Gordian and one of the unidentified coins (found during surface cleaning of X80) that archaeologists proposed in 2011 might be of Alexander Severus or Gordian; but they have since retracted this claim.³⁰² Harper's hypothesis is not implausible, but with so little known about the burials in rooms X80, X81, X83, and X84, it is hasty to connect them to the Plague of Cyprian without more excavation. If the earlier rooms (tombs T15, T16, and T18) hold victims of a pandemic, the minimal material evidence from the second century AD points to the Antonine Plague as the event. But here too we currently lack the evidence that definitively connects it to a specific pestilence.

Mass grave at London Road, Gloucester, U.K.

Further northwest, in Britain, excavations at a Roman cemetery in Gloucester uncovered a mass grave containing at least 91 burials (Fig. 40).³⁰³ Most of the skeletons in the pit were entangled, suggesting the bodies were dumped in a hurried manner. Based on the pottery found in the grave, the archaeologists Simmonds, Márquez-Grant, and Loe have dated the mass burial to the second half of the second century AD. While many of the skeletons are young and male, the grave also

³⁰² Harper 2015, 226; Castex *et al.* 2011; Blanchard *et al.* 2015, 201-203.

³⁰³ Simmonds, Márquez-Grant and Loe 2008, 34-41.

contains a number of females, as well as adolescent and elderly skeletons. The archaeologists note that the skeletons show no signs of trauma, demonstrating that these individuals were not casualties of war or other violence.³⁰⁴

If not war, what about climate change? Climatic events like those detected for the second century CE could have affected crops and strained food stores in Britain. Simmonds, Márquez-Grant, and Loe note that famine typically affects the oldest and



Figure 40. Haphazard burials at London Road cemetery, Gloucester, UK. Dated to the second half of the second century AD. (Simmonds, Márquez-Grant, and Loe 2011)

the youngest ends of a population, which was not reflected in the mass grave at London Road.³⁰⁵ Thus, famine via climate change does not seem to have been responsible for the deaths in Gloucester.

The most likely cause was pandemic disease. While no scientific analysis has yet revealed the cause, Simmonds, Márquez-Grant, and Loe propose: “An epidemic, such as plague, that did not target either sex or any particular age group, therefore provides a powerful explanation for the deaths among this random cross section of the population.” In addition, the dating corresponds to the mining stoppages across

³⁰⁴ For examples of mass burials with skeletons that do exhibit trauma from battle see: Fiorato, Boylston and Knüsel 2000; Signoli *et al.* 2004; Kooijmans 2005.

³⁰⁵ Simmonds, Márquez-Grant, and Loe 2011.

Europe (including Britain) in the wake of the pestilence.³⁰⁶ We await scientific confirmation for what caused these deaths, but in considering the mass burial's contents, arrangement, dating, and location, it is hard to refute this as a possible case of the Antonine Plague.

The Southern Empire: Roman Egypt

There is no good evidence from ancient writers that explicitly says the Antonine Plague ravaged Egypt. In addition, there is a glaring dearth of papyrological evidence from Roman Egypt, which, compared to other provinces throughout the Empire, documented a fair amount, both public and privately – such as, amongst other things, registers, census data, tax-assessments, court records, accounts, sales, and edicts. In all of this documentation, there is just one known piece of papyrological evidence which mentions a disease in Egypt at the time of the Antonine pandemic.³⁰⁷ The idea proposed by Duncan-Jones that the Antonine Plague is not mentioned much by ancient writers or elsewhere since plague outbreaks were a “matter of course”, or, because “epidemic disease was over-familiar”, is unconvincing.³⁰⁸ In the most immediate sense, it is surprising that multiple economic and demographic models have been created based on the pandemic's presumed effect in Egypt, when there is minimal direct evidence that it reached the Roman province.³⁰⁹ It is possible that the Antonine Plague did not reach some of the most populated parts of Egypt. While papyrological data suggest the

³⁰⁶ McConnell *et al.* 2018.

³⁰⁷ Elliot 2016, 10-11; *P. Thmouis* [*Papyrus Thmouis* 1], 104; *P Oxy.* [*Papyrus Oxyrhynchus*], 14.1666; Lucian, *How to Write History*, 15.

³⁰⁸ Duncan-Jones 1996, 110.

³⁰⁹ Scheidel in Lo Cascio 2012, 265-295; Scheidel 2002, 97-114; Bagnall 2002, 114-120; Duncan-Jones 1996, 1080-136.

pestilence did affect the area of the Nile Delta, there is no other direct evidence that indicates it reached regions outside the Delta.³¹⁰ Thus, caution is needed: we should question economic and demographic models that attempt to assess the impact of the Antonine Plague by utilising indirect evidence, with no foundational evidence to support the claim.

I am not arguing that the Antonine Plague did not reach nor affect Roman Egypt at all, but, instead, challenging the immediate acceptance that it had a major impact on Egyptian society in the latter half of the second century AD. Why have scholars readily accepted with little evidence the idea that the pandemic was the main cause of economic downturn and population loss in second-century AD Egypt?

Collin Elliot's 2016 contribution to the study of Roman Egypt calls for a major re-think of our assessment of the Antonine Plague in the province.³¹¹ He points out how in assessing the epidemic's impact in Egypt, many scholars are reliant on indirect evidence for their narratives and models.³¹² He calls into question the use of evidence based on land rents, wages, and pricing, citing Katherine Blouin's 2014 work on revolts and land abandonment in the Nile Delta under Roman rule.³¹³ Most importantly for the scope of this chapter, Elliot works to connect mid-second-century-AD volcanic eruptions with data for failed Nile floods in the 160s and 170s.³¹⁴ His argument is convincing, although the data for volcanic activity that he

³¹⁰ *P. Thmouis* 1, 104, 9-105, 22; Blouin 2014, 255.

³¹¹ Elliot 2016: Again, Elliot's paper is essentially a fleshing-out of Andrew Wilson's "revisionist questions" about plague in Egypt: Wilson 2013, 265.

³¹² Among others, Elliot cites: Duncan-Jones 1996; Bagnall 2000 and 2002; van Minnen 2001; Scheidel 2002.

³¹³ Elliot 2016, 4; Blouin 2014.

³¹⁴ Nile flooding, which is the result of the annual East African monsoon that brings precipitation to the Ethiopian Highlands, can be substantially disrupted by volcanic eruptions: Manning *et al.* 2017.

uses do not precisely align with the flood data (figures 41 and 42). This is explained by his use of Zielinski's volcanic age-scale, which, as shown in Chapter 1, has since been revised. Elliot's alignment of Zielinski's eruption data with failed-flood data

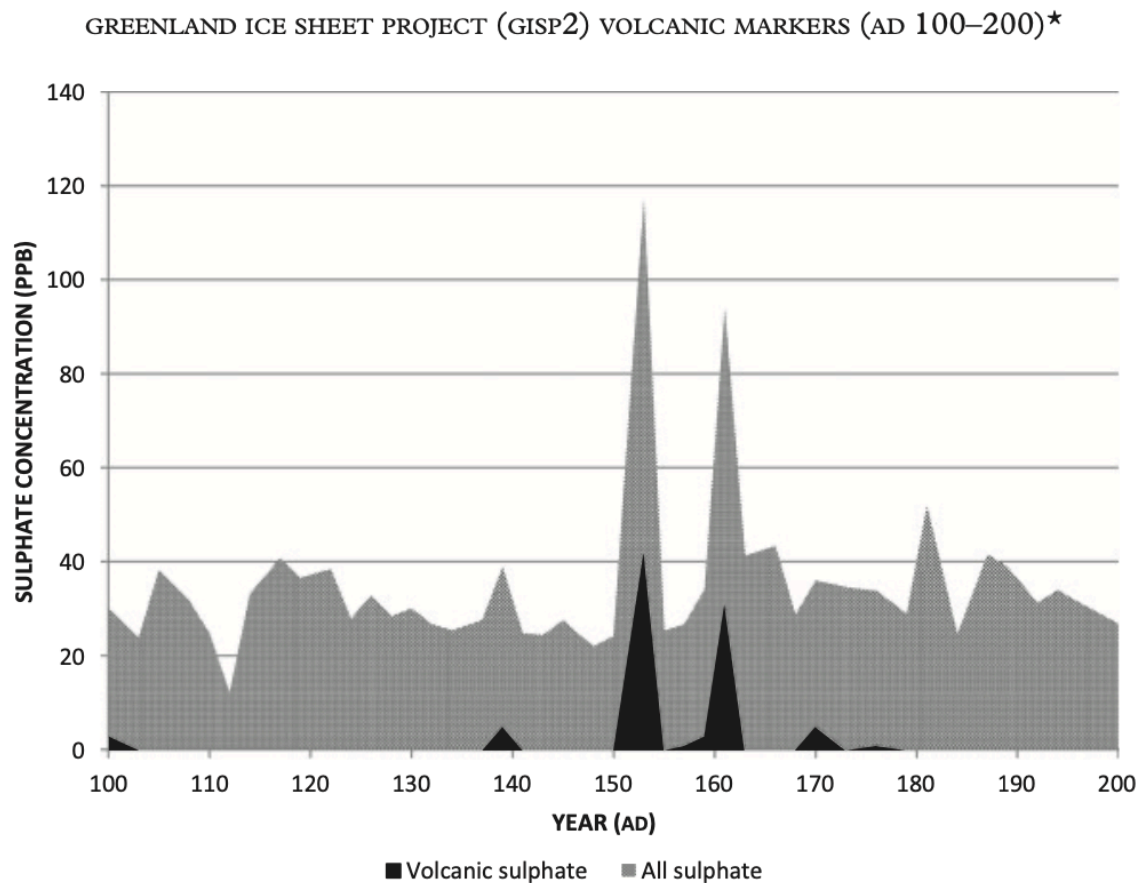


Figure 41. Zielinski's sulphate data indicating volcanic eruptions used by Elliot in his 2016 paper. The eruption chronology has since been changed (Chapter 1). (Elliot 2016)

from Habermann and Bonneau, implies that it is possible that the eruption spikes he shows – a peak in the early 150s and another in the early 160s – could be responsible for the apparent flood failures at AD 163-164 and AD 168-170 (Fig. 42).³¹⁵ However, the newly revised eruption timescale presented in Chapter 1 of this thesis aligns much more closely with the Nile-flood data (figures 42 and 43), and suggests

³¹⁵ Bonneau 1971 Bonneau 1993; Habermann 1997; Elliot 2016.

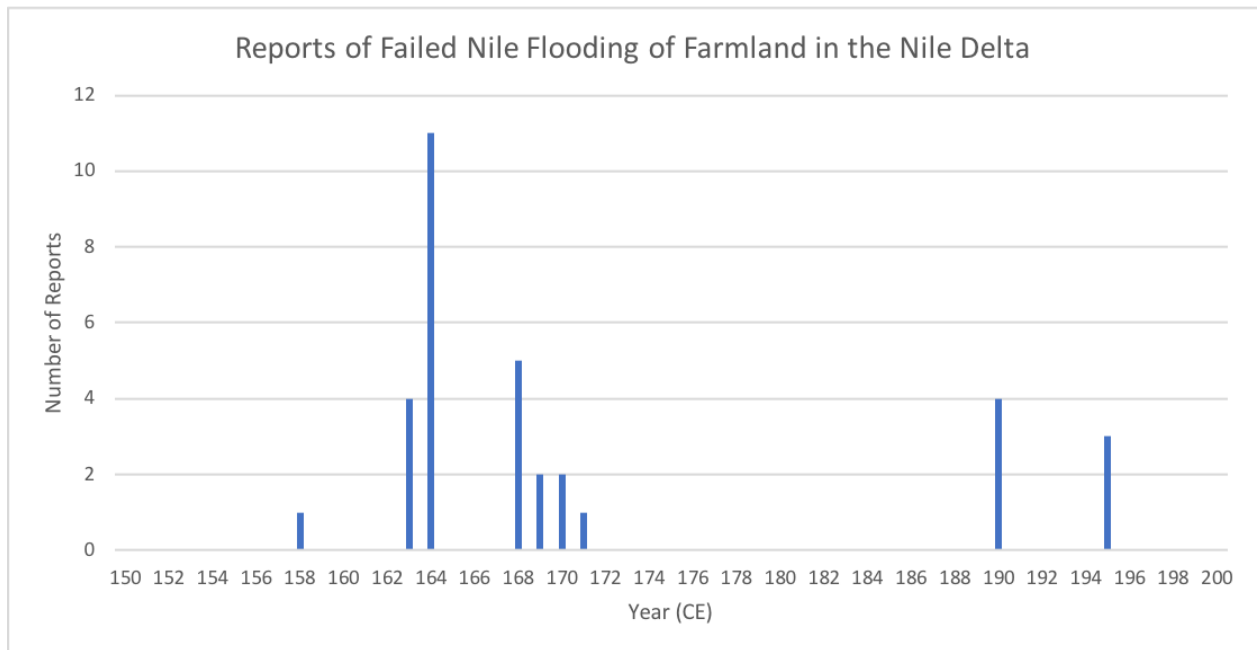


Figure 42. Failed Nile flooding (*abrochia*) in the Nile Delta. (My figure. Data: Habermann 1997; Elliot 2016)

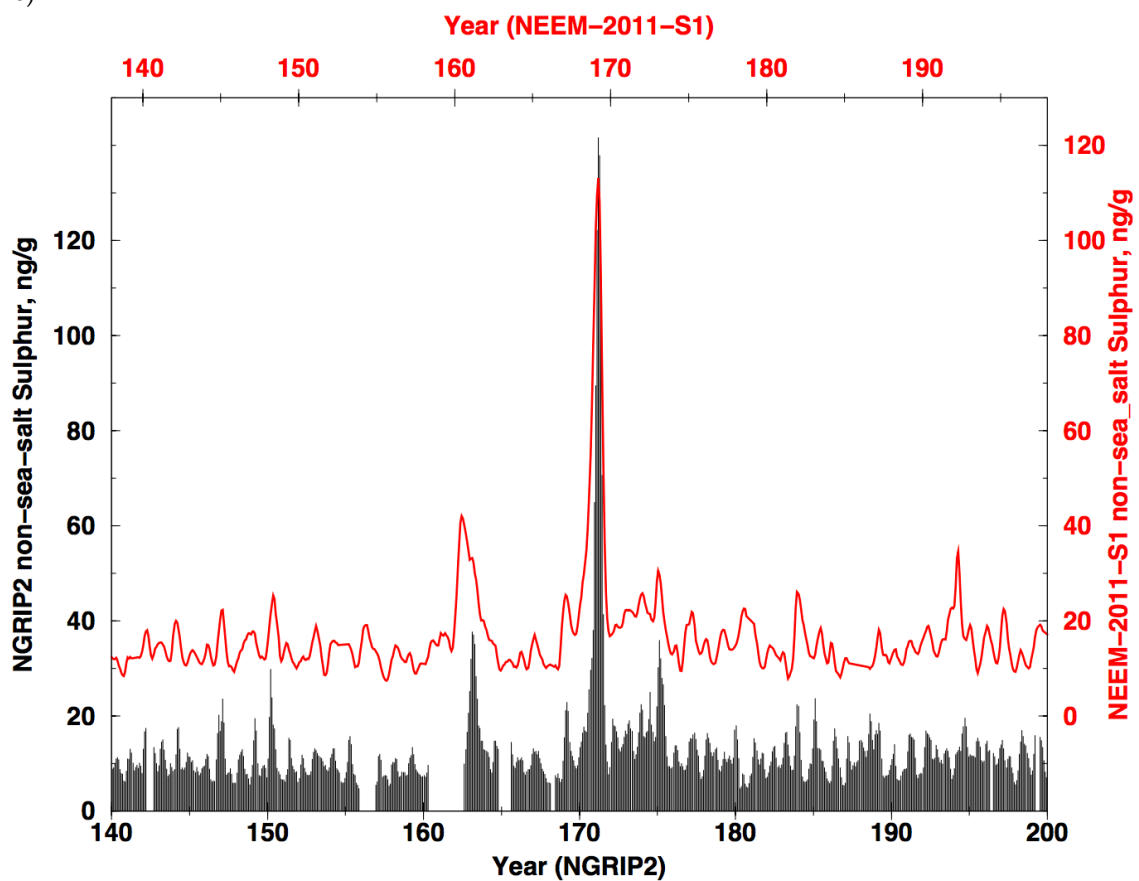


Figure 43. Sulphur measurements ($\sim$ monthly) from Greenland ice cores NGRIP2 (bottom “x” axis) and NEEM-2011-S1 (top “x” axis). Note that the two “x” axes are slightly offset. Data from: McConnell *et al.* 2018: NGRIP2; and Sigl *et al.* 2015: NEEM-2011-S1. (my figure)

that the eruptions at 163 and 170, and subsequent climatic variability recorded around the Mediterranean, had immediate effects in Egypt in the form of failed Nile floods.³¹⁶ The Nile is the lifeline to agriculture in Egypt. Climatic variability that potentially disrupted Nile floods, enough so to cause crop failure, could be a key supporting factor, or even the main factor, explaining land abandonment, revolts related to taxation, and overall decline uncovered in the province at the time of the Antonine Plague.

The idea of climate change as the key to decline in Roman Egypt is made more compelling by the fact that during the Antonine Plague there is evidence of pestilence only in the Nile Delta. Those who assume that second-century decline throughout Egypt was caused by the pandemic are relying on one piece of written evidence from the Delta. Elliot cites three potential pieces of direct evidence, but I argue that there is just one reliable account.³¹⁷

If we recall the written evidence discussed above, the two that are unreliable are the account from Crepereius Calpurnianus via Lucian, and a letter from Oxyrhynchus dated to the third century AD. Calpurnianus apparently wrote that the pestilence “descended into Egypt” from Ethiopia on its way to striking the Roman army at Nisibis, but Lucian calls this into question. Lucian notes the striking similarities between Calpurnianus’ narrative and Thucydides’ account for the Plague of Athens. He points out identical sentences and phrases in Thucydides, including that the disease “descended into Egypt” from Ethiopia.³¹⁸ Lucian implies that, while

³¹⁶ For more on deficient Nile flooding in the latter half of the second century CE see: McCormick *et al.* 2012, 188-194.

³¹⁷ Elliot 2016, 10-12.

³¹⁸ Thucydides, *History of the Peloponnesian War*, 2.48.1; Lucian, *How to Write History*, 15, trans. by A.M. Harmon, K. Kilburn, M.D. Macleod.

Calpurnianus probably witnessed or heard of an outbreak at Nisibis, he plagiarised its presentation, including the epidemic's origin. The second unreliable account is a letter from Oxyrhynchus from one Pausanias writing to his brother.³¹⁹ He warns him that he heard that a pestilence struck his brother's town (Antinoöpolis). The editors of this papyrus date it to the third century, with no indication of which part.³²⁰ With that said, since there are no apparent accounts of the Antonine Plague after AD 190, if this is to be linked with a pandemic it would be more reasonable to associate it with the Plague of Cyprian (AD 251/52 – 270).

The one reliable account of an epidemic in Egypt at the time of the Antonine Plague is from Thmouis (mentioned above), among the group of reports detailing Roman administration within the Mendesian Nome in the Delta of Egypt. As mentioned, in one report the *basilikos grammateus* accounts for the abandonment of Kerkenouphis, a village thought to have been located within the toparchy of Psanitēs or Ptempathiō, in AD 168-169.³²¹ He writes: “most of the men were killed by the godless Nikochites who attacked and burnt the village. The remainder died from the pestilential situation and only a small number fled”.³²² This account from the Nile Delta is the only direct evidence we have for an epidemic in Egypt during the second half of the second century AD. Since the account from Thmouis calls it “the pestilential situation”, it is fair to assume there were additional outbreaks in the Nile Delta. But it is difficult to extrapolate the situation in the Delta to the entire province of Egypt by corresponding it with evidence of societal disruption; especially since

³¹⁹ *Papyrus Oxyrhyncus* 14.1666.

³²⁰ Hunt and Grenfell 1914.

³²¹ *P. Thmouis* 1, 104.

³²² *P. Thmouis* 1, 104.12-18.

climatic variability was affecting the Nile-flood regime around the same time, as Elliot pointed out in 2016.

Port Said

<http://www.port-said.climatemps.com/temperatures.php>

	<u>Jan</u>	<u>Feb</u>	<u>Mar</u>	<u>Apr</u>	<u>May</u>	<u>Jun</u>	<u>Jul</u>	<u>Aug</u>	<u>Sep</u>	<u>Oct</u>	<u>Nov</u>	<u>Dec</u>	<u>Annual</u>
Average Max Temperature °C (°F)	17 (62.6)	18 (64.4)	19 (66.2)	22 (71.6)	25 (77)	28 (82.4)	30 (86)	30 (86)	29 (84.2)	27 (80.6)	24 (75.2)	20 (68)	24.1 (75.4)
Average Temperature °C (°F)	14.4 (57.9)	15.1 (59.2)	16.7 (62.1)	19.6 (67.3)	22.4 (72.3)	25.3 (77.5)	27.2 (81)	27.3 (81.1)	26.3 (79.3)	24.1 (75.4)	20.4 (68.7)	16.5 (61.7)	21.3 (70.3)
Average Min Temperature °C (°F)	11 (51.8)	12 (53.6)	14 (57.2)	16 (60.8)	20 (68)	23 (73.4)	24 (75.2)	25 (77)	24 (75.2)	22 (71.6)	19 (66.2)	14 (57.2)	18.7 (65.6)

Cairo

<http://www.cairo.climatemps.com/temperatures.php>

	<u>Jan</u>	<u>Feb</u>	<u>Mar</u>	<u>Apr</u>	<u>May</u>	<u>Jun</u>	<u>Jul</u>	<u>Aug</u>	<u>Sep</u>	<u>Oct</u>	<u>Nov</u>	<u>Dec</u>	<u>Annual</u>
Average Max Temperature °C (°F)	19 (66.2)	21 (69.8)	24 (75.2)	28 (82.4)	32 (89.6)	35 (95)	35 (95)	35 (95)	33 (91.4)	30 (86)	26 (78.8)	21 (69.8)	28.3 (82.9)
Average Temperature °C (°F)	13.6 (56.5)	14.9 (58.8)	16.9 (62.4)	21.2 (70.2)	24.5 (76.1)	27.3 (81.1)	27.6 (81.7)	27.4 (81.3)	26 (78.8)	23.3 (73.9)	18.9 (66)	15 (59)	21.4 (70.5)
Average Min Temperature °C (°F)	9 (48.2)	9 (48.2)	12 (53.6)	14 (57.2)	18 (64.4)	20 (68)	22 (71.6)	22 (71.6)	20 (68)	18 (64.4)	14 (57.2)	10 (50)	15.7 (60.2)

Asyut

<http://www.asyut.climatemps.com/temperatures.php>

	<u>Jan</u>	<u>Feb</u>	<u>Mar</u>	<u>Apr</u>	<u>May</u>	<u>Jun</u>	<u>Jul</u>	<u>Aug</u>	<u>Sep</u>	<u>Oct</u>	<u>Nov</u>	<u>Dec</u>	
Average Max Temperature °C (°F)	21 (69.8)	23 (73.4)	27 (80.6)	32 (89.6)	35 (95)	38 (100.4)	37 (98.6)	37 (98.6)	35 (95)	32 (89.6)	27 (80.6)	23 (73.4)	30.6 (87.1)
Average Temperature °C (°F)	11.7 (53.1)	13.9 (57)	17.4 (63.3)	23.2 (73.8)	27.2 (81)	29.6 (85.3)	29.6 (85.3)	29 (84.2)	26.9 (80.4)	23.4 (74.1)	17.4 (63.3)	13.3 (55.9)	21.9 (71.4)
Average Min Temperature °C (°F)	6 (42.8)	7 (44.6)	11 (51.8)	15 (59)	19 (66.2)	22 (71.6)	20 (68)	22 (71.6)	20 (68)	18 (64.4)	13 (55.4)	9 (48.2)	15.2 (59.3)

Luxor

<http://www.luxor.climatemps.com/temperatures.php>

	<u>Jan</u>	<u>Feb</u>	<u>Mar</u>	<u>Apr</u>	<u>May</u>	<u>Jun</u>	<u>Jul</u>	<u>Aug</u>	<u>Sep</u>	<u>Oct</u>	<u>Nov</u>	<u>Dec</u>	
Average Max Temperature °C (°F)	23 (73.4)	26 (78.8)	30 (86)	35 (95)	40 (104)	41 (105.8)	41 (105.8)	41 (105.8)	39 (102.2)	37 (98.6)	31 (87.8)	25 (77)	34.1 (93.4)
Average Temperature °C (°F)	13.8 (56.8)	15.9 (60.6)	20.2 (68.4)	25.6 (78.1)	29.6 (85.3)	32.2 (90)	32.3 (90.1)	31.8 (89.2)	29.7 (85.5)	25.9 (78.6)	20 (68)	15.1 (59.2)	24.3 (75.8)
Average Min Temperature °C (°F)	5 (41)	7 (44.6)	10 (50)	15 (59)	21 (69.8)	21 (69.8)	23 (73.4)	23 (73.4)	21 (69.8)	19 (66.2)	12 (53.6)	7 (44.6)	15.3 (59.6)

Table 3. Average temperatures for four modern-day cities in Egypt ranged in order from north to south along the Nile. (<http://www.climatetemps.com/temperatures.php>)

As the discussion above of the epidemiology for smallpox showed, the virus is sensitive to climatic factors – it survives better in cooler temperatures and lower

humidity, and it loses infectivity above 30°C and 55% humidity.³²³ It is plausible, despite the cool and dry phase in the second half of the century, that much of Egypt, and especially the Nile Valley, was still too hot for the Antonine Plague to survive year-round. Table 3 shows the annual average temperatures today for four cities at different points along the Nile, and Figure 44 shows the location of these cities along the Nile. The table and figure show that it gets hotter as one moves southward along the Nile. Even if average temperatures for each were a few degrees cooler in ancient

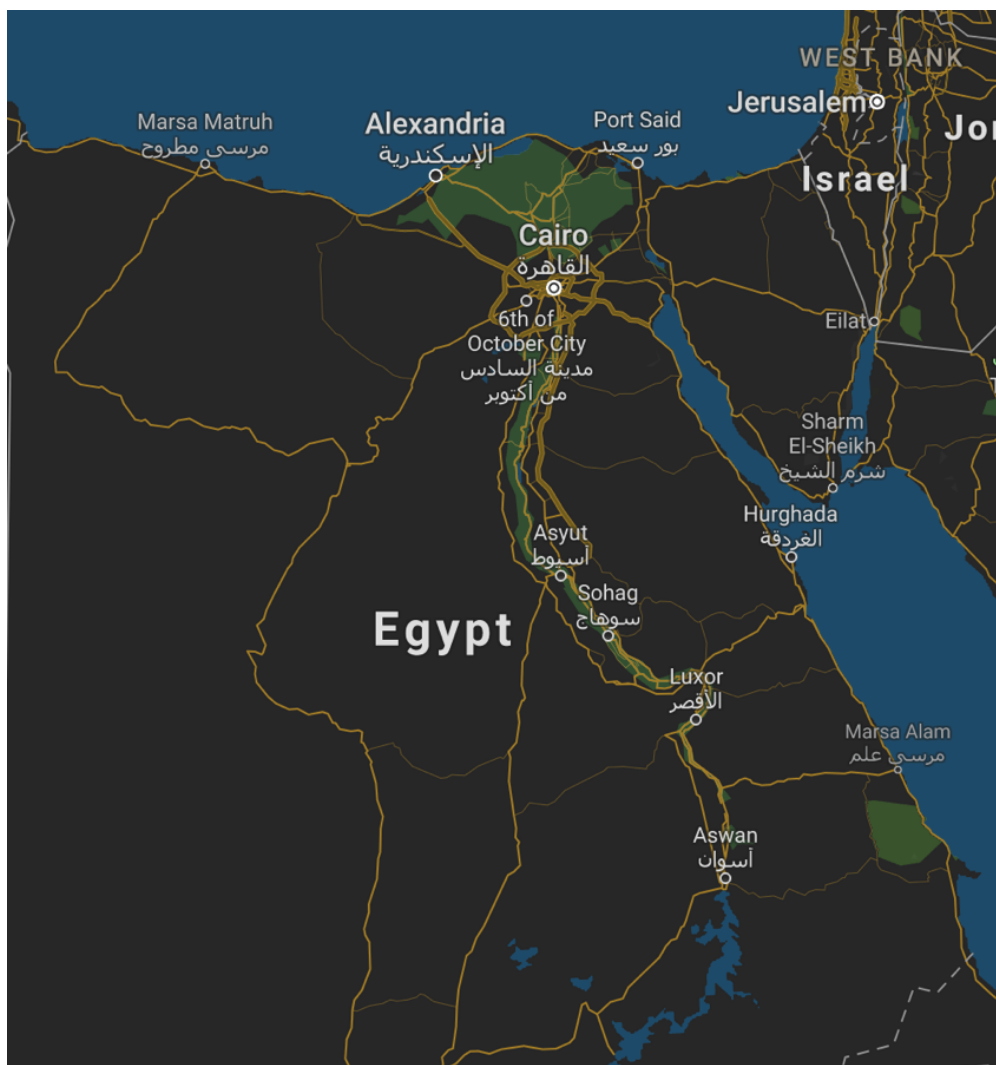


Figure 44. Map of Egypt showing the locations of cities in Table 2. (Google maps)

³²³ Nishiura and Kashiawagi 2009, 5.

times, areas around Asyut and Luxor may have been too hot for smallpox (or potentially a closely related ancestor) to survive there. The virus could have moved south during the winter but would have probably died out when temperatures rose again in the spring and summer. The hot climate of Egypt could be why we have just one piece of direct evidence for the Antonine Plague in Egypt, and no evidence of outbreaks south of the cooler Delta.

Discussion

Evidence from the northern half of the Roman Empire indicates that the Antonine Plague had a substantial effect. The lead-pollution evidence is most compelling, exhibiting the halting nature of the pandemic, both demographically and economically. While not definitive, the evidence from the Catacombs of Marcellinus and Peter may be archaeological evidence for the Antonine Plague in Rome; it would certainly cohere with the accounts of Galen, Herodian, and Cassius Dio. The mass grave at Gloucester in Britain potentially shows the pandemic's far-reaching extent, and documentary evidence from the gold mine at Roşia Montană might demonstrate a mining settlement depressed by both disease and war. Documentary evidence from Egypt confirms that the pestilence at least struck the Delta, but possible not further south, while the Nile-flood data suggests that the province as a whole was at least equally affected by climate change in the latter half of the second century. Overall for the Empire, the evidence indicates that Asia Minor and parts of Europe (i.e. Italy, Gaul, Britain, Spain) were probably greatly affected by the Antonine

Plague, as was the Nile Delta, while we lack such evidence for the rest of Egypt, the Levant, and Roman North Africa.

Conclusions

The written evidence indicates that a devastating pestilence affected the Roman Empire from AD 165 until around 190. I have argued here that the disease probably spread from a reservoir in central Asia to legions of the Roman army, who had been fighting the Parthians in the area under emperor Lucius Verus since AD 162. The army then transferred the disease to Roman territory in western Asia in AD 165, after which, it spread to nearly every region of the Empire, as the written evidence, archaeological evidence, and proxy evidence suggests. The disease was probably smallpox, or a closely related ancestor of smallpox, as illustrated by Galen's symptomology, its virulence, and the speed with which it spread across the Empire. The smallpox virus is sensitive to changes in climate, with incidence rising when temperatures cool and humidity decreases. The climate-proxy evidence shown in Chapter 1 indicates that central Asia experienced substantial reductions in temperatures (over 4°C in some regions) around the start of the pandemic, and evidence from western Asia shows rises in aridity around the same time. A large volcanic eruption of the Ksudach volcano in eastern Russia in AD 163 was the most likely cause of this dramatic cooling and drying. It seems not to be a coincidence that the disease that probably caused the Antonine Plague, which, if smallpox or a closely-related ancestor, has increased incidence in cooler and drier conditions, struck the Roman army, and eventually the Empire, soon after those climatic changes. The Antonine pandemic had a significant effect on the Empire. The written

and archaeological evidence suggest that many people died, and the proxy evidence, such as the lead pollution data, indicate that the Roman economy suffered. In some areas (e.g. Egypt), it is hard to tell whether the decline observed in the proxy evidence was caused by the pandemic or climate change. But in many areas (e.g. Europe), the decline is more indicative of pandemic disease. In any case, the Antonine pandemic appears to have had an important effect on the Empire just before the most tumultuous century it was to see. In the next chapter, we turn our focus to that century as we examine potential causes and effects of the third-century Plague of Cyprian.

The Plague of Cyprian

Introduction

With his article in the *Journal of Roman Archaeology* (2015), Kyle Harper put the Plague of Cyprian (AD 251/2 – 270) on the map of the “great plagues of antiquity”, arguing that the pestilence best described by St. Cyprian of Carthage was not an insignificant event in the Empire’s history.³²⁴ Harper’s tracing of every source, contemporary or later, that mentions a mid-third-century AD epidemic is painstaking; the first work of its kind for the Plague of Cyprian. Ultimately, however, he works to make the pandemic bigger than it is; extrapolating the limited firm evidence we have to the whole empire. The impact of the third-century epidemic along the northern coast of Africa is apparent, attested by strong literary evidence, and some funerary evidence that potentially shows the deleterious footprint it left in Upper Egypt.³²⁵ Evidence also suggests that it struck, and may have had an important effect in, Rome. Its impact on the rest of the Empire is far less clear, largely because the “Crisis of the Third Century”, encompassing the Plague of Cyprian, comprises much more than a pestilence. The “crisis”, typically dated from the assassination of Severus Alexander (AD 235) until the rise of Diocletian (mid-280s), also includes barbarian invasions, rebellions, civil wars, political unrest, coin debasement, and notable climate change. Harper is right to think that the Plague of Cyprian affected the Empire to a degree, and that it should get more attention in scholarly discussion; the literary mentions alone warrant that. But, with the evidence

³²⁴ Harper 2015.

³²⁵ Tiradritti 2014.

we do and do not have, and with the other elements of the “crisis” clouding the picture, he is over-hasty in suggesting that its impact was comparable to the Antonine or Justinianic plagues.³²⁶ That, at this stage, is not visible.

As in the previous chapters of this dissertation, science will here help tease out information about the Plague of Cyprian and the “Crisis of the Third Century.” Where literary sources can be exaggerated or ambiguous, other aspects of the so-called crisis, such as coin debasement and third-century climatic variability, are more precisely understood thanks to scientific analysis.³²⁷ For example, climate cooling, as we saw in Chapter 1, is clear in the climate proxies; particularly the evidence for early-third-century reductions in solar activity, and the massive volcanic eruption of the 260s and subsequent cold phase. These climatic phenomena factor into my assessment of whether or not climatic variability played a role in the appearance of the Plague of Cyprian. But before examining the possible relationship between climate and epidemic, we must first diagnose the disease, which I believe Harper did correctly in 2015 by proposing a viral haemorrhagic fever as the culprit.³²⁸ I review the relevant epidemiology for the best candidates to reaffirm his choice, but I propose a more specific form of the disease as that responsible for the third-century pandemic. Thus, science has no small place in this chapter.

The chapter first reviews the ancient sources, considering which of the written sources carry more weight than others. This helps give the epidemic a chronology and trace its path through the Empire, while keeping in mind that each fleeting mention of disease may not carry the same weight as our main source for

³²⁶ Harper 2015, 224.

³²⁷ Butcher and Ponting 2015.

³²⁸ Harper 2015.

symptomology, St. Cyprian of Carthage, or the five other contemporary sources – Dionysius of Alexandria (through Eusebius), Pontius of Carthage, the *Thirteenth Sibylline Oracle*, and Dexippus and Philostratus related through later sources.

Harper's fundamental article and the scant additional secondary literature that discuss the Plague of Cyprian is also discussed throughout this section.

The next section considers the archaeology, reviewing evidence that might bear on our pestilence. The following section focuses on symptomology, diagnosis, and epidemiology, assessing the best candidate for the disease responsible for the pestilence; it reviews the symptomology mentioned by Cyprian, using his detailed description of the disease as the principal evidence for diagnosis. Additionally, I use the other literary sources for epidemiology. Next, the section considers virological and epidemiological studies to help confirm, through examination of genealogical histories of viral haemorrhagic fevers, that our diagnosis is not contradicted by the science. Finally, I develop Harper's argument that the Plague of Cyprian might have been a viral haemorrhagic fever, proposing, with help from a 2018 Master's thesis, that an early strain of Ebolavirus is the most likely cause of the third-century pandemic.³²⁹

The penultimate section focuses on the potential relationship between third-century climate change and the Plague of Cyprian. I briefly review the climatic situation for the period before and during the epidemic, indicating when and where the Empire had experienced climatic variability in the mid-to-late third century. Whether or not climate change does coincide with the epidemic, I review the epidemiology for Ebola to determine if studies have shown any connection between

³²⁹ Kearns 2018.

climate and the virus. Ultimately, the section concludes by showing that the data on the relationship between Ebola (as well as other VHFs) and climate show no positive association.

The last section of this chapter works to assess the impact of the Plague of Cyprian on the Empire, and where it fits into the “Crisis of the Third Century”. For this, I first revisit the ancient sources, determining, based on qualitative analysis, where the epidemic actually struck, and where it hit the hardest. The section considers the lenses used by Harper – financial, socio-political, geo-political, and cultural – to assess the potential effects of the epidemic, determining if we can move beyond the questions he poses. I take into account the literary sources, archaeological evidence, and economic proxy evidence in an attempt to disentangle the effects of the Plague of Cyprian from other factors contributing to the “Crisis of the Third Century”. The analysis shows that, despite what Harper suggests, it is even more difficult to measure the effects of the Plague of Cyprian than of the Antonine Plague with so much else in a downward spiral in the mid-third century AD.

A picture of the Plague of Cyprian emerges from the superficial mentions by numerous non-contemporary sources, many of whom have reproduced the accounts of their predecessors, of a pandemic for which there is a lack of clear archaeological evidence, that has seemingly no relationship with climate, and happened amidst a flurry of other difficulties that the Empire did not settle until long after the pestilence subsides. The only thing clear about the Plague of Cyprian is the vivid description of the disease told by the man after which it is named.

Written Evidence

As mentioned, we have five sources contemporary with the Plague of Cyprian. It should again be noted that we have Harper to thank for compiling even the most obscure of sources for this pestilence.³³⁰ However, his way of arranging them is somewhat convoluted, jumping between contemporary and later sources without much semblance of timeline. Therefore, it makes sense here to go through the sources in chronological order, giving most attention to those contemporary with the pandemic (AD 251 – 270).

St. Cyprian of Carthage

The first source to mention our plague is qualitatively the most important. Amidst persecution and exile, St Cyprian was bishop of Carthage from AD 249 until his public execution in 258, during Valerian's Christian persecution (AD 257 – 260).³³¹ He mentions the third-century pestilence in two of his works, *Ad Demetrianum* and *De Mortalitate*. The former is an address to the proconsul of Africa, Demetrianus, responding to the proconsul's claims that Christians are responsible for the pestilence, war and famine consuming Carthage at the time. The latter is a sermon addressed to the Christians of Carthage, seemingly when the gravity and biological effects of the pestilence were being fully realised.³³²

On the works' dating, Harper points out that *Ad Demetrianum* was probably written just before *De Mortalitate*, citing Strobel and Sage.³³³ Sage thinks the former

³³⁰ Harper 2015.

³³¹ Sage 1975, 95-97; Strobel 1993, 146-184; Harper 2015, 229.

³³² Price 1999, 113.

³³² Scourfield 1996, 12-14.

³³³ Harper 2015, 229.

was written later than AD 251, since much of its content reveals political chaos, which he says, “the death of Decius alone would justify” (June 251); Strobel agrees.³³⁴ Sage further constrains its date to early 252, since there is no direct mention by Cyprian of Christian persecution, indicating the approximate year-long period of relative peace that occurred between the rise of Trebonianus Gallus (June 251) and Gallus’ own Christian persecution (in late AD 252).³³⁵ In this line of thought, *Ad Demetrianum* could have been written between mid 251 and late 252. Therefore, as *De Mortalitate* mentions both the persecution and full-scale effects of the plague, Sage dates that work after *Ad Demetrianum* to late 252, during Gallus’ oppression of Christians, a plausible conclusion.³³⁶

The mention of plague in *Ad Demetrianum* is brief and grouped in with mentions of war and famine, making it, qualitatively, far less important than the later work.³³⁷ However, as the first mention of our pestilence it is still significant, since it allows us to date the start of the Plague of Cyprian in Africa Proconsularis to between mid 251 and late 252. Its start was recently dated to AD 249, but I discuss below (in the section on Dionysius of Alexandria) why 249 does not fit with the evidence. *De Mortalitate* is very important for both the epidemic’s symptomology and its impact on Carthage. As an address to his Christian brethren during a difficult time – a sermon so to speak – we can trust this work; Cyprian composed it for a contemporary Christian community, one experiencing the pestilence first-hand, so

³³⁴ Sage 1975, 171; Strobel 1993, 171.

³³⁵ Sage 1975, 380-381.

³³⁶ Sage 1975, 381.

³³⁷ Cyprian, *Ad Demetrianum*, 2.2.

arguing that he manufactured its symptomology is implausible.³³⁸ Cyprian's description of the disease goes as follows:

*"This test of faith, that now as the strength of the body is dissolved, the bowels dissolve in a stream; a fire that begins in the inmost depths burns up into wounds in the throat; that the intestines are shaken with continuous vomiting; that the eyes are set on fire from the force of the blood; that the infection of the deadly putrefaction cuts off the feet or other extremities of some; and that as weakness prevails through the failures and losses of the bodies, the gait is crippled or the hearing is blocked or the vision is blinded."*³³⁹

Earlier in *De Mortalitate*, Cyprian says that "the pain in the eyes, the attack of fevers, and the ailment of all the limbs are the same among us and among the others, so long as we share the common flesh of this age," the second half of the sentence indicating that the disease affects both Christians and pagans alike.³⁴⁰ Cyprian describes a disease that begins with high fever (and loss of strength), which develops into a severe strep-like throat (most likely with lesions or ulcers), burning of the (probably bloodshot) eyes, and gastrointestinal affliction. The later stage of the disease causes blindness (probably related to the burning in the eyes), hearing loss, and rotting of the limbs, which surely adds to the loss of strength, and probably leads to the "crippled gait."

Without Cyprian's account we would have very little to help identify the disease, except for the short passage by Dionysius of Alexandria (below) that adds to

³³⁸ Harper 2017, 229.

³³⁹ Cyprian, *De Mortalitate*, 14: *Hoc, quod nunc corporis vires solutus in fluxum venter eviscerate, quod in faucium vulnera conceptus medullitus ignis exaestuat, quod assiduo vomitu intestina quatiuntur, quod oculi vi sanguinis inardescunt, quod quorundam vel pedes vel aliquae membrorum partes contagio morbidae putredinis amputantur, quod per iacturas et damna corporum prorumpente languore vel debilitatur incessus vel auditus obstruitur vel caecatur aspectus.* (my translation)

³⁴⁰ Cyprian, *De Mortalitate*, 8: *et oculorum dolor et impetus febrium et omnium ualitudo membrorum cum ceteris communis est nobis, quamdiu portatur in saeculo caro ista communis.* (Harper's translation)

the disease's epidemiology. In the next section, I use mainly Cyprian's account to diagnose the disease. Cyprian also gives us a definitive *terminus post quem* for the pestilence, which was recently argued to have been first mentioned by Dionysius. However, for the reasons discussed in the subsection below, Cyprian should be considered the first literary source to mention the third-century-AD pestilence.

Dionysius of Alexandria

Our plague is mentioned in letters written by Dionysius of Alexandria in the AD 250s (after the summer of 251), possibly the early 260s. The original letters are lost, but they are related, word-for-word, by Eusebius in book seven of his *Ecclesiastical History* (7.21 and 7.22). Dionysius, Bishop of the Coptic Orthodox Church in Alexandria, wrote the letters to Christian "brethren" suffering large-scale persecution throughout the metropolis during the imperial reigns of Decius (September 249 – June 251) and his son Herrennius Etruscus (May – June 251), Trebonianus Gallus (June 251 – August 253) with Decius' other son Hostilian (July – November 251) and then Gallus with his own son Volusianus (November 251 – August 253), Aemilianus (June – September 253), and finally Valerian (253 – 260) and Gallienus (253 – 268) (Table 4).³⁴¹ Karl Strobel dates the letters mentioning pestilence to 249 and 250, with which Harper uncritically agrees.³⁴²

³⁴¹ Eusebius, *Ecclesiastical History*, 7.21.

³⁴² Strobel 1993, 190-194; Harper 2015, 227: in a footnote Harper claims, "The letter has been variously dated [without supplying the other citations], as late as the 260s, but Strobel argues convincingly that the letter should date to the Spring of 249."

The letters appear sequentially in the *Ecclesiastical History* as festal letters written to brethren at Easter. The letter in book 7.21 depicts an Alexandria racked by Christian persecution, civil war, and repeated visits by the third-century pestilence. The letter in 7.22 focuses mainly on the pestilence, describing the ubiquitous death

Christian Persecutor(s)	Beginning	End
Pagan Alexandrians	Early 249	Summer 249
Decius (250 edict)	January 250	Spring 251 ³⁴³
Trebonianus Gallus (possibly localised to Rome and Carthage)	May 252 ³⁴⁴	Late 252/Early 253
Valerian (2 edicts: 257 and 258) ³⁴⁵	Summer 257 ³⁴⁶	Early 260

Table 4. Persecutions during Dionysius' time as Patriarch of Alexandria.

and suffering it was causing in Alexandria. The debate on dating the letters is ongoing; Strobel argued that both letters were written amidst a brief but concentrated persecution in the reign of Phillip the Arab in 249 – 250 that preceded the Decian persecution. The Decian persecution followed the issuing of an edict in 250 by Decius ordering an Empire-wide sacrifice to the gods, an act in which Christians could not take part, resulting in their persecution. Strobel thinks that Dionysius is referring to the pre-Decian pogrom described in book six of Eusebius' *Ecclesiastical History* – which covers the period leading up to and including the reign

³⁴³ Drinkwater 2005, 39.

³⁴⁴ Frend 1970, 94-95.

³⁴⁵ Keresztes 1975, 83-84; Drinkwater 2005, 42.

³⁴⁶ Keresztes 1975, 83.

of Decius – implying that, chronologically, the plague letters belong there.³⁴⁷

Discussing Dionysius' letter from book 7.21, Strobel proposes that the "civil war ended again before the outbreak of the great epidemic, the so-called plague, which in turn had its full effect in Alexandria before the decree of sacrifice of Decius arrived and before the beginning of Lent in AD 250. Christians had only been given a short respite in the city in AD 249 before the news of the violent change of throne in Rome, the end of the Christian-friendly rule of Phillip the Arab, came and the fear of new adversity grew."³⁴⁸ Strobel is, seemingly, connecting the events in time by Dionysius' use of the phrase "briefest breathing-space" (or, as Strobel calls it, "kurze Atempause": short respite) in the earlier letter mentioned above – to Fabius of Antioch that details the pre-Decian persecution AD 248 – 249 (quoted by Eusebius in the preceding book, 6.41) – with the same phrase in the letter on the pestilence in 7.22. He cites the similarity in wording between the lines (6.41.9 and 7.22.6), as well as Dionysius' depiction of Alexandria up in arms amidst the oppression as reasons that both pestilence letters must pre-date the Decian persecution (early 250).³⁴⁹ Harper, in his article on the Plague of Cyprian in 2015, took Strobel's hypothesis at face value.³⁵⁰

However, the similarities in phrasing are just that, similar wording, and it does not "convincingly" connect the two letters. Christians in Alexandria

³⁴⁷ Strobel 1993, 191; Eusebius, *Ecclesiastical History*, 6.41.

³⁴⁸ Strobel 1993, 191: Dieser Bürgerkrieg ging wiederum noch vor dem Ausbruch der großen Epidemie, der sogenannten Pest, zu Ende, die ihrerseits bereits vor dem Eintreffen des Opferediktes des Decius und vor dem Beginn der Fastenzeit des Jahres 250 n. Chr. ihre volle Wirkung in Alexandria entfaltet hatte. Den Christen war 249 n. Chr. in der Stadt nur eine kurze Atempause vergönnt gewesen, ehe die Nachricht von dem gewaltsamen Thronwechsel in Rom, dem Ende der christenfreundlichen Herrschaft des Phillipus Arabs, eintraf und die Furcht vor neuer Bedrängnis wuchs. (my translation)

³⁴⁹ Eusebius, *Ecclesiastical History*, 6.41.9: καὶ σμικρὸν μὲν προσανεπνεύσαμεν; *ibid*, 7.22.6: βραχυτάτης δὲ ἡμῶν τε καὶ αὐτῶν τυχόντων ἀναπνοῆς.

³⁵⁰ Harper 2015, 227.

experienced waves of persecution from AD 249 until the early 260s, amounting to numerous points in time when Christians had a chance, metaphorically, to “breathe again” (more on this below). Also, while book six of Eusebius details an earlier persecution of Christians, the chronology suggested by Strobel does not fit with the overall content of the letters mentioning pestilence, which are quoted in the following book. Additionally, Eusebius’ *Chronicon*, a work now lost but preserved in Latin translation by the fourth-century Saint Jerome, says that the epidemic hit Alexandria in AD 253.³⁵¹ It is also clear that Eusebius himself believed that these two letters belonged amongst those Dionysius wrote after Decius’ demise in AD 251. Book seven begins with the murder of Decius and the rise of Gallus (when Christian persecution briefly abated but returned later in his reign), continuing with letters describing Valerian’s oppression of Christians throughout the mid-to-late 250s, and finally the benevolence of Gallienus (son of Valerian) towards Christians after his father’s death, all organised in a relatively coherent timeline, with the pestilence letters in the middle.³⁵² In a recent article (2020), David DeVore proposes that Eusebius deliberately displaced Dionysius’ letters to better fit the thematically Christian aim of his *Ecclesiastical History*. DeVore suggests that the pestilence letters belong either grouped with the letter to Fabius of Antioch (detailing events that happened in early 249) in Eusebius’ book six, due to their similar content (DeVore’s preferred choice, and the same stance as Strobel), or in book seven within the history of Gallus and Volusianus (AD 253), matching Eusebius’ *Chronicon*.³⁵³ As will be

³⁵¹ Jerome, *Chronicle* 2.CCLVIIIa p. 219 Helm = the Armenian translation of the Chronicle: Karst , 226.

³⁵² Eusebius, *Ecclesiastical History*, 7.

³⁵³ Sordi 1962, 127-132: first proposed that the pestilence letters should match *Chronicon* and be dated to around AD 253; DeVore 2020, 12-13.

shown below, the latter theory is the most likely scenario for 7.22, and the letter in 7.21 appears to have been written a few years later still.

Dionysius' letter in book 7.21 of the *Ecclesiastical History* portrays an Alexandria suffering from persecution for many years. He also explains how "constant plagues" had been harrowing the city. His description does not fit the brief, but severe, persecution that occurred in Alexandria in AD 249, and there is no evidence that the Plague of Cyprian had yet begun in the 240s. In the letter that Eusebius quotes in book 7.21, Dionysius, back in Alexandria *after* a period of exile (Table 5), notes the large-scale mortality suffered there as follows:

*"Yet men marvel and are at a loss as to whence come the constant plagues, whence the grievous diseases, whence the various forms of death, whence the manifold and great human mortality, why this greatest of cities no longer contains within it so great a multitude of inhabitants, from infant children up to those extremely advanced in years, as it used formerly to support of those known as men of green old age! Nay, those of forty years old and up to seventy were then so numerous, that the full total of their number is not to be reached now, when those from fourteen to eighty years have been registered and reckoned together for the public food-ration; and the youngest in appearance have become of equal age, so to speak, with those who long ago were the oldest."*³⁵⁴

Dionysius deduces, apparently from public records for the civic annona, that more than half of the population have perished (more on this below).³⁵⁵ He attributes this mortality to both persecution and the "constant plagues" that visited the city. If Dionysius wrote the letter in 249, it would mean the population of Alexandria had been devastated (even if the numbers are exaggerated) in a few months' time, which

³⁵⁴ Eusebius, *Ecclesiastical History*, 7.21.9, trans. by J.E.L. Oulton.

³⁵⁵ Eusebius, *Ecclesiastical History*, 7.21.9-10.

is implausible. It would also mean that this is the first mention of the epidemic in Alexandria (and anywhere), two years before Cyprian. If the pestilence was a factor contributing to substantial population loss, which it probably was, then, to account for such loss, we must assume that it had been affecting the city for multiple years leading up to AD 249. It is not wholly impossible that the Plague of Cyprian began in Alexandria in the mid-240s; but this would be a much earlier start-date than the other literary evidence indicates. We will come back to dating the letter in book 7.21, but first let us look at the letter in book 7.22.

Exiling Emperors	Date exiled	Probable return to Alexandria
Decius	January AD 250	Spring AD 251 – 253
Valerian and Gallienus	Easter AD 258 – Summer 258	Early AD 260

Table 5. Periods that Dionysius of Alexandria was in exile. We presume that Dionysius was in Alexandria outside of these periods of exile.

In the second pestilence letter, in Eusebius' book 7.22, Dionysius explains how the epidemic wreaked havoc in Alexandria, saying, "...all men mourn, and wailings resound throughout the city because of the number of dead and of those that are dying day by day."³⁵⁶ His language suggests that this was the first wave of the pestilence; that is, the worst phase of a pandemic. If we compare his language in 7.22 with that in 7.21 ("the constant plagues"), it implies that the letter in 7.22 might pre-date the letter in 7.21. Dionysius continues:

"At first they drove us out, and alone we kept our festival at that time also, persecuted and put to death by all, and every single spot where we were afflicted became for us a place of festive assembly, field, desert, ship, inn, prison; but the brightest of all festivals was kept by the perfect martyrs, when they feasted in heaven. And, after that, war and famine came upon us, which we bore along with the heathen. Alone we endured all the injuries

³⁵⁶ Eusebius, *Ecclesiastical History*, 7.22.3.

*they inflicted upon us, while we had the benefit besides of what they wrought upon each other and what they suffered: and we found our joy once more in the peace of Christ, which He has given to us alone. But when the briefest breathing-space had been granted us and them, there descended upon us this disease..."*³⁵⁷

Strobel and Harper think the letter in book 7.22 dates to just after the pre-Decian persecutions.³⁵⁸ However, there is no evidence that Dionysius was driven out of Alexandria during the pre-Decian persecution, while there is evidence of his ousting both in 250 and in the later 250s. In book 6.40, Eusebius quotes a letter concerning the pursuit of Dionysius by Decius and his prefect of Egypt, Aurelius Appius Sabinus (prefect from September 14, 249 until at least July 17, 250, but probably later), which is said immediately to follow Decius' imperial edict (January 250).³⁵⁹ After hiding for four days in his own house, Dionysius was able to escape Alexandria unseen, ending up in Taposiris (30 miles southwest of Alexandria).³⁶⁰ It is not clear when exactly he returned to the city, but presumably at some point after Decius' demise in the summer of 251 (Table 5). Dionysius was exiled again under Valerian, during "the storm of persecution that raged during his day", after Dionysius was brought before Aemilianus, deputy prefect of Egypt in 258.³⁶¹ Dionysius claims, responding to Aemilianus' interrogation about his Christian faith, "I testified outright that I worship the only God and none other, nor would I change or ever cease to be a Christian. Thereupon he bade us to depart to a village near the desert, called Cephro."³⁶²

³⁵⁷ Eusebius, *Ecclesiastical History*, 7.22.4.

³⁵⁸ Strobel 1993, 199; Harper 2015, 228.

³⁵⁹ Eusebius, *Ecclesiastical History*, 6.40.

³⁶⁰ Eusebius, *Ecclesiastical History*, 6.40.4.

³⁶¹ Eusebius, *Ecclesiastical History*, 7.11.3.

³⁶² Eusebius, *Ecclesiastical History*, 7.11.5.

Additionally, in the letter from book 7.22 Dionysius mentions war and famine (above), the latter probably the result of a drought, around the same time that Cyprian mentions drought and famine in his *Ad Demetrianum* (AD 251/252).³⁶³ In *Ad Demetrianum*, Cyprian says, “And you wonder or complain in this your obstinacy and contempt, if the rain comes down with unusual scarcity; and the earth falls into neglect with dusty corruption; if the barren glebe hardly brings forth a few jejune and pallid blades of grass; if the destroying hail weakens the vines; if the overwhelming whirlwind roots out of the olive; if drought staunches the fountain; a pestilence breeze corrupts the air; the weakness of disease wastes away man.”³⁶⁴ Could the drought mentioned by Cyprian be the same drought/famine Dionysius is talking about? In looking at Daneille Bonneau’s data for Nile-river floods, the only low-flood year in the AD 240s is year 242, and the sole low-flood year for the 250s is year 258; however, she has no data for years 251 – 253. AD 242 is too early for Dionysius’ letter 7.22, and 258 is too late – given that Eusebius’ *Chronicon* puts the third-century pestilence in Alexandria in AD 253, and that Dionysius’ language in 7.22 indicates the first wave of the pandemic (probably beginning in Alexandria in the early 250s, similar to Carthage), it is very unlikely the letter was written in 258.³⁶⁵ Cyprian most likely composed *Ad Demetrianum* in late 251/early 252 (above). He discusses the drought happening alongside the third-century pestilence. Dionysius says that Alexandria’s drought happened after the Christian community was “persecuted and put to death by all”, but before the pestilence, and at the same time as war.³⁶⁶ It is

³⁶³ Cyprian, *Ad Demetrianum*, 7 and 10.

³⁶⁴ Cyprian, *Ad Demetrianum*, 7, trans. by Ernest Wallis; later in book 10 Cyprian says: “You complain of barrenness and famine, as if drought made a greater famine than rapacity...you complain that the heaven is shut up from showers.”

³⁶⁵ Bonneau 1971, 256.

³⁶⁶ Eusebius, *Ecclesiastical History*, 7.22.4.

plausible that the famine/drought in letter 7.22 is the same as Cyprian's. The chronology of events fits. In addition, palaeoclimatic data for precipitation from the Libyan Sahara (1600 km south of Tunis/Carthage) show a high point of aridity in the early AD 250s (Chapter 1, Fig. 9), as does a proxy from the Ethiopian Highlands (a possible indicator for Nile flood quality and thus food supply at Alexandria).³⁶⁷ These arid events mentioned by the bishops of the two Mediterranean coastal African cities could be the same environmental event, and may indicate the date of the pestilence.

Since Cyprian portrays the drought/famine in Carthage as current, Dionysius describes Alexandria's famine as having happened just before the pestilence began, and since Eusebius notes in his *Chronicon* that the pestilence arrived in Alexandria in 253, it is thus probable that letter 7.22 was written in AD 253, and the famine happened in 252, around the same time as the famine in Carthage. The persecution that Dionysius says happened before it is probably Decius', and the war he mentions that occurred simultaneously with famine could be fighting that resulted from the short persecution by Gallus in 252 (Table 5).³⁶⁸

It then becomes most likely that the letter in book 7.21 was written in the later AD 250s, since letter 7.22 probably depicts the start of the Plague of Cyprian in Alexandria in 253 and the 7.21 letter makes note of "constant plagues", indicating recurrences of the third-century pestilence. With this in mind, it is most likely, since these are festal letters and there were no persecutions from 252 until the summer of 257, that letter 7.21 was written at Easter in AD 258, as persecutions and fighting, like

³⁶⁷ Nash *et al.* 2016, 6.

³⁶⁸ Frend 1970, 94.

that described in 7.21, would have occurred following Valerian's first edict/letter in August of AD 257, and Dionysius was still in town before his exile by deputy prefect of Egypt, Aemilianus, in 258.³⁶⁹ Exactly when Dionysius is exiled in 258 is unknown, but it was probably before the second edict (Summer 258), since the first edict was a "bloodless" persecution, only requiring Christians unwilling to sacrifice to be exiled, while the second edict/letter declared that bishops, priests, and deacons were to be put to death immediately.³⁷⁰ Thus, it is most likely that Dionysius' letter in Eusebius' *Ecclesiastical History* book 7.21 was written at Easter in AD 258, and the letter in book 7.22 was written five years earlier at Easter in AD 253.

With the chronology cleared up, Dionysius has plenty to offer in these two letters about our epidemic. He addresses the first letter to the Egyptian bishop Hierax, detailing the bloodshed he has witnessed in all parts of Alexandria, but also mentions events that he implies are divinely related, such as how Alexandria's "calm and wave-less harbours have become an image of the sea", where "Egyptians were drowned", and that "the river [Nile] that flows on past the city at one time appeared drier than the waterless desert", while "at another time it overflowed to such an extent that it submerged the whole neighbourhood."³⁷¹ He goes on to talk more about the conflicts and death amongst men, describing a period when Alexandria is riddled with death, emphasizing civil war brought on by persecution, but also including the third-century pestilence as a factor contributing to city-wide mortality in the 250s.³⁷² Since Dionysius was a prominent figure in the church, his discourse

³⁶⁹ Frend 1965, 423.

³⁷⁰ For dating the edicts/letters see Keresztes 1975, 83-84; for the details about the edict see Cyprian, *Epistles* 80.

³⁷¹ Eusebius, *Ecclesiastical History*, 7.21.4-6.

³⁷² Eusebius, *Ecclesiastical History*, 7.21.9.

could be viewed as spiritually-charged.³⁷³ But he is not just casually mentioning that disease occurred simultaneously with persecution. The detail he employs to describe the magnitude of loss at Alexandria transcends fabrication, qualitatively speaking. That he estimates demographic loss based on the public food ration indicates an Alexandrian encircled by the epidemic, an experience he shared with fellow citizens who probably made use of the food ration. To concoct here would inevitably blacken his Christian credibility. He cites data to convey validity and the scale of loss witnessed; to give the pestilence a detailed shape. Whether his estimates are accurate or not, is uncertain – it is, of course, not uncommon for ancient writers to exaggerate numbers – but it is less common to invent a pestilence out of thin air.³⁷⁴ Nevertheless, that Dionysius cites anything at all, and ties it to the food ration, a public service in Alexandria of which the details were probably accessible, points less to an allegorical anecdote and more to an account grounded in reality.

The second letter Dionysius addresses to his Christian “brethren”, in which he relates the disease’s harsh nature and contagiousness, as well as its ubiquity throughout Alexandria. His descriptions make apparent the scale of mortality. Near the letter’s start, Dionysius proclaims, “indeed all is lamentation, and all men mourn...for there is not a house where there is not one dead”.³⁷⁵ Here the indiscriminate nature of the pestilence is clear, as Harper points out.³⁷⁶ That the epidemic is unselective further attests to the veracity of Dionysius’ account – a religiously-biased portrayal would favour the Christians. Dionysius does at times

³⁷³ Strobel views these references to pestilence as embellishments: Strobel 1993, 196; Harper 2015, 227.

³⁷⁴ I will not here go into detail on demographic-loss estimates based on these numbers, but for a realistic calculation see: Parkin 1992, 62-64; Harper 2015, 228.

³⁷⁵ Eusebius, *Ecclesiastical History*, 7.22.

³⁷⁶ Harper 2015, 244.

vaguely intimate that Christians have a slight advantage against the disease – e.g. “For indeed it did not leave us untouched, although it attacked the heathen with great strength” – but never does he propose Christians were not at risk, which would have been a falsehood harshly refuted by his followers.

The disease’s person-to-person transmissibility is also clear in Dionysius’ narrative. He claims that Alexandrians were, “visiting the sick without a thought to the danger...being infected with the disease from others”.³⁷⁷ He goes on to contrast Christian behaviour amidst the pestilence with that of the heathens. He claims Christians “cared for and restored health to others...transferring their death to themselves”. Heathens were the “exact opposite”:

*“Even those who were in the first stages of disease they thrust away, and fled from their dearest. They would even cast them in the roads half-dead, and treat the unburied corpses as vile refuse, in their attempts to avoid the spreading and contagion of the death-plague.”*³⁷⁸

This again showcases a difference in moral values, but, for our purposes, the disease’s indifference and contagiousness.

In the second letter, the epidemic *does* predominantly serve as a device for comparison between Christians and everyone else, but through the slights we glean information about the disease. Most importantly: (1) transmission of the disease most likely resulted from contact, person-to-person or via clothing or objects; and (2) the epidemic was widespread across a large urban centre, for there were no houses “where there is not one dead”. The latter point is, of course, an exaggeration – he

³⁷⁷ Eusebius, *Ecclesiastical History*, 7.22.

³⁷⁸ Eusebius, *Ecclesiastical History*, 7.22.10.

could not know the situation in every house of Alexandria – but his point is taken; it was affecting a majority of the city. Thus, our second source for the Plague of Cyprian, Dionysius of Alexandria reports a deadly, communicable infectious disease disrupting life in the city of half a million people in the AD 250s and early 260s.

Thirteenth Sibylline Oracle

The Sibylline oracles were composed to appear as prophetic declarations, compiled in fourteen books spanning from the life of Christ to the Arab conquest.³⁷⁹ Of course, most of the events (at least the factual ones) described were witnessed before the writing of each book. The work does not read like a history, but the historical events mentioned within it are accurate; that is, the wars, political tensions, and epidemics depicted from that time. The *Thirteenth Sibylline Oracle* is a history of the Roman Empire from Augustus to the rise of the Palmyrene king Odaenathus as ruler of the East (AD 260).³⁸⁰ David Potter claims the work was written by a Syrian citizen (along with book 12), and represents the “sole surviving contemporary narrative account of the years AD 240 – 260 from within the Roman Empire ... also one of the rare pictures that we have of Roman history in any period from the pen of a provincial who had no obvious connection with the government.”³⁸¹ Thus, the work considers nearly a decade of our focus period, from an eastern perspective, and we can view it as fairly unbiased. Potter dates the work to 253, because the book makes no mention of the eastern Roman usurper Uranius Antoninus who was put down by Valerian in

³⁷⁹ Potter 1990, 96-101.

³⁸⁰ Potter 1990, 101.

³⁸¹ Potter 1990, vii.

254.³⁸² However, Potter believes a second writer/compiler wrote the last 19 lines, which include many events from the span of 253 – 260 in a small space, compared to the “thorough treatment” of the years before it.

At the eighth line of the oracle, the author proclaims, “for there will be many wars and battles and killings of men and famines and plagues and terrible earthquakes and lightning strikes and lightning bolts throughout the entire world.”³⁸³ The mention of pestilence does not tell us much, other than that our plague may have reached the East (probably Syria) by 253. Ninety lines later, the author says, “When another king of Rome will rule, then ruinous Ares with his bastard son will bring the disorderly races against the Romans, against the walls of Rome. And then suddenly there will be famine, plague, dreadful lightning bolts, horrible wars and the destruction of cities.”³⁸⁴ Both passages seem to be referring to the same point in time, from the rise of Trebonianus Gallus (“another king of Rome”). This hypothesis would match with Potter’s dating based on the absence of Uranius Antoninus in the narrative; and he too thinks the author is referring to Gallus.³⁸⁵ It is the most plausible theory, and demonstrates that the oracle depicts the period from his rise in late-251 until his death in the summer of AD 253.

While these mentions of plague are fleeting, they do locate *a* pestilence in the East after AD 251, more likely 252, around the same time as Cyprian’s account and slightly earlier than Dionysius’. It is more difficult to trust this work, compared to

³⁸² Potter 1990, 151.

³⁸³ Potter 1990, 167.

³⁸⁴ Potter 1990, 173.

³⁸⁵ Potter 1990, 285 and 315: While it appears Potter correctly identifies the “king of Rome” to be Gallus, he seems to be misreading the sources. He thinks the author’s mention of “Ares and his bastard son” to be referring to Gallus and his son Volusianus as those that “will [inadvertently] bring disorderly races against the Romans”. But the author seems to just be talking about war and terror personified through the mythological “Ares and his bastard son”; Andrew Wilson pers. comm.

Cyprian and Dionysius, as the source presents itself as a prophecy. While much of the narrative coheres with confirmed historical events, based on the allegorical nature of the work, it could be argued that the author exaggerated, or altogether fabricated the mentions of famine and plague. Potter points out that “such lists of catastrophes are a common feature of Sibylline prophecies”, citing many examples, but not commenting on whether he thinks the examples themselves are fabricated.³⁸⁶ However, that these mentions originate at the same time as the more trustworthy accounts of Cyprian and Dionysius – to the year for Cyprian’s account – is, I think, telling. The account comes from a part of the Empire not very near Alexandria or Carthage, and Potter notes of the author that the concise way he treats certain major events elsewhere in the Roman world reflects his “fundamental lack of interests in matters beyond his immediate area.”³⁸⁷ If the author seems unconcerned with events beyond Syria, one could argue he might lack knowledge of events outside of Syria; his muddling of the succession and names of emperors showcases that.³⁸⁸ If that is the case, he might not have been aware of the pestilence raging in Alexandria and Carthage at the same time as his account, potentially giving his mentions of plague more weight. It would be quite a coincidence if the Plague of Cyprian began under Trebonianus Gallus in Carthage and the author of the *Thirteenth Sibylline Oracle* happened to fabricate the same catastrophe at the same time for Syria, declaring that a pestilence came soon after Gallus became emperor.

Despite the frequency of such calamities in prophecies, the timing of the references to plague suggests they are factual. Therefore, with the accounts from

³⁸⁶ Potter 1990, 187; Harper 2015, 231.

³⁸⁷ Potter 1990, 282.

³⁸⁸ Potter 1990, 285 and 315.

Cyprian and Dionysius, the *Thirteenth Sibylline Oracle* helps situate the extent of the third-century pestilence from Africa to Syria by AD 253 at the latest.

Pontius of Carthage

Pontius of Carthage (lived early-to-late third century AD) comes next in the contemporary chronology for the Plague of Cyprian. Pontius, a deacon in Carthage under Cyprian, and his biographer, describes catastrophic loss at Carthage caused by the third-century epidemic, confirming the accounts of Cyprian.³⁸⁹ After discussing Cyprian's flight from Carthage during the Decian persecution in book 8 of his *Vita Cypriani*, in book 9, Pontius explains: "Excessive destruction of a hateful disease invaded every house in succession of the trembling populace, carrying off day by day with abrupt attack numberless people, everyone from his own house."³⁹⁰ The text, written in late 258, gives an account similar to Dionysius' for Alexandria, as Pontius tells how not one Carthaginian household was spared by the disease, as well as implying substantial population loss.³⁹¹ Like both Cyprian's and Dionysius' accounts, there is no reason to believe Pontius was fabricating or exaggerating to a great degree his description of the pestilence, since the disease would have been well known to his audience. The chronology related in *Vita Cypriani* does not contradict the chronology that we have observed up to this point, as Pontius discusses the Decian persecution (January 250), Cyprian's flight from Carthage (250), Cyprian's experience with the pandemic upon his return (mid 251 – late 252), and his eventual

³⁸⁹ Jerome, *De viris illustribus*, 68: "Pontius, Cyprian's deacon, who up to the day of his martyrdom endured exile with him, wrote a remarkable book of the life and passion of Cyprian."

³⁹⁰ Pontius, *Vita Cypriani*, 9, trans. R. E. Wallis.

³⁹¹ For the work's date see Sage 1975, 393: he thinks *Vita Cypriani* was composed soon after Cyprian's death.

exile to Curubis (257), in that order. However, that Pontius uses the past tense to describe the epidemic suggests that by AD 258 it had subsided in Carthage, and possibly left for good. With that, we can say the plague was probably still affecting Alexandria in 258, while by then the pestilence may have died out in Carthage.

Dexippus and Philostratus: the lost works of two contemporary Athenians and those that draw on them

Dexippus and Philostratus, two contemporary pagan historians from Athens, wrote about our pestilence in works now lost.³⁹² We know about their works from later historians who used the earlier histories for their own. As contemporaries of the Plague of Cyprian, Dexippus and Philostratus are still important to its history, but since their works are not extant, and they are not related word-for-word like Eusebius does for Dionysius, they add less substance to the narrative.

Harper sees the Greek statesman Dexippus as the primary source of history for the period from 238 (post-Herodian) until Eunapius in 270, which seems to echo in the fourth-century *Historia Augusta*, the fifth-century historian Zosimus' *Historia Nova*, and possibly in the writings of the sixth-century diplomat Peter the Patrician.³⁹³ Photios I, the ninth-century saint from Constantinople, discusses three important works by Dexippus: (1) a history of the events after Alexander the Great; (2) a history of the wars between the Goths and the Roman Empire; and (3) the *Chronike Historia*, a twelve-book history covering a thousand years up to AD 270.³⁹⁴ It

³⁹² Harper 2015, 231.

³⁹³ Harper 2015, 231-237.

³⁹⁴ Photius, *Codices*, 82; Harper 2015, 235.

is assumed that the latter work must be the main source informing the aforementioned later historians.

In the book on emperor Gallienus (co-emperor with Valerian 253 – 260; sole emperor 260 – 268) in the notoriously unreliable *Historia Augusta*, the author claims, “Therefore the favour of the gods was sought by consulting the Sibylline Books, and, according to their command, sacrifices were made to Jupiter Salutaris. For so great a pestilence, too, had arisen in both Rome and the cities of Achaea that in one single day five thousand men died of the same disease. While Fortune thus raged, and while here earthquakes, there clefts in the ground, and in divers places pestilence, devastated the Roman world, while Valerian was held in captivity and the provinces of Gaul were, for the most part, beset, while Odaenathus was threatening war, Aureolus pressing hard in Illyricum, and Aemilianus in possession of Egypt...”³⁹⁵ The work notes specifically when the pestilence was occurring: from when Valerian was captured by the Sassanian King Sapor I after the Battle of Edessa (AD 260), while the Palmyrene King Odaenathus was preparing for his first Persian campaign (around AD 262), and when prefect of Egypt Mussius Aemilianus took control of the province and proclaimed himself emperor (AD 261 – 262).³⁹⁶ The most significant piece here is this is one of the only accounts of outbreaks in Rome and in Greece (the province where Dexippus was writing). The *Historia Augusta*’s account does not make clear if 5,000 a day died in Rome, or in each city, or if it is the total between the cities. Nevertheless, if the *Historia Augusta* did indeed borrow from Dexippus, can we be sure Dexippus, living in Athens, knew disease reached Rome? More importantly, can we trust the

³⁹⁵ *Historia Augusta, The Two Gallieni*, 5.5-5.6.

³⁹⁶ Potter 1990, 381-394; for the story on Aemilianus see Ando 2012, 169.

Historia Augusta here? Could this not be another fanciful fabrication by the author of the *HA*, who, in reading Dexippus' accounts of pestilence in the East decided to also place our pestilence in the all-important capital? This is possible; but it is more likely that it did reach the capital, given Rome's maritime connectivity with two cities (Carthage and Alexandria) that we know had massive outbreaks.³⁹⁷ The *Historia Augusta* probably drawing from Dexippus, however, is here not the convincing source (next subsection).

The fifth-century historian Zosimus also seems to be drawing on Dexippus, as pointed out by Harper.³⁹⁸ Zosimus mentions the pestilence on four occasions in his *Historia Nova*, spanning the period from the rise of Gallus (late AD 251) to the death of emperor Claudius Gothicus in AD 270 (who himself might have died from the disease).³⁹⁹ At book 1.26.3, after discussing Gallus' lacklustre attempts to contain the eastern Scythians, Zosimus claims, "With war thus pressing heavily on the empire from all sides, a plague afflicted cities and villages and destroyed whatever was left of mankind: no plague in previous times wrought such destruction of human life."⁴⁰⁰ Later, at book 1.36.3, in discussing events under Valerian (around late AD 258), Zosimus says, "While Sapor conquered every part of the East, a plague struck Valerian's troops, carrying off the majority," after describing Sapor I's capture of Valerian.⁴⁰¹ In the very next section, again speaking of the Scythians in the East, he claims, "Then, when Illyricum was in extreme danger from the Scythians' invasion and the whole Roman Empire was tottering towards destruction, an unprecedented

³⁹⁷ Andrew Wilson, pers. comm.

³⁹⁸ Harper 2015, 235-236.

³⁹⁹ Zosimus, *Historia Nova*, trans. R.T. Ridley, 8-14.

⁴⁰⁰ Zosimus, *Historia Nova*, trans. R.T. Ridley, 8.

⁴⁰¹ Zosimus, *Historia Nova*, trans. R.T. Ridley, 1.36.1.

plague struck the cities.”⁴⁰² The last reference to pestilence also discusses the Scythians, as well barbarians that died of the pestilence while “cruising around” Crete and Rhodes, as well as some barbarians who died in Thrace and Macedonia.⁴⁰³ In that same section, Zosimus explains how Romans and Roman soldiers also died from the pestilence, including the emperor Claudius. Thus, Zosimus’ account (probably taken from Dexippus) places the plague mostly in the East, and not further west than Macedonia. Interestingly, nowhere does Zosimus imply that the Plague of Cyprian reached the Empire’s capital.

We hear much of the same from the sixth-century Justinianic official, Peter the Patrician. In describing the conflicts between Valerian and Sapor I, Peter writes, “Valerian, alarmed at the advance of the Persians (for the plague raged among his army, and especially among the Moorish contingent), gathered an enormous amount of gold and sent ambassadors to Sapor, hoping to end the war by such great gifts. Sapor, having both learned about the plague, and even more roused by the plea of Valerian, turning away the ambassadors, and sending them off with the mission unaccomplished, immediately followed them.”⁴⁰⁴ Here again, a historian gleaned information from Dexippus discusses the pestilence in relation to conflicts in the East. Most of the references using Dexippus deal with Valerian, his troops, and Sapor I, but none so far, besides the untrustworthy *Historia Augusta*, situates the Plague of Cyprian further west than Macedonia. It is possible that the account in the *Historia Augusta* was embellished to include Rome, and that Dexippus’ lost history only mentioned an eastern epidemic occurring amidst war between the reigns of

⁴⁰² Zosimus, *Historia Nova*, trans. R.T. Ridley, 1.37.3.

⁴⁰³ Zosimus, *Historia Nova*, trans. R.T. Ridley, 1.46.1-2.

⁴⁰⁴ Harper 2015, 236.

Gallus and Claudius Gothicus (AD 251 – 270). Otherwise, why would Zosimus (in his four references to the pestilence) and Peter the Patrician, both also drawing from Dexippus, not mention the plague having reached Rome?

Philostratus, the other Greek historian contemporary with the Plague of Cyprian whose works are now lost (aside from one fragment), appears to have been drawn on by five later writers.⁴⁰⁵ The fifth-century Roman priest and historian Orosius, a tenth-century collection of texts copied and compiled in the seventeenth century by Claudius Salmasiana titled the *Excerpta Salmasiana II*, the tenth-century Greek historian Symeon the Logothete, the eleventh-century Byzantine historian Georgios Kedrenos, and the twelfth-century Constantinopolitan John Zonaras all give similar accounts of the third-century pestilence that have been attributed to Philostratus.⁴⁰⁶ All of the accounts borrowing from Philostratus mention a pestilence that lasted for fifteen years, starting at some point after the summer of 251. The main pieces linking Philostratus to the later accounts are: (1) the late-sixth-century historian Evagrius Scholasticus' description of the Justinianic Plague: "So then, as I have said, this misfortune has been prevalent up to the present for 52 years, surpassing all previous ones. For Philostratus is amazed that the plague in his time prevailed for 15 years"; and (2) the Byzantine chronicler George Syncellus' claim that an Athenian historian called Philostratus existed in the early 270s during Aurelian's reign.⁴⁰⁷ Orosius, the *Excerpta Salmasiana II*, Symeon the Logothete, George Kedrenos, and John Zonaras, all provide a brief entry about a mid-third-century

⁴⁰⁵ Harper 2015, 231-237.

⁴⁰⁶ Harper 2015, 231-234.

⁴⁰⁷ Harper 2015, 231; Evagrius Scholasticus, *Ecclesiastical History*, 4.29, trans. M. Whitby; first pointed out by Jones 2011, 321.

pestilence lasting for 15 years, each accompanied by varying details. All describe the epidemic moving from east to west, with the *Excerpta Salmasiana II* and Zonaras asserting it started in Ethiopia, and Symeon the Logothete and the *Excerpta Salmasiana II* claiming the disease was transmitted through clothing or “simply by sight”.⁴⁰⁸ Orosius’ accounts are the only ones to offer more than that above.

However, his descriptions seem exaggerated and fantastical. This is probably a result of Orosius’ position as priest, theologian, and ecclesiastical historian contending with portrayals of the continual persecutions on the Christian community from Decius to Gallienus – an agenda which Harper fails to discuss.⁴⁰⁹ In his quotation of Orosius, Harper leaves out an important previous line:

*“Vengeance for the defiling of Christians now made itself manifest. And wherever Decius’s edict to cast down churches had reached, there fell a plague of inconceivable ills. There was hardly a province of the Roman Empire, a city, or a home that was not seized and emptied of its inhabitants by this universal plague.”*⁴¹⁰

Harper, starting with “Decius’s edict”, overlooks the fact that this excerpt might carry biased weight. Of course, from the second sentence alone one might sense Orosius’ agenda, but with the omission – erasing a previous sentence that starts the section – Harper seems to be diverting attention away from Orosius’ partiality. Neither does Harper broach the general subject of the work: a “history against the Pagans”. Just from the excerpt above it is clear Orosius believes the persecutor

⁴⁰⁸ Orosius, *Seven Books of History Against the Pagans*, 7.21.5-6, 7.22.1, 7.27.10; for *Excerpta Salmasiana II* see Roberto ed. 2005, 154; for George Kedrenos’ mention see Wahlgren, 77; Zonaras, *Extracts of History*, 12.21, trans. T. Bancich and E. Lane.

⁴⁰⁹ Harper 2015, 233-234.

⁴¹⁰ Orosius, *Seven Books of History Against the Pagans*, 7.21.5-6, trans. A.T. Fear.

(Decius) was responsible for the pestilence, as he works to inflate its impact. The account resembles no other account of our plague, besides its chronology. Five books later, Orosius says, making a comparison with the Biblical plagues of Egypt:

“There, the plague ranked as the seventh was the hail that condensed out of the air to such degree that it could kill men, animals and crops; here, a similar seventh plague happened in the reign of Gallus and Volusianus who soon succeeded the persecutor Decius on his death. Disease came forth from the corrupted air and poured over all the bounds of Rome’s kingdom from east to west, killing almost the whole race of men and beasts.”⁴¹¹

Like other accounts, this one notes the pestilence starting after Decius, during the reign of Gallus and Volusianus (post-summer 251). He mentions a disease moving east to west, probably taken from Philostratus, but he also claims it affected all parts of the Empire, an obvious exaggeration if he is borrowing from Philostratus, as none of the other sources that draw from Philostratus suggest anything of the sort. Additionally, he mentions that the pestilence affected livestock too. It is not impossible that the disease responsible for the Plague of Cyprian was zoonotic (capable of affecting animals and humans), but Orosius is the only source to claim this. If true, it is strange that the Christians Cyprian and Dionysius left out this crucial characteristic, especially since infected animals are a common feature in religiously-charged portrayals of plague to depict utmost destruction in times of persecution. If this was happening, it is surprising that Cyprian and Dionysius did not mention it. The timing related by Orosius for the Plague of Cyprian matches up

⁴¹¹ Orosius, *Seven Books of History against the Pagans*, 7.27.10, trans. A.T. Fear: “There” refers to Egypt and the ten terrible “plagues” suffered there, which in book 7.27 he compares with Roman distresses such as the Antonine Plague (7.27.7) and this quote that refers to the Plague of Cyprian.

with our other ancient sources, but much else appears to be exaggeration by a priest with a theological agenda.

The “Kaisergeschichte”, Aurelius Victor, and Eutropius

Harper points out that the late-antique writers Aurelius Victor and Eutropius both mention the plague, and that they probably borrowed from a now lost fourth-century Latin text, as postulated by Alexander Enmann in 1884, referred to as the *Kaisergeschichte*, which was a source not only for Aurelius Victor and Eutropius, but also for the *SHA*, Jerome, and Ammianus Marcellinus.⁴¹² Recently, George Woudhuysen and Justin Stover have argued that the so-called *Kaisergeschichte* was in fact the lost long-form history of the fourth-century historian Aurelius Victor, which has survived only in epitomies by others, as the *Epitome de Caesaribus* and the *Liber de Caesaribus* – two different abridgings of Aurelius Victor’s full work.⁴¹³ In any event, for the purposes of this chapter, we focus on the mentions of plague by Victor and Eutropius.

The references by Victor to plague are more important than Eutropius’ brief mention amidst his discussion of the reign of Gallus and Volusianus: ‘their reign was notable only for the plague, diseases and other afflictions.’⁴¹⁴ Victor’s references in fact help place the plague in Rome, which may legitimise the reference in the *Historia Augusta*, which seemed at first to have been borrowing from Dexippus; Enmann

⁴¹² Harper 2015, 240; Enmann 1884, 338-510.

⁴¹³ Andrew Wilson, pers. comm. citing G. Woudhuysen and J. A. Stover (2015). ‘The lost *Historia* of Sextus Aurelius Victor’, paper for Late Roman Seminar, Corpus Christi College Oxford, 5 November 2015.

⁴¹⁴ Eutropius, *Breviarium Historiae Romanae*, 9.5, trans. by H.W. Bird; Harper 2015, 240.

demonstrated that the lost *Kaisergeschichte* was an important source for the *Historia Augusta*. The *Epitome de Caesaribus* says:

*"Vibius Gallus, with Volusianus, his son, ruled two years. In their time, Hostilianus Perperenna was made emperor by the senate and, not much later, was consumed by the plague."*⁴¹⁵

The *Liber de Caesaribus* writes:

*"When the senators had learned of this they voted the rank of Augustus to Gallus and Hostilianus and appointed Volusianus, the son of Gallus, as Caesar. Thereafter a plague broke out and while it raged ever more violently Hostilianus died but Gallus and Volusianus won popular favour because they meticulously and assiduously arranged the burials of all the poorest folk."*⁴¹⁶

And later in the same work:

*"And at the same time the plague was ravaging Rome which often occurs in times of unbearable anxieties and spiritual despair."*⁴¹⁷

The references to plague in the epitomies of Victor indicate that the pandemic hit the capital. The reference to Hostilianus dying of plague seems to demonstrate that, as there is no mention in the sources of Hostilianus being out on campaign, which means that after he was appointed co-emperor by Gallus, he most likely contracted the disease while presiding over Rome. That Gallus and Volusianus "meticulously and assiduously manage burials for the poorest folk" also suggests that the disease was in Rome by the early 250s. If this is correct, it might indicate that the *Historia*

⁴¹⁵ Aurelius Victor, *Epitome de Caesaribus*, 30, trans. by H.W. Bird.

⁴¹⁶ Aurelius Victor, *Liber de Caesaribus*, 30, trans. by H.W. Bird.

⁴¹⁷ Aurelius Victor, *Liber de Caesaribus*, 33.5, trans. by H.W. Bird.

Augusta was borrowing from Aurelius Victor and not from Dexippus (since none of the other authors who we think borrow from Dexippus mention Rome); although we still do not know where the *SHA* learned that “5,000 died per day”, whether the work was referring to the capital or Greece, or just fabricating for effect. Nevertheless, the accounts from Aurelius Victor appear to put the pandemic in Rome in the early 250s.

P. Oxy 14.1666

The *Oxyrhynchus Papyri* (*P. Oxy*) are a series of papyri found in the urban rubbish dump of ancient Oxyrhynchus, Egypt. Uncovered by archaeologists in the late nineteenth and early twentieth centuries, approximately ten percent of the documents and literary works have been examined and translated. As mentioned in chapter 2, amongst the manuscripts is a letter written by one Pausanius to a man who is seemingly his brother. The letter mainly concerns the writer chronicling his attempts to transfer his son’s military service from that of a legionary soldier to a cavalryman.⁴¹⁸ At the end of the letter the author mentions hearing about plague reaching Antinoöpolis (Chapter 2, Fig. 30).⁴¹⁹ Pausanius appeals: “I urge you, brother, to write to me about your safety, since I heard at Antinoöpolis that there has been plague in your neighbourhood. Do not therefore neglect this, that I may rest more assured about you.”⁴²⁰

⁴¹⁸ *P. Oxy* XIV.1666; Grenfell and Hunt 1914, 121-123.

⁴¹⁹ *P. Oxy* XVI.1666, 19-22.

⁴²⁰ Grenfell and Hunt (trans. by) 1914, 122.

Pausanius' letter has been dated to the third century AD by the papyrologists.⁴²¹ However, there is no indication by the editors of the papyrus which part of the century it dates to. If the papyrus was written very early on in the third century AD it is not impossible that the pestilence Pausanius heard about was the Antonine Plague. However, since there is no evidence of the Antonine Plague after AD 190, that is unlikely. It is probable that the account refers to the mid-third-century Plague of Cyprian. This likelihood may be strengthened by the skeletal evidence possibly connected to the Plague of Cyprian from Thebes, also in the Nile Valley, 450km south of Antinoöpolis (below).⁴²² It is not certain if the neighbourhood of the person whom the letter was meant for was close to Antinoöpolis, but it is probable.

Discussion

This review of the ancient sources suggests that Harper's case for a large-scale pandemic is probably accurate, but, so far, not on par with the Antonine or Justinianic plagues. He argues that it started earlier than in fact it probably did – in AD 249 (or earlier), based on Strobel's dating of Dionysius of Alexandria's letters; but, after careful review, it appears that the letters should be dated to the 250s, and that the written evidence indicates a start date for the Plague of Cyprian between late 251 and the end of 252. Harper also thinks the pestilence affected all parts of the Empire, but the literary evidence does not demonstrate that. It indicates that Carthage, Alexandria, Syria, Greece, Rome, and possibly the Nile Valley had

⁴²¹ Grenfell and Hunt 1914, 121-123.

⁴²² Tiradritti 2014, 15-18: evidence of a mass grave in ancient Thebes (Egypt) dated to the period of the Cyprianic Plague.

outbreaks, while so far we have no evidence that puts the pestilence in Gaul, Spain, Britain (although our sources are limited for the northwest), or Asia Minor.

Therefore, after thorough inspection of the literary evidence, although we might conclude that the Plague of Cyprian probably had an important effect on the Empire, we should be cautious in claiming that it was a devastating, Empire-wide epidemic.

In the next section we will see if archaeology changes the picture.

The Archaeological Evidence

Unfortunately, there are not many pieces of archaeological evidence that are plausibly connected to the Plague of Cyprian. I present two potential pieces of evidence below.

The “body-disposal unit” at Thebes, Egypt

Remains of a so-called “body-disposal unit” were found near the west bank of the ancient site of Thebes in Egypt, at the funerary complex of Harwa and Akhimenru.⁴²³

Archaeologist Francesco Tiradritti published on the find in 2014, noting that three kilns were found where lime was produced to seal bodies, as well as a large bonfire nearby, where bodies were incinerated (figures 45 and 46). The material finds (mainly ceramics) associated with the lime kiln and bonfire date to the third or early fourth centuries. However, because the majority of finds date to the third century, Tiradritti dated its main phase of operation to the second half of the third century.

⁴²³ Tiradritti 1998, 3-7; Tiradritti 2014, 15-18.

With that said, analysis of the evidence by Tiradritti was brief and published in the magazine *Egyptian Archaeology* in 2014, having yet to receive full-scale treatment in an academic journal. In their critique of *The Fate of Rome*, John Haldon and colleagues take issue with Harper linking the evidence at Thebes to the Plague of Cyprian. They explain that the lime produced at the site was not necessarily to cover victims of a pandemic; indeed there is plenty of evidence that lime was used for burials that did not involve disease.⁴²⁴ Haldon and company also note that the small number of bodies found hardly constitutes a mass grave. However, that does not necessarily mean the incinerated bodies at Thebes are not linked with the Plague of Cyprian. The dating broadly aligns, and the plague *was* raging in the province at Alexandria (Dionysius) in the mid-third century. Additionally, as just mentioned, the papyrus letter *P. Oxy 14.1666*, dated to the third century AD, mentions pestilence at Antinoöpolis, 450 km along the Nile north of Thebes.



Figure 45. Skeletal remains of bodies incinerated in a bonfire at a funerary complex of Harwa and Akhimenru. (Tiradritti 2014)

⁴²⁴ Reece 1977; Philpott 1991; Schotsmans *et al.* 2015; Haldon *et al.* 2018b, 6.



Figure 46. The remains of three lime kilns used to produce lime for burials at a funerary complex of Harwa and Akhimenru. (Tiradritti 2014)

Rooms X80, X81, X83, X84 of the Catacombs of Marcellinus and Peter, Rome

The next piece of archaeological evidence potentially connected to the Plague of Cyprian is not definitive either. As discussed in the previous chapter, multi-burial tombs containing more than 1300 individuals were found at the catacombs of Marcellinus and Peter in the early 2000s; however, only rooms/tombs X80/T16, X82/T18, and X78/T15 have been fully excavated, while just the upper respective layers of rooms X83 and X84 were excavated, and rooms X80 and X81 were not excavated at all aside from two small soundings in X81.⁴²⁵ The investigating archaeologists hypothesise a connection to the Antonine Plague for the fully excavated rooms, but do not offer an opinion about later rooms X80, X81, X83, or X84 with so little of the excavation complete.⁴²⁶ As mentioned, Harper thinks the burials

⁴²⁵ Blanchard *et al.* 2007, 989-998.

⁴²⁶ Blanchard *et al.* 2015, 204; Blanchard *et al.* 2007, 996; Philippe Blanchard, personal communication: based on my conversation with Blanchard, it appears excavation of these rooms will not continue any time soon.

in the later rooms are victims of the Plague of Cyprian, which is plausible based on the coin of emperor Gordian (found during minimal excavation), which establishes a *terminus post quem* of AD 238, as well as the radiocarbon date of a human bone that returned a chronological range of AD 140 – 380 at 2 sigma (Chapter 2, pp. 122-128); but without further excavation we cannot know that an epidemic was the cause of death. If it does prove to be associated with the Plague of Cyprian, this evidence would cohere with the references in the *Historia Augusta* and in the epitomes of Aurelius Victor that the pandemic hit Rome, but as Blanchard and his colleagues say, it is too early to connect it to the third-century pandemic.

Numismatic Evidence

Numismatic evidence from the latter half of the third century does suggest that an epidemic was affecting the empire, enough to necessitate a change of imagery on imperial coinage. From the reign of Trebonianus Gallus (late 251) until Gallienus (268), the legend APOLL SALVTARI featured on the reverse of the silver Antoninianus (figures 47 and 48).⁴²⁷ In the third century we see the usage of APOLL SALVTARI only during the period of the pandemic, with the most examples during the reign of Trebonianus Gallus, i.e. the emperor under whom I argue (above) the pandemic began (Fig. 49). Erika Manders says that the appearance of Apollo, healer and protector against evil under the gods, on imperial coinage of the later third century might indicate a response to pestilence.⁴²⁸ This theory aligns with

⁴²⁷ APOLL SALVTARI was also featured on the *aureii* and *sestertii* of Gallus, and the *sestertii* of Volusian. Manders 2012, 131-32; *RIC* IV.3: Trebonianus Gallus numbers 5, 19, 32, 103, and 104a-b; *RIC* IV.3: Volusianus numbers 188, 247, 248a-b; *RIC* IV.3: Aemelianus number 27; *RIC* V.1, Valerian, number 76.

⁴²⁸ Manders 2012, 131-32.



Figure 47. An Antoninianus of Trebonianus Gallus (post-251) with APOLL SALVTARI on the reverse.
(numismatics.org/ocre : RIC IV Trebonianus Gallus 19)



Figure 48. A Sestertius of Volusian (AD 251-253) with APOLL SALVTARI on the reverse.
(numismatics.org/ocre : RIC IV Volusian 247)

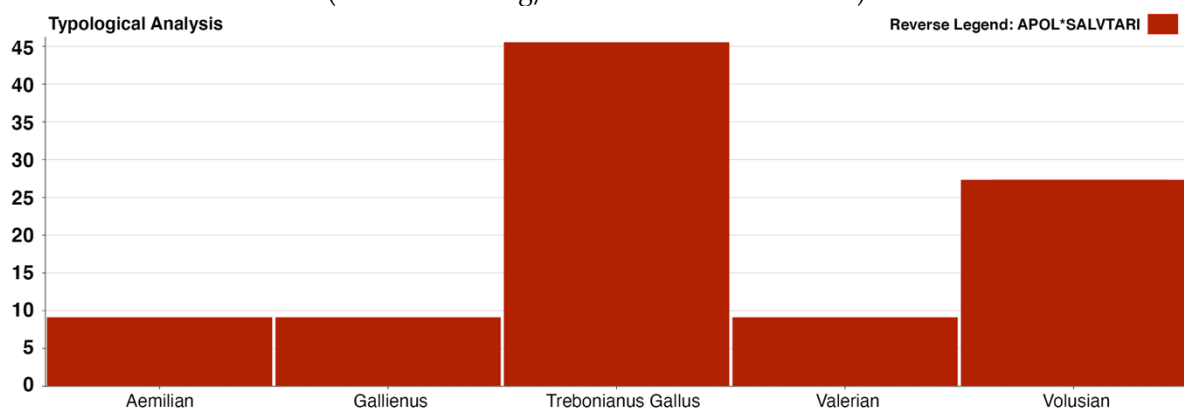


Figure 49. The collections of coins with APOL SALVTARI or APOLL SALVTARI from the Online Coins of the Roman Empire database, all from the period of the Plague of Cyprian. Note that the category with most (45) are coins with emperor Trebonianus Gallus (AD 251-253) on the obverse.
(numismatics.org/ocre)

the chronology for our plague, as derived from the literary evidence. Harper thinks this is evidence of the third-century pestilence in Rome and its large-scale effects throughout the Empire. It does seem to support the notion that the pestilence struck Rome, since the epithet “salutaris”, accompanying Apollo on coinage of the AD 250s, denotes “health” or “healthful”; and it coheres with the written accounts from Aurelius Victor – the death of Hostilianus, and Gallus’ and Volusianus arranging burials for the poor – which appear to be evidence of the pandemic in Rome. With the pestilence killing an emperor in Rome, and the emperors “meticulously and assiduously” managing plague burials in Rome, one can imagine a commission to change the imperial coinage to improve morale in the capital. However, it is a stretch to say the coinage is indicative of the pandemic’s large-scale effects across the Empire.

Discussion

It may be that the archaeological evidence from Thebes is connected to the Plague of Cyprian – it would indeed be consistent with the written evidence from Egypt – but with only three skulls visible in the published photo of the site, and no full excavation report, currently it indicates nothing other than a few deaths around the time of the third-century pandemic. The evidence from the Catacombs of Marcellinus and Peter is also suggestive, yet far from definitive. The fully excavated rooms seem to date to the second century AD and, perhaps, may be connected to the Antonine Plague. The evidence from the partially excavated rooms point to a third-century date; but, at this stage, the cause of mortality is unclear; however, it is not implausible that the burials are victims of the Plague of Cyprian. The numismatic

evidence is probably indicative of our pandemic. Just as the Plague of Cyprian was devastating areas of the Empire, the emperors began minting APOLL SALVTARI on coins – Apollo the protector against evil, and the epithet “salutaris” meaning “healthful”. I think it would be quite a coincidence if this minting decision was not related to the Plague of Cyprian. The coins do seem to support the idea that the pandemic was having an important effect on the Empire, and probably Rome itself; but I think proposing that they demonstrate empire-wide mortality, as Harper suggests, is an exaggeration.

Symptomology, Epidemiology, and Diagnosis

Most of what we know about symptomology for the Plague of Cyprian comes from Cyprian himself. Harper’s 2015 article pulls this information together for us in an orderly and, I think, accurate way. There is not much need to rehash his work in detail, but let us go over the symptoms to confirm, and slightly sharpen, his diagnosis.⁴²⁹

Symptomology and epidemiology

Cyprian describes a severe disease with violent diarrhoea, high fever, pain and bleeding in the eyes which sometimes leads to blindness, decaying/rotting limbs, continual vomiting, debilitating weakness, and in some cases hearing loss.⁴³⁰ In addition to Cyprian’s symptomology, our other ancient sources depict epidemiological characteristics of the disease, as pointed out by Harper.⁴³¹ First, the

⁴²⁹ Harper 2015, 241-248.

⁴³⁰ All of these symptoms are described in books 8 and 14 of Cyprian’s *De mortalitate*; Harper 2015, 241-242.

⁴³¹ Harper 2015, 243-245.

sources who draw from Philostratus – Orosius, the *Excerpta Salmasiana II*, Symeon Logothete, Georgios Kedrenos, and John Zonaras – all describe a disease moving east to west, with the *Excerpta Salmasiana II* and Zonaras claiming it started in Ethiopia; however, we must keep in mind, as we saw in the last chapter, that pestilence coming from Ethiopia was a literary topos of the time, going back to Thucydides’ description of the Plague of Athens. Harper believes the disease moved fast, stating that “It seems incontestable that a pandemic disease event erupted by Easter of 249 in Egypt and quickly spread across the empire, reaching Rome by the second half of 251 at the latest and recurring for the next two decades.”⁴³² However, above we reviewed how it *is* contestable that the disease started in Egypt in 249. A 251 start-date would indicate that the disease diffused even faster than Harper suggested, spreading from North Africa to Rome in the same year. Based on the literary evidence mentioned above, we know that the disease was in Carthage in late-251/early-252 (Cyprian), it was in the East by the early 250s (Thirteenth Sibylline Oracle), Alexandria probably by 253 (Dionysius via Eusebius), near Macedonia and Illyricum in the late-250s/early-260s (Dexippus via Zosimus), and in Rome probably by 251 (Aurelius Victor). Harper’s deduction that the epidemic was indiscriminate does appear to be accurate; multiple sources, most notably Dionysius, describe a disease capable of infecting all people, young or old, rich or poor, Christian or pagan.⁴³³ However, Harper’s claim that the disease had a seasonal nature is not a strong one. He claims, “sources that seem to derive from Philostratus report the seasonal cycle of the disease. It began in autumn and waned in

⁴³² Harper 2015, 241.

⁴³³ Harper 2015, 244.

summer.”⁴³⁴ The “sources” he speaks of is, in actuality, one source; Symeon the Logothete mentions a fifteen-year pestilence, each year “starting in the autumn and abating at the rising of the Dog Star”.⁴³⁵ Symeon’s claim could be truthful, but that this detail is not mentioned by the four other writers borrowing from Philostratus leads one to question its veracity.

However, it is indisputable that wherever there were outbreaks of the disease, the mortality rate was high. Even considering exaggeration, such as perhaps the *HA*’s claim that 5,000 died in a single day, or Dionysius’ claim that over half of Alexandria’s population perished (although, this is probably closer to the truth since it is based on the public annona records), all sources relate a disease that killed many when it struck. We also glean from the literary sources that the pestilence was highly transmissible. Dionysius explains how Christians were considered brave to help the sick or handle dead bodies, and two historians drawing from Philostratus say the plague transmits through clothing or simply by sight, implying that the disease was extremely contagious.

Diagnosis

Harper thinks that the disease responsible for the Plague of Cyprian is a form of Viral Haemorrhagic Fever (VHF), which, with the evidence we currently have, I think is the correct diagnosis.⁴³⁶ He rightly eliminates smallpox, measles, and bubonic plague, given the lack of description by the literary sources of the characteristic rash brought on by the two former diseases – additionally, measles did

⁴³⁴ Harper 2015, 244.

⁴³⁵ Wahlgren 2006, 77; Harper 2015, 244.

⁴³⁶ Harper 2015, 246-248.

not yet exist – and the distinctive buboes of the latter.⁴³⁷ He also eliminates cholera (the disease was confined to India until 1917) and epidemic typhus (different symptomology).⁴³⁸ He does not explore typhoid fever as an option, but we can eliminate it based on dissimilar gastrointestinal symptoms. Harper moves on to propose a VHF as the possible cause, which, based on the symptomology, appears to be right. However, he does not choose a particular form of viral haemorrhagic fever as the culprit.

In 2018, Amber Kearns proposed Ebolavirus as the best fit for the disease responsible for the Plague of Cyprian in a Master's thesis. Using the ancient symptomology related by Cyprian, Kearns employs differential diagnosis to home in on Ebolavirus as the probable culprit.⁴³⁹ Her argument is compelling, as the ancient symptomology is a near match for modern symptoms of the disease caused by the virus: Ebola Virus Disease (EVD). To reiterate its symptoms, the third-century pandemic was a severe disease, causing violent diarrhoea, high fever, pain and bleeding in the eyes that sometimes led to blindness, decaying/rotting limbs, continual vomiting, debilitating weakness, and in some cases hearing loss. As listed by the Center for Disease Control and Prevention (CDC), the symptoms of the Ebola Virus Disease include: "high fever; aches and pains, such as severe headache, muscle and joint pain [rotting limbs?]; weakness and fatigue; gastrointestinal symptoms including diarrhea and vomiting; abdominal pain (stomach) pain; unexplained haemorrhaging, bleeding or bruising; other symptoms may include red eyes, skin

⁴³⁷ On measles: Furuse, Suzuki and Oshitani 2010, 52.

⁴³⁸ Harper 2015, 246.

⁴³⁹ Kearns 2018.

rash, and hiccups (late stage)".⁴⁴⁰ In addition, Ebolavirus is transmitted mainly via bodily fluids, matching the literary sources that describe the third-century pestilence transferring through clothing and handling the sick or dead.⁴⁴¹ Also, some modern survivors of EVD experience hearing loss. Lastly, Dionysius and Cyprian say that one could become infected by handling dead bodies, which is not common of the other diseases, but is a characteristic of Ebola; it was suggested in 2015 that historically lime may have been used in burials involving Ebola, since the disease can remain active in the dead for "several days" after one perishes; if it is found that the so-called "body-disposal unit" at Thebes, Egypt, is in fact connected to our pandemic, this may explain the "unit's" lime kiln.⁴⁴²

The symptomology of other VHF's do not match that of Plague of Cyprian as well as EVD does. For example, Dengue and Yellow Fever have some symptoms in common, but lack gastrointestinal problems, the first and perhaps the most apparent symptom described by Cyprian.⁴⁴³ Lassa fever also has similar symptoms, including hearing loss, but has a low case-mortality rate at 1%.⁴⁴⁴ The Marburg Virus, also a member of the *filoviridae* family, causes Marburg Virus Disease (MVD). It is the next closest match to the Plague of Cyprian's symptomology, and is Ebola's closest ancestor. However, Marburg is missing bloodshot eyes and hearing loss.

⁴⁴⁰ <https://www.cdc.gov/vhf/ebola/symptoms/index.html>, accessed: September 29th, 2020.

⁴⁴¹ <https://www.cdc.gov/vhf/ebola/transmission/index.html>, accessed: September 29th, 2020.

⁴⁴² Dallatomasina *et al.* 2015, 448; Schotsman *et al.* 2015, 465.

⁴⁴³ <https://www.who.int/news-room/fact-sheets/detail/yellow-fever>, accessed: September 29th, 2020.

⁴⁴⁴ <https://www.who.int/en/news-room/fact-sheets/detail/lassa-fever>, accessed: September 29th, 2020.

Genealogical history of Ebolavirus

Before making a diagnosis, we must check the genealogical history for Ebolavirus to see whether the disease existed before the third century AD. A molecular evolution (molecular-clock analysis) study by Suzuki and Gojobori in 1997 calculated that Ebolavirus and Marburg Virus diverged somewhere between 7,100 and 7,900 years ago, demonstrating that the earliest ancestor of Ebolavirus existed over five millennia before the Plague of Cyprian began in the early AD 250s.⁴⁴⁵ A more recent and scientifically-up-to-date study (2013) indicated that the Ebolavirus and Marburgvirus had more likely diverged a few millennia earlier, demonstrating a most recent *filoviridae* common ancestor 10,436 years ago (Fig. 50).⁴⁴⁶ It calculated

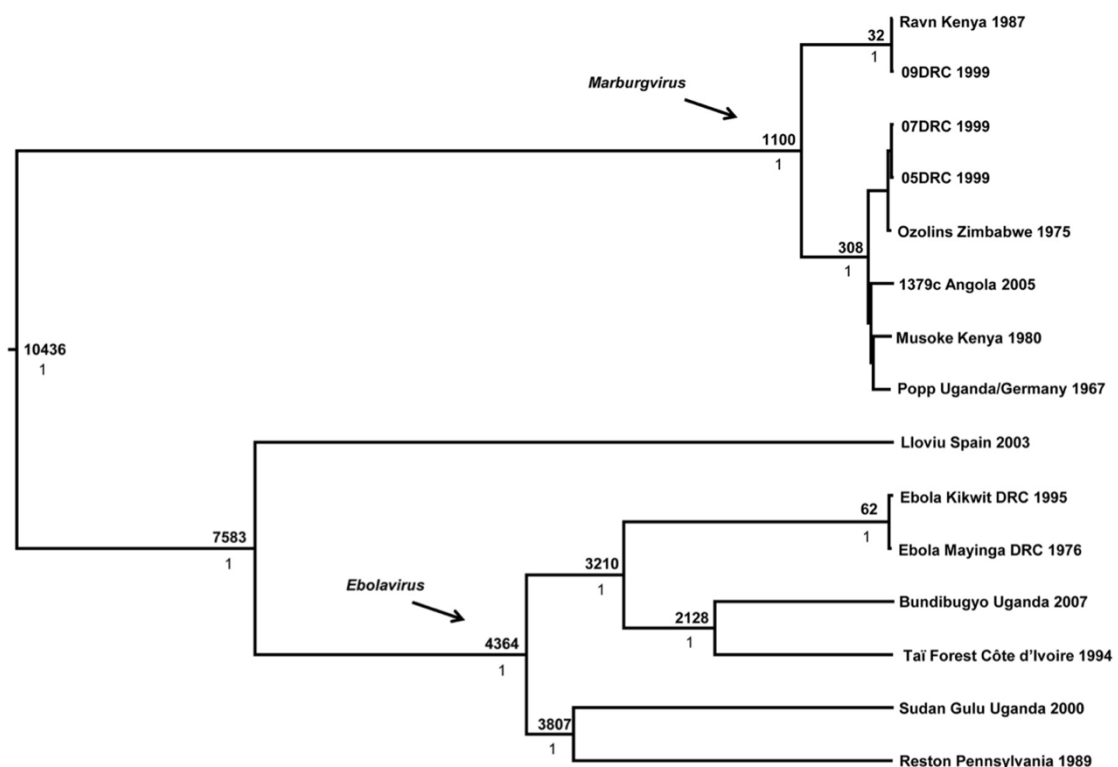


Figure 50. Bayesian coalescent phylogenetic analysis determining the molecular evolutionary rate *filoviridae* virus family. Numbers indicate years before present. (Carroll et al. 2013, 2615)

⁴⁴⁵ Suzuki and Gojobori 1997, 803.

⁴⁴⁶ Carroll et al. 2013, 2614.

that Ebolavirus, before splitting into specific African strains, is estimated to have emerged around 4,000 years ago (Fig. 50), indicating that early strains of Ebolavirus probably existed over two-and-a-half millennia before the first outbreaks of the Plague of Cyprian.

Discussion

It appears the best fit for the disease related by the ancient sources in the mid-third century is most likely an early strain of Ebolavirus, probably not unlike the strains which we are familiar with today. The symptoms and epidemiology related by Cyprian and others best match the modern symptoms for EVD, and early strains of the virus existed well before the start of the Plague of Cyprian. Of course, without aDNA evidence we cannot know for sure that Ebolavirus was the cause of the third-century pestilence, but, in the absence of that, Ebolavirus is our best working hypothesis.

The Plague of Cyprian and mid-Third-Century Climate

Using Ebolavirus as a working hypothesis for the disease responsible for the Plague of Cyprian, this section will consider whether there might be a positive correlation between the incidence of Ebolavirus and climate.

Third-century climate

Recalling the climatic proxy evidence for the third century from Chapter 1, compared to the preceding period AD 150 – 200, climate in the first half of the third century was far more favourable. However, in the second half of the century climate began to deteriorate, mostly from the AD 260s onward. This is evident from nearly

all of the climatic proxies, aside from those, such as in Spain and Turkey, that show cooling and aridity for the whole century. We also see third-century high points in aridity in the early 250s in tree-ring data from Libya and at Lake Turkana in Kenya (a proxy for Ethiopian-Highlands precipitation), both of which may relate to the drought/famine described by Cyprian and Dionysius of Alexandria around the same time and in the same regions.⁴⁴⁷ No other data exhibit climatic deterioration like in Libya and at Lake Turkana. However, many proxies display conspicuous variability in the mid-260s, undoubtedly brought on by the massive eruption in AD 263 (location unknown).

For most of the Empire, climate appears to have been relatively stable up until the 263 eruption, while our pestilence was present by the early-250s, a decade before Empire-wide climatic variation began. The spikes in aridity in Libya are important to note; especially since the first written evidence for our pestilence comes 1600 km north. However, as we will see in the next section, if Ebolavirus (or any VHF) was the disease responsible for the Plague of Cyprian, it is unlikely that climate change played a role in its rise and spread.

Ebolavirus and climate

The link between climate change and Ebolavirus incidence is very weak. There has not been much investigation on climate and Ebola, but the research that has been conducted has returned negative results. In 2015, a paper was presented at a conference on modern climate change in Paris, titled, “Climate Change and Ebola Outbreaks: Are they connected?”.⁴⁴⁸ It is clear from the conference poster and

⁴⁴⁷ Nash *et al.* 2016, 6.

⁴⁴⁸ Kassie, Bourgarel and Roger 2015.

abstract that no real data-based research had been done by the Ebolavirus researchers, but instead they were asking questions and posing possible scenarios about how climate change *might* affect Ebola incidence. For example, they propose climate change may affect the “population density, migration, habitat utilization, reproduction, feeding behaviour, nature or intensity of inter-specific contact” of bats, which are all valid points. But there are two problems with this: (1) they present no data to support the argument; and (2) the bat is the *suspected* reservoir for Ebola – as the bat is the reservoir for Marburg virus and the two viruses are closely related – but this has yet to be confirmed scientifically.⁴⁴⁹ Next, they suggest that climate change can bring about increased contact between humans and wildlife by “impacting the natural habitats of the reservoir species and modify human behavior and activities.” These are important points to consider, but they are not biological responses of the disease to climate change. Thus, by 2015, the research conducted on Ebola and climate change was minimal and insufficient.

In 2017, a study led by Gytis Dudas, that included 97 co-authors, examined the virus’ genomes to show factors that spread and sustain Ebolavirus epidemics for which we have data. They tested the economic, administrative, climatic, demographic, and infrastructural features of each region to gauge the dynamics of Ebolavirus. Dudas and company found that “climatic effects were not significantly associated with virus dispersal.”⁴⁵⁰ Additionally, as epidemics progressed, they feared that “increased rainfall and humidity might prolong environmental persistence of EBOV particles, increasing the likelihood of transmission. Although

⁴⁴⁹ Perng and Solbrig 2013, 351; Dudas *et al.* 2017.

⁴⁵⁰ Dudas *et al.* 2017, 7.

we found no evidence of an association between EBOV dispersal and any aspects of local climate, we find that regions with less seasonal variation in temperature, and more rainfall, tended to have larger EVD outbreaks.”⁴⁵¹ Thus, their fear that increased precipitation may be strongly linked to transmissibility was not firmly substantiated by the data.

While there is no strong relationship between climate and Ebola incidence, the study led by Dudas revealed, predictably, relationships with factors related to urbanisation, particularly population sizes. But they also note “a significant inverse association with travel times to the nearest settlement with >50,000 inhabitants,” confirming that widespread transmission in denser areas is an important contributing factor to the size of Ebola epidemics.⁴⁵² In addition, they discuss how regional connectedness was a contributing factor, explaining how “international borders [within Africa] were associated with decreased rates of transmission compared to national borders,” and that “individual outbreaks were constrained by the degree of connectedness among contact networks.”

Discussion

With no major shifts in climate preceding or coinciding with the onset of our plague, and our best hypothesis for the disease responsible showing no strong relationship with climate, it appears that the mid-third-century epidemic was unassisted by climate change. Instead, keeping in mind the study led by Dudas, which emphasises connectivity and urbanisation, the connectedness of the Roman Empire might have

⁴⁵¹ Dudas *et al.* 2017, 10.

⁴⁵² Dudas *et al.* 2017, 9.

been the main factor contributing to the Plague of Cyprian's geographical extent. The maritime routes between Africa and Rome were presumably the reason the disease made it across the Mediterranean in the same year. Where it began, however, is uncertain.

How does the Plague of Cyprian fit into “the Crisis of the Third Century”?

Harper speculates that the Plague of Cyprian was the root cause of the third-century crisis, citing financial, socio-political, geo-political, and cultural dimensions that may have resulted from the pandemic.⁴⁵³ There is no doubt that our plague affected a certain portion of the Empire, but given the little evidence we have for it, to hint, as Harper does, that the Plague of Cyprian caused the problems above is an overestimate, speculation or not.⁴⁵⁴ Did it exacerbate those problems? Most likely.

For the financial dimension, the crux of Harper's argument is the coincidence of timing between the pandemic and debasement of coinage. This is not strong evidence since there was much political strife and turnover occurring from the end of the Severan dynasty. Additionally, he thinks when efforts came to restore the currency, the third-century epidemic is what prevented success, proposing the pestilence played a causal role in “disrupting the flow of precious metals, undermining the emperors' legitimacy, or provoking disequilibria of supply and demand.”⁴⁵⁵ This is a nice idea, but there is little evidence to support it. The 2018 lead-pollution data *does* illustrate mid-to-late century disruption (Fig. 49), but why should this be connected to the Plague of Cyprian with the many additional troubles

⁴⁵³ Harper 2015, 249-259.

⁴⁵⁴ Kearns 2018, 53: Kearns also notes the difficulties of assessing impact with the little evidence we have.

⁴⁵⁵ Harper 2015, 252.

occurring at the time? It is not implausible that widespread mortality would have decimated the tax base, but was mortality Empire-wide? Definitive evidence is lacking here. Aside from the timing of both events, the argument that our plague was a principal factor bringing about the infamous third-century debasement of Roman coinage is, at the moment, rather empty.

He also ponders whether our pestilence was a factor contributing to the reorganisation of the state and the army.⁴⁵⁶ He is a bit more cautious here, noting a correlation, but admits there are no visible causal links. Similar to Rome's financial issues, it is difficult to associate any socio-political changes with a pestilence rather than a response to the continual usurpations from the mid-230s onward. That Gallienus eliminated senatorial command of the legions seems more likely a product of his desire not to be usurped himself than anything related to an exogenous shock like an epidemic.

It is clear from the literary sources that the geopolitical situation was worsened by the Plague of Cyprian. The Sibylline Oracle, the *Historia Augusta* (if we are to trust it), and Zosimus – the *HA* probably drawing on Aurelius Victor and the latter on Dexippus – all portray war and disease occurring simultaneously, with the pestilence exacerbating an already tense situation near the eastern borders. In fact, Zosimus reports that the pestilence was partly the reason for Valerian's capture by Sapor I.⁴⁵⁷ But how much can we attribute geo-political difficulties to a pestilence?

⁴⁵⁶ Harper 2015, 253-254.

⁴⁵⁷ Zosimus, *Historia Nova*, 1.36: "Valerian had by this time heard of the disturbances in Bithynia, but his district would not allow him to confide the defense of it to any of his generals. He therefore sent Felix to Byzantium, and went in person from Antioch into Cappadocia and after he had done some injury to every city by which he passed, he returned homeward. But the plague then attacked his troops, and destroyed most of them, at the time when Sapor made an attempt upon the east, and reduced most of it into subjection. In his weakness, he despaired of ever remedying the present crisis and tried to buy peace. Sapor, however, dismissed the Roman ambassadors empty-handed and demanded that the emperor himself come to negotiate

Harper says, “historians [ancient] described the pandemic both as a trigger for the invasions and a reason for Rome’s failure to contain the threat.”⁴⁵⁸ My reading of the sources reveals no such judgement from the ancient writers who document our plague. Even the *Thirteenth Sibylline Oracle*, the most apocalyptic work of the bunch, notes that the plague started amidst the eastern wars, almost as a divine response to the fighting.⁴⁵⁹ Yes, the third-century pestilence probably worsened geo-political conditions for the Empire. But did it incite tensions? Aside from possibly stirring up Christian/pagan tensions within the Empire, there is no strong evidence that the Plague of Cyprian *started* wars between Rome and its neighbours.

Harper uses cultural impacts as the last lens with which to measure our pestilence, specifically through the expansion of the Christian church. He is passive here, saying, “One could suggest that a pandemic had the potential to amplify [the presence of Christianity]”, but does not go so far as to propose it. However, later he makes a good point: “It is not that the pandemic sparked a crisis of malaise and disbelief, but that its effects undermined the stability of a religious culture that was embedded in society.”⁴⁶⁰ It is possible, and quite likely in fact, that the third-century pandemic further stirred a period of religious transition, well-depicted by Cyprian of Carthage and Dionysius of Alexandria in the AD 250s. Changes to the Empire’s religious fabric eventually culminated less than a century later with Constantine I’s (AD 306 – 337) conversion to Christianity and his empire-wide edict of tolerance for

the important points. The latter imprudently consented to this demand, and setting out incautiously with only a small retinue to meet Sapor to discuss peace, was suddenly taken by the enemy. So he was reduced to the rank of a slave and died in the hands of the Persians, bequeathing the greatest shame to the Roman reputation ever after.”

⁴⁵⁸ Harper 2015, 256.

⁴⁵⁹ Potter 1990, 173 and 285.

⁴⁶⁰ Harper 2015, 252.

the religion. However, how can we precisely gauge the impact of the pestilence on the rise of Christianity, from Africa in the 250s to Constantine's Edict of Milan (AD 311), beyond citing examples of its expansion and posing the question? I do not see a way, and neither, it seems, does Harper.

A symbol for the difficulties in disentangling the third-century pestilence from everything else – barbarian invasions, rebellions, civil wars, debasement of coinage, and much political instability – is the recent ancient lead-pollution evidence detected in Greenland ice (Fig. 51).⁴⁶¹ Major reductions in lead pollution (a proxy for mining, and thus economic activity) just after the first outbreaks of the Antonine Plague work well in showing the impact of that epidemic, since, aside from the Marcommanic Wars (that were restricted to areas of modern-day Germany, the Balkans, and Danube frontier) and later-second-century climate change (which could not have been responsible for immediate reductions), the Antonine Plague was the chief calamity stressing the Empire at the time. We are not so lucky with the Plague of Cyprian. The picture is much cloudier. Additionally, at the start of the Plague of Cyprian lead pollution is already at an all-time low for the Roman Empire. This shows that political instability, and everything that came with it, from the end of the Severan period was at least partly responsible for the decline attested in the mid-third century; it cannot all be attributed to the pestilence. Surely, the Plague of Cyprian contributed to regional downturn after 250, but was it a leading cause of the third-century crisis, as Harper suggests? Evidence is lacking to assert such a hypothesis. What the evidence does show is: the Plague of Cyprian began in either Africa or Europe in the early AD 250s under Trebonianus Gallus and Volusian; it

⁴⁶¹ McConnell *et al.* 2018.

devastated Carthage (Cyprian) and Alexandria (Dionysius); it eventually made it to the Near East (*Thirteenth Sibylline Oracle*) while the Empire was contending with barbarian invasions; two sources (one unreliable: *Historia Augusta*; and one non-contemporary: Zosimus) say it reached Greece; two sources (one unreliable: *Historia Augusta*; and one non-contemporary: Aurelius Victor) say it reached Rome; outside of these affected cities/regions the evidence for Europe is nil. Thus, to say the Plague of Cyprian “ravaged the empire” is, at this stage, a tenuous claim.

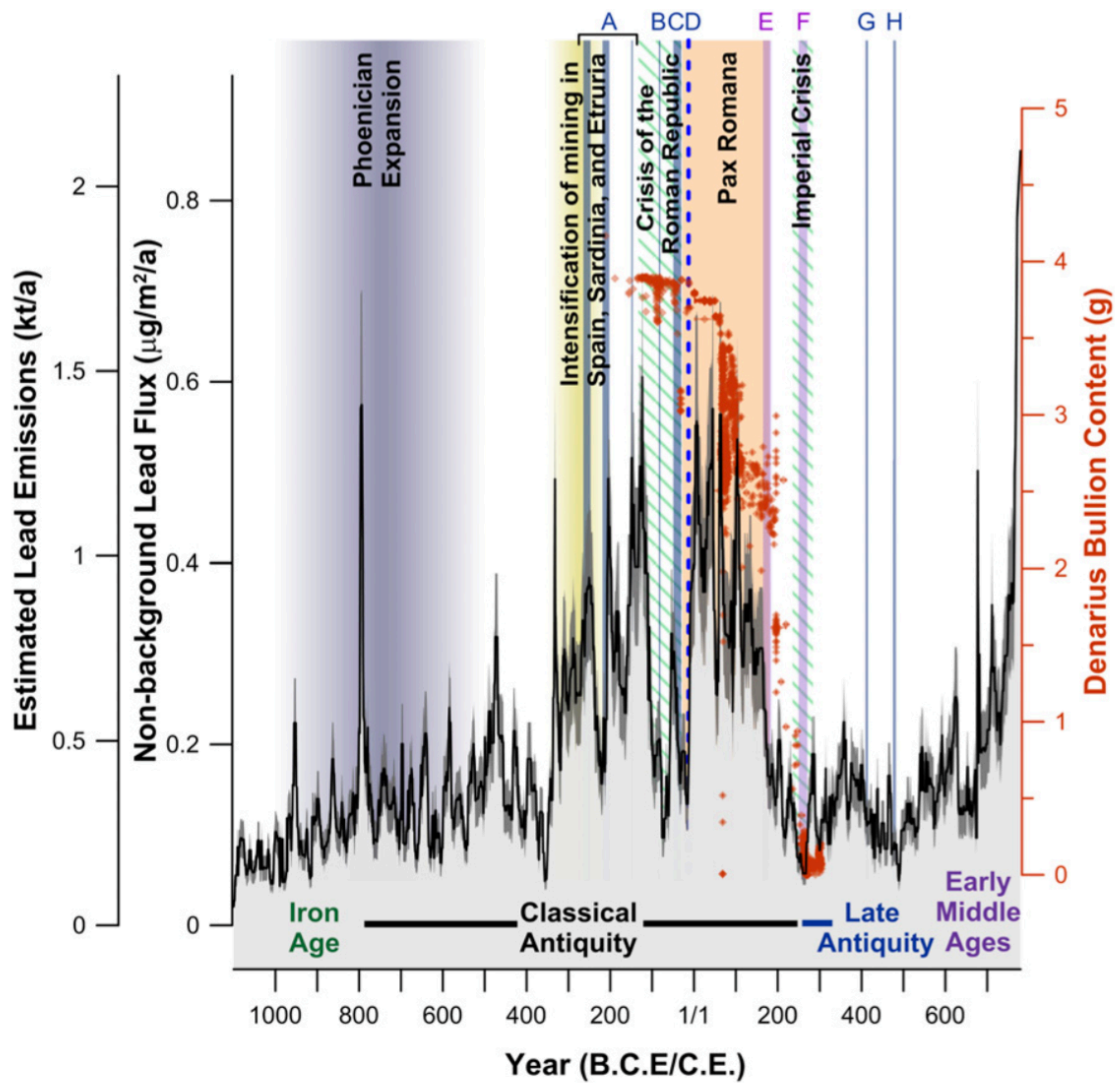


Figure 51. Measurements of lead deposition in Greenland ice, showing European lead emissions and *denarius* coinage (silver) bullion content. “E” in purple shading represents **the Antonine Plague**. “F” represents **the Plague of Cyprian**. (McConnell et al. 2018)

The Justinianic Plague

Introduction

There is a consensus among scholars of late antiquity and the early middle ages that in mid-July 541 the first wave of a deadly epidemic broke out in the eastern Nile Delta, and over the course of four years spread throughout the Empire, reaching most of its boundaries, and as far north as Britain. While the effects of the Justinianic Plague are still being debated (vehemently) today, few scholars, if any, doubt that most of the Empire's regions experienced the pestilence; and thanks to ancient DNA analysis the debate on its etiological agent can now be closed: true plague was the culprit.⁴⁶² Yet, another debate remains open. Was sixth-century climate change a major factor contributing to the rise and spread of the plague? Some academics and popular historians suggest that it was; some have said the biological processes of plague dynamics are too complex to form conclusions; and others all but reject any connection.⁴⁶³ Even among those who do see a causal link, there is no convincing explanation of how such a link might have worked in practice. There is, however, no denying that major climatic events coincided with the sixth-century pandemic. The Late Antique Little Ice Age (LALIA) occurred right alongside the Justinianic Plague – in fact, as we saw in Chapter 1, the second eruption in the string of three that

⁴⁶² That debate was still ongoing at Lester K. Little's 2001 conference in Rome and in his subsequent 2007 volume compiling the conference papers. Most contributors argued for bubonic plague as the disease-causing agent.

⁴⁶³ For those proposing or hinting that climate change did play a role: Keys 1999; Rosen 2007; Harper 2017. Newfield 2016 does not discount the theory as impossible, but he thinks that, at this stage, climate/plague research is too young and underdeveloped to reach a conclusion. For the most sceptical: Mordechai and Eisenberg 2019; Mordechai *et al.* 2019.

helped trigger the LALIA happened in AD 540, around one year before the first recorded outbreak of the plague.⁴⁶⁴ While the eruption of AD 536 resulted in significant changes to Mediterranean climate, the eruption of 540 was even larger, and compounded with the 536 eruption, caused temperatures to drop considerably in the Mediterranean region and beyond, and brought dramatic fluctuations in wetness in some areas.

The main objective of this chapter is to consider whether the sixth-century-AD climate change that we observe in palaeoclimatic proxies played a large role in the proliferation of the Justinianic Plague (first wave: AD 541 – 544) by investigating the complexities of plague dynamics. To do this, we must examine three elements that help bubonic plague emerge in humans: 1) the bacterium responsible for the plague, *Yersinia pestis*; 2) the vectors that transmit *Y. pestis* and help the plague spread, which have been proven to be mainly rat fleas (e.g. *Xenopsylla Cheopis*); and 3) the rodent, specifically *Rattus rattus* (the black rat), the principal mammal bridging the gap for *Y. pestis* to reach humans. Each plays a significant role in the appearance of bubonic plague pandemics, and the life cycles and reactions of each appear to be influenced by climate. This chapter investigates the interactions between the components of bubonic plague and climate, specifically, temperature and wetness (precipitation and humidity). On the basis of these findings, and the palaeoclimatic evidence for the sixth century, I then consider the likelihood that sixth-century climate change was a catalyst in the irruption and persistence of the Justinianic Plague.

⁴⁶⁴ Büntgen et al. 2011; Büntgen et al. 2016; Luterbacher et al. 2016; McCormick et al. 2012; Göktürk 2011; LeRoy et al. 2015; Stoffel et al. 2015; Holzhauser, Magny, Zumbühl 2005.

Investigating the relationship between climate and plague is the foremost goal of this chapter, but the chapter has additional targets. One is assessing the effects of the Justinianic Plague on society in late antiquity; that is considering the influence the epidemic had on the economy and demography. That means commenting on the ongoing maximalist/minimalist debate which has experienced revitalised fervency in the past two decades. While there is no overwhelming consensus on its macro-historical effects, a majority of scholars think our late-antique plague was no minor event; that even if it did not shape the Empire's tail end, it was certainly no small bump in the road.⁴⁶⁵ There are, however, some who believe, unequivocally, that the Justinianic Plague was the leading actor in the Empire's demise.⁴⁶⁶ A new group of historians has set out to challenge this, not merely taking an anti-catastrophist stance, but even arguing that the plague was inconsequential.⁴⁶⁷ This stance is mistaken. While it is a leap to suggest, based on the evidence we have, that the Justinianic Plague was *the* major factor that brought late antiquity to an end, it is equally a leap to propose, despite evidence for the plague reaching a multitude of the Empire's most important cities and towns, that our pandemic was insignificant and only affected the places where we have found evidence of it.

Another objective of this chapter is investigating whether the downturn we observe from the mid-sixth century was caused by the Justinianic Plague or the LALIA. Both were significant events that affected regions throughout the Empire to varying degrees. Both probably brought demographic loss, directly by way of the

⁴⁶⁵ Stathakopoulos 2000; McCormick 2001; Stathakopoulos 2004; Horden 2005; the many contributors to Little 2007; McCormick 2015.

⁴⁶⁶ Meier 2016; Harper 2017.

⁴⁶⁷ Mordechai and Eisenberg 2019; Eisenberg and Mordechai 2019; Mordechai *et al.* 2019.

plague, and indirectly from the LALIA through famine brought on by catastrophic climate change. This chapter showcases the difficulties encountered in disentangling the effects of the plague from the LALIA.

The chapter begins with a historiographical discussion, where I review the more recent work on the Justinianic Plague, mostly from the last twenty years, which inevitably leads to the maximalist/minimalist debate discussed above. However, the section is not wholly devoted to that debate. Ample attention is paid to Dionysios Stathakopoulous' efforts to reinvigorate the idea of the Justinianic Plague as an important part of late antiquity.⁴⁶⁸ Equal recognition is given to Lester K. Little's 2001 conference in Rome that gathered twelve specialist historians and archaeologists to give papers and discuss the importance of our pestilence, culminating six years later in a volume based on those papers.⁴⁶⁹ Kyle Harper's contribution to the topic, and the maximalist stance, must be emphasised.⁴⁷⁰ And the new work of Lee Mordechai and Merle Eisenberg, working in direct opposition to Harper, is essential to our pandemic's historiography.⁴⁷¹ Possibly most important is the recent aDNA research and phylogenetic analyses on the Justinianic Plague, which has changed the way we write about it.⁴⁷² However, these papers enter the discussion later in the chapter, in the section on archaeology.

We take with us what we learned from the historiography into the following section on the ancient literary sources. The section focuses predominantly on seven important contemporary sources – Procopius, John Malalas, John of Ephesus,

⁴⁶⁸ Stathakopoulos 2000; 2004.

⁴⁶⁹ Little 2007.

⁴⁷⁰ Harper 2017.

⁴⁷¹ Mordechai and Eisenberg 2019; Eisenberg and Mordechai 2019; Mordechai *et al.* 2019.

⁴⁷² Drancourt *et al.* 1998; Drancourt and Raoult 2004; Harbeck *et al.* 2013; Rasmussen *et al.* 2015; Feldman *et al.* 2016b; Keller *et al.* 2019; Rascovan *et al.* 2019.

Gregory of Tours, Simeon Stylites the younger, Evagrius Scholasticus, and Agathias – but a few non-contemporary writers are also discussed. From the ancient sources I trace the path of the plague, focusing on locations that we know for certain were struck by it, while also speculating about their surrounding regions, and other towns and cities that in all likelihood were affected. The next section looks at the relevant archaeological remains, mainly consisting of mass burials. This brings us to the ancient DNA analysis, leading to a discussion on the diagnosis of our pestilence.

The following sections focus on the epidemiology of plague, carriers and vectors, and the plague cycle and factors affecting transmission, in which I investigate the bacterium itself, *Y. pestis*, vectors of the plague, and reservoirs. The origin and evolution of *Y. pestis* is crucial to the chapter, as I examine the important and most recent phylogenetic studies on the bacterium to determine potential plague reservoirs and how *Y. pestis* evolved up to the first outbreak of the Justinianic Plague. The evolution of *Y. pestis* ties directly to its transmitters, thus analysis of the relevant vectors precedes it; however, special attention is given to its apparent most important vectors: the oriental rat flea (*Xenopsylla cheopis*), the northern rat flea (*Nosopsyllus fasciatus*), and *Xenopsylla brasiliensis*. Next we observe the main reservoirs of plague, rodents. The black rat (*Rattus rattus*) will feature heavily, but additional rodent carriers, such as the brown rat (*Rattus norvegicus*), are considered.

The section just discussed unavoidably analyses the effect of climate on vectors. However, the effects of climate on the plague cycle and speculation about the role of sixth-century climate change in the first wave of our plague is reserved for the following section. In that section, I recall the climatic situation in the first half of the sixth century (Chapter 1), briefly revisiting the palaeoclimatic evidence in the

relevant regions, as well as the biological processes of the plague cycle to determine the plausibility of whether sixth-century climate change, and more specifically the double whammy of the eruptions of AD 536 and AD 540, led to an outbreak of bubonic plague at Pelusium, Egypt in AD 541.

The final section considers whether discernible sixth-century downturn should be attributed to the Justinianic Plague or to the LALIA. It cannot be an exhaustive effort, but the section examines an important case-study region in the Near East, which has recently been the focus of productive research papers that stand as good examples of how to approach resolving this difficult question. This section also revisits the maximalist/ minimalist debate, as I comment on which side, if any, seems to be on the right track according to the present catalogue of evidence.

We learn that the Justinianic Plague was most likely influenced by the Late Antique Little Ice Age. While scholars may continue to argue that the plague cycle is too complex to say definitively that climate change played a role, it is undeniable that each component of the plague cycle is affected by climate in the ways we see climate reacting in the sixth century. We may never know for sure if climate change was the leading catalyst that ignited the Justinianic Plague, but science indicates that dramatic fluctuations in climate, such as those at the beginning of the LALIA, can stir up plague reservoirs, assist the bacillus in reproducing, and allow plague vectors to become better transmitters; thus, probability is high. It is more difficult to tell if the Justinianic Plague or the LALIA had a larger and more lasting impact on late antiquity. But one thing is certain, neither was inconsequential.

Historiography

The Justinianic Plague has received significant attention in the last two decades. A number of scholars have argued that the multi-wave pandemic spanning two centuries was an important part of late antiquity. Just over fifty years ago, one of the more important 20th-century works on our pestilence was published by Jacques Le Goff and Jean-Nöel Biraben, in which they related, mainly based on the literary evidence, most of the main historiographical aspects of the Justinianic Plague that we still refer to today, particularly the pandemic's path and its waves. Their work, however, contains a number of inaccuracies.⁴⁷³ The next important work came in 1989 from the French historian Jean Durliat, who argued that scholars of our plague have relied so heavily on the written evidence that we have failed to recognize the lack of archaeological evidence; in effect, founding the minimalist camp.⁴⁷⁴ Aside from those two papers, 20th-century scholarship on the Justinianic Plague was minimal.

That changed at the turn of the millennium, but first, in 1999 an archaeology correspondent for *The Independent* newspaper made his mark on Justinianic Plague scholarship; and one of his theories is most relevant to the main thesis of this chapter. In his book, *Catastrophe: An Investigation into the Origins of the Modern World*, David Keys was the first to propose a connection between climatic events of the sixth century and the initial outbreak of the Justinianic Plague.⁴⁷⁵ Keys advances, a bit too neatly and dogmatically, that "it was almost certainly the climatic chaos of the mid 530s which initiated the epidemiological process which sent plague and death

⁴⁷³ Biraben and Le Goff 1969; for their errors see Stathakopoulos 2004, 113-24.

⁴⁷⁴ Durliat 1989.

⁴⁷⁵ Keys 1999, 18-23.

rolling across so much of the planet and which helped change so much of subsequent history.”⁴⁷⁶ He thinks it was excessive rainfall, which can cause explosions in rodent populations, that forced plague-carrying rodents to seek new territories, and thus led to the first outbreaks of our plague; he does not explore the effect of climate change on the bacillus or its vector. Many scholars have taken issue with the work. One claimed most of the evidence presented is “based on poor sources or is simply incorrect.”⁴⁷⁷ This must be kept in mind when assessing *Catastrophe*. With that said, it was Keys who first directed attention to the possible connection between the onset of the LALIA and the Justinianic Plague, provoking interest in the subject; and based on today’s evidence many of his theories have been upheld, so he should be duly credited for his efforts.

The first paper of the new millennium on our plague, starting what would become a busy and productive two decades of Justinianic Plague scholarship, was Dionysios Stathakopoulos’ 2000 article “The Justinianic Plague Revisited”. Stathakopoulos surveyed everything we knew about our plague up to that point, and more.⁴⁷⁸ Stathakopoulos followed up his survey from 2000 with a monumental volume in 2004 that covers every aspect of the Justinianic Plague (as well as other late-antique ecological calamities), which also includes a catalogue of all mentions of famine and pestilence from the end of the third-century-AD crisis to the mid-eighth century.⁴⁷⁹ The volume is comprehensive and seminal in its hypotheses.

Stathakopoulos distinguishes eighteen waves of the Justinian Plague based on the

⁴⁷⁶ Keys 1999, 18.

⁴⁷⁷ Dark 1999, 21-22.

⁴⁷⁸ Stathakopoulos 2000.

⁴⁷⁹ Stathakopoulos 2004.

written evidence. In some cases, his diagnostic reasoning is weak, but, on the whole, his efforts have revived the subject.⁴⁸⁰ Stathakopoulos undoubtedly stands in the maximalist camp, and not just because of his eighteen waves (Table 6). He thinks the Justinianic Plague had a substantial short-term impact on the Empire, as well as consequential long-term effects. While Stathakopoulos feels that “estimates of the total population decline caused by the plague are pure guesswork”, it is evident that he thinks our pestilence helped ravage the Empire to the point of transition and eventual disappearance.⁴⁸¹ Stathakopoulos is well versed in the plague cycle and its biological actors, but he does not speculate on the role of climate change in the emergence of plague in the sixth century AD.

One year later in 2005, Peregrine Horden wrote a paper on our plague for *The Cambridge Companion to the Age of Justinian*. In it, Horden focuses on historiography, following the plague’s path, discussing its severity and diagnosis, comparisons with the Black Death and the third plague pandemic, and comments on the current direction of research and scholarship. Most of his analysis is based on the written evidence. However, Horden does consider some relevant science and environmental stimuli.⁴⁸² In his section on outstanding questions that need to be addressed, he refers to then recent aDNA work on *Y. pestis*. Afterwards, he discusses the possible effects of seasonality and climate on bubonic plague, and thus the Justinianic Plague, implying more research is needed this area. Horden broaches the question of the

⁴⁸⁰ As pointed out by Eisenberg and Mordechai 2019, on multiple occasions in his catalogue Stathakopoulos assumes nondescript mentions of disease by ancient authors are references to the plague, if they fall within one of his self-declared plague waves, or if the location of the ancient source was near a well-defined outbreak. Many mentions are very clearly referencing plague, specifically bubonic plague, but in a few instances where the diagnosis should at least be scrutinised, Stathakopoulos assigns it as an outbreak of plague: e.g. 2004 catalogue #115 and #117.

⁴⁸¹ Stathakopoulos 2004, 166-173.

⁴⁸² Horden 2005.

effect of temperature on the rat flea's reproduction, but, more importantly, he talks about sixth-century climatic events as the possible driving force behind the first outbreaks of plague, contemplating whether climate change extended a plague reservoir's spatiotemporal boundaries.⁴⁸³ He reflects on this from the perspective of rat migration and the improved survival of *Y. pestis* under worsening climatic conditions, proposing, "if *Yersinia pestis* was involved, the reduced temperatures of the 530s expanded the area within which plague survived enzootically." But he explores no further. Nevertheless, Horden is one of the first academics to propose climate change as an influencer in a paper on our plague, and to suggest how that link might have worked. On the impact of the Justinianic Plague, Horden cautiously leans towards the maximalist camp, asserting that the plague was "a major ingredient in the changes that we detect in the Age of Justinian and the seventh to eighth centuries, but only one such ingredient among several."⁴⁸⁴

A volume edited by Lester Little in 2007, based on a conference held at the American Academy in Rome in 2001, was the next important development in Justinianic Plague research. The volume, filled with chapters by historians, archaeologists, and a molecular biologist, establishes a chronology for our plague, introduces new non-literary evidence, and addresses a number of key debates, paying particular attention to diagnosing the pestilence as bubonic plague without definitive scientific evidence.⁴⁸⁵ The use of archaeological evidence to complement

⁴⁸³ Horden 2005, 149-153.

⁴⁸⁴ Horden 2005, 155.

⁴⁸⁵ Little 2007. Michel Drancourt presented a paper at the 2001 conference, based on aDNA work done by him and colleagues on sixth-century mass graves, that indicated *Y. pestis* was present in the mass burial, thus they proposed *Y. pestis* was responsible for the Justinianic Plague; however, their findings were later contested as flawed by contamination: Gilbert *et al.* 2004; Vergnaud 2005; Harbeck *et al.* 2013; Mordechai and Eisenberg 2019.

the written evidence, featured in the chapters by Hugh Kennedy, John Maddicott, and Michael McCormick, is a refreshing departure from previous scholarship.⁴⁸⁶ As a whole, the book leans towards the maximalist camp, urging, not always explicitly, that the Justinianic pandemic was a significant late-antique event; a view especially heard in the voices of Peter Sarris, Stathakopoulos, Kennedy, and McCormick.

Robert Sallares and McCormick both explore the potential role of climate in the emergence of the Justinianic Plague in Little's volume. Sallares considers the effects each season might have on rats and fleas, but his exploration is very brief.⁴⁸⁷ He proposes correctly (I believe) that it is perfectly possible for the oriental rat flea *X. cheopis* to breed in northern Europe.⁴⁸⁸ Sallares goes on to ponder a connection between the onset of the Late Antique Little Ice Age (before its name was coined) and the Justinianic Plague, citing Keys' rodent-explosion theory, saying that it is plausible but not subscribing to it.⁴⁸⁹ Later in his chapter Sallares claims that "it is not inconceivable that the exceptionally cold conditions in AD 540 – 541 turned one of these frequent small-scale highly localized epidemics of the airborne form of the disease in a region of sylvatic plague, somewhere in the vicinity of Libya, Egypt, or the Levant, into a massive epidemic of pneumonic plague affecting humans on a large scale for the first time."⁴⁹⁰ His proposition is not impossible, but, due to its lethality, besides localised occurrences pneumonic plague cannot explain plague outbreaks throughout the Mediterranean – e.g. travelling north from Egypt to Constantinople is probably too far for pneumonic plague to maintain

⁴⁸⁶ As pointed out by Eisenberg and Mordechai 2019, 160.

⁴⁸⁷ Sallares 2007b, 283.

⁴⁸⁸ Citing: Hirst 1953, 272-275; Shrewsbury 1970, 3. A few 20th-century studies had indicated that *X. cheopis* cannot have been the vector for plague in the northern parts of the Empire because it is too cold.

⁴⁸⁹ Sallares 2007b, 284.

⁴⁹⁰ Sallares 2007b, 286.

transmissibility. Moreover, the theory does not account for the multiple references to “buboes” in the written evidence (below), which is not a symptom of pneumonic plague. Sallares does not consider the influence of sixth-century-AD climate change on the bubonic plague cycle, and thus the Justinianic Plague. Nevertheless, Sallares’ questions about the role of climate change as a potential catalyst in the first outbreak of our plague are important.

Also in Little’s 2007 volume, McCormick talks about the “demonstrated epidemiological link” between extreme wetness and the bubonic plague, citing a study from the American southwest. Similar to Keys’ hypothesis, McCormick explains that “Periods of above-normal precipitation produce increases in plague outbreaks; conversely, periods of exceptional dryness have the opposite effect. That link operates by means of a trophic cascade – a surge in sustenance all along the plant, insect, and rodent food chain.”⁴⁹¹ McCormick then briefly discusses the role of rodent carriers and insect vectors in plague transmission, and advises that “careful attention will have to be paid” to the influence of climate on them, but his review stops there.⁴⁹² Overall, his chapter advocates the use of science in research on the Justinianic Plague, a sentiment he has put into practice with contributions to science-based publications.⁴⁹³ McCormick has been thinking about the Justinianic Plague since researching it for his book, *Origins of the European Economy*, published in 2001. Since then he has become an important figure in the field. Two years later, he wrote a now highly-cited article about the role of rats in the spread of plague, which, in

⁴⁹¹ McCormick 2007, 308.

⁴⁹² McCormick 2007, 308-309.

⁴⁹³ Keller *et al.* 2019; McCormick *et al.* 2012.

essence, is an appendix to *Origins*.⁴⁹⁴ Similar to his chapter in the volume edited by Little, McCormick talks generally about the effects of excessive precipitation on rodent populations and the implications that may have for plague outbreaks.⁴⁹⁵ But the paper also examines the potential influence of sixth-century climate change on the emergence of our plague, particularly the role of the 536 “dust cloud” event (its cause still unclear in 2003 but now confirmed to have been a massive volcanic eruption, as we saw in Chapter 1), without proposing conclusions.⁴⁹⁶ McCormick’s more recent contributions are archaeological. His “tracking mass death” articles in the 2015 and 2016 volumes of the *Journal of Roman Archaeology*, are an analysis and catalogue of mass graves for late antiquity.⁴⁹⁷ These articles helped broaden and gather the non-literary, mortality-based evidence for our plague, in effect responding to Durliat; the details are discussed in the section below on archaeology. McCormick stands firmly in the maximalist camp, believing the Justinianic pandemic both caused significant population loss and was consequential in the transition from late antiquity to the Middle Ages. His work has been seminal for the direction of scholarship on the Justinianic Plague.

One historian influenced by McCormick whose work has been crucial to the historiography of this pandemic is his former student Kyle Harper. Harper, who has already been mentioned extensively in this thesis, published his book *The Fate of Rome* in 2017, a portion of which is devoted to the Justinianic Plague.⁴⁹⁸ Before 2017, Harper had published previously on the other “great plagues of antiquity”, but this

⁴⁹⁴ McCormick 2003.

⁴⁹⁵ McCormick 2003, 18-20.

⁴⁹⁶ McCormick 2003, 20-21.

⁴⁹⁷ McCormick 2015; McCormick 2016.

⁴⁹⁸ Harper 2017, 199-245.

was his first work that investigated the Plague of Justinian. McCormick's impact on Harper is clear; Harper is a maximalist with the same essential view as his ex-supervisor: the late antique pandemic was a significant, if not *the* most significant, event that helped bring about the Empire's demise. Climate change and the LALIA also have a distinct place in his work, as he argues that optimal weather benefited the Empire as much as significant climate cooling affected it adversely. With his book, Harper, unwittingly or not, assumed the role of leader of the maximalist camp. His use of sensationalist language and a catastrophist's viewpoint has both roused interest in the fields of environmental history and the history of pandemics and brought criticism of Harper as an academic. At the heart of his thesis in *The Fate of Rome*, Harper proposes that, in combination with destruction wrought by the LALIA, the Empire's connectedness and propensity for trade and commerce helped spread the three great pestilences (especially the Justinianic pestilence), thus ushering in the end of the Empire. Harper explores the biological processes of the plague cycle effectively, and he discusses the flea's sensitivity to temperature as well as the bacillus's inability to reproduce within the flea outside of a certain temperature range.⁴⁹⁹ He even suggests that there may be a connection between the onset of LALIA and the plague, saying, "The sharp cooling of the 530s and 540s may have opened geographical possibilities that *Y. pestis* had never seen"; but he does not follow up and investigate what these possibilities might have been.

Harper's book spawned a number of reviews, both good and bad. The most extensive is a three-part review in *History Compass* led by the Byzantine historian

⁴⁹⁹ Harper 2017, 219-220.

John Haldon.⁵⁰⁰ The review is largely critical, taking issue with a number of inconsistencies throughout *The Fate of Rome*, including, Harper's selective use of primary and secondary sources, his interpretation and use of palaeoclimatic data, contradictions in the work, and his generally deterministic approach that results in a neat and tidy narrative. The third section of the review focuses specifically on the Justinianic Plague, and is mainly a critique of Harper's extrapolation from minimal evidence to Empire-wide destruction.⁵⁰¹ Haldon and company undeniably have a point – Harper's use of the evidence we *do* have to deduce demographic loss amounting to half the Empire's population approaches irresponsibility. And they are right to think that in his deterministic outlook Harper fails to consider societal coping mechanisms; that is, resilience to catastrophes like the Justinianic Plague that are apparent in some regions of the Empire, instead of decline that led to inevitable collapse. However, Haldon and colleagues, whether playing devil's advocate or not, are also misguided in their intimation that only the parts of the Empire with direct evidence of the Justinianic Plague were affected, suggesting that the plague was inconsequential.

An author on the 2018 three-part review, and a former student of Haldon, Lee Mordechai, has spearheaded the minimalist campaign for the Justinianic Plague, essentially picking up where the Haldon-led review left off. With his frequent collaborator Merle Eisenberg, Mordechai has authored four new papers (two as lead author and two as second author) focusing on the Justinianic pandemic, and

⁵⁰⁰ Haldon *et al.* 2018a; Haldon *et al.* 2018b; Haldon *et al.* 2018c.

⁵⁰¹ Haldon *et al.* 2018c.

championing the minimalist view started by Durliat in 1989.⁵⁰² Mordechai and Eisenberg's 2019 publication, "Rejecting Catastrophe", is a fleshing out of the 2018 review, which is of course a direct response to Harper's book.⁵⁰³ While it is clear that that paper acts as an alternative perspective to Harper's imaginative catastrophism, like the 2018 review led by Haldon, its deficiency of imagination leads to an unrealistic outlook. The Justinianic Plague did not just strike the places where we have direct and unambiguous evidence for it, which is, in essence, what Mordechai and Eisenberg argue by not acknowledging the possibility that the pandemic affected areas (connected by travel/trade routes) in between the places for which we have direct evidence. We are nearly 1500 years removed from the first wave of this pestilence; it should not have to be stated that not all evidence – literary, archaeological, or otherwise – has survived from the age of Justinian. As Tim Newfield said in 2018: "plague was not necessarily absent where and when there is no evidence for it."⁵⁰⁴ That is not to say that we should extrapolate our evidence to the whole Empire, like Harper, but to imply this pandemic was inconsequential because the direct evidence is not as abundant as some scholars would like only works to stifle further investigation.

What is further troubling, is that in "Rejecting Catastrophe" Mordechai and Eisenberg push the minimalist view so strongly that they nearly reject evidence. One example comes in their review of the archaeological material. They examine all of the skeletal evidence associated with the Justinianic Plague for which aDNA analysis

⁵⁰² Mordechai and Eisenberg 2019; Mordechai *et al.* 2019; Eisenberg and Mordechai 2019; White and Mordechai 2020.

⁵⁰³ Mordechai and Eisenberg 2019.

⁵⁰⁴ Newfield 2016, 102.

has produced positive results for *Y. pestis*. The central argument is that the amount of and range for the skeletal evidence we do have that has tested positive for *Y. pestis* does not support a maximalist interpretation, which is a fair point. However, a new study led by Marcel Keller revealed additional mass graves dated to the first wave (AD 543 – 544) that tested positive for *Y. pestis*, which included mass graves as far west as Spain (Valencia) and Britain (Edix Hill), regions where the plague was previously only hypothesised.⁵⁰⁵ While that study may have arrived too late for Mordechai and Eisenberg to give it full analytical treatment, they did see it, and relegated analysis of it to a footnote, listing the publication and saying, “Our overall point, however, remains.”⁵⁰⁶ But their overall point should not remain. The evidence available changed, in terms of the archaeological evidence that displays the geographical range for our plague. We have gone from having Justinianic-Plague-era evidence of *Y. pestis* from only France and Germany, to two additional, distant regions of western Europe, which suggests the possibility that the plague spread through the regions in between.

The other paper led by Mordechai from 2019, published in the *Proceedings of the National Academy of Sciences*, was more data-driven and calculated, and, overall, operates from the same minimalist approach that implies the Justinianic Plague was not a societal disrupter, because evidence is lacking to say that it was.⁵⁰⁷ In the same year, Eisenberg teamed up with Mordechai to lead a comprehensive interdisciplinary review of this pandemic, which was, essentially, an updated

⁵⁰⁵ Keller *et al.* 2019.

⁵⁰⁶ Mordechai and Eisenberg 2019, 31 (n107).

⁵⁰⁷ Mordechai *et al.* 2019; for a rebuke of this paper with responses see: Meier 2019; Mordechai *et al.* 2020a; Meier 2020; Mordechai *et al.* 2020b.

version of the Stathakopoulos's study from 2000.⁵⁰⁸ They gathered and reviewed all evidence for the Justinianic pandemic up to that point, promoting their minimalist position further. A year later, Lauren White and Mordechai published a paper that modelled hypothesised transmission routes for our plague. Using primary written evidence of outbreaks in Constantinople as a case study, their results show that bubonic and bubonic/pneumonic transmission models exceeded the maximum mortality estimates proposed in the primary source evidence, but that the outbreaks were probably shorter in duration (lasting 3 months instead of 4) than those described in the primary sources.⁵⁰⁹ Their results, indicate that if bubonic plague reached Constantinople (and probably other major ancient cities), its effects would have been devastating.

Three of these studies do not examine the possible influence of climate on the appearance of the Justinianic Plague, but, Eisenberg and Mordechai's interdisciplinary review does include a small review on the hypothesised connection between climate and plague, downplaying any link as "uncertain at best, if not unfounded."⁵¹⁰ In this section, they briefly review the central hypothesis, as well as some of the literature that considers the link (including Harper's book), but they do not bring up the cycle of plague transmission. Ultimately, they conclude in one and a half pages that a link between the onset of LALIA and the Justinianic Plague is tenuous. At the end of the section they cite Tim Newfield's 2016 paper, "Mysterious and Mortiferous Clouds", which looks at climate and disease in late antiquity. However, Newfield is far more optimistic about a link. Newfield, who is an author

⁵⁰⁸ Eisenberg and Mordechai 2019.

⁵⁰⁹ White and Mordechai 2020.

⁵¹⁰ Eisenberg and Mordechai 2019, 171-73.

on the 2019 *PNAS* article led by Mordechai, takes a more measured approach in considering a connection between the LALIA and the plague; that is, he does not all but extinguish it as a possibility.⁵¹¹ While Newfield mainly asks questions rather than providing deep analysis, he examines the subject more thoroughly than anyone has to date. He recognizes that the complexities of the plague cycle make it difficult to connect climatic variability as the impetus to underlying outbreaks. However, Newfield also recognizes the potential and implications that investigation in this specific area has for Justinianic Plague historiography. He advises: "to overcome any lingering suspicion that late antique plague-climate connections are simply coincidental, it will be essential to establish the various mechanisms through which climate could have influenced plague-carrying rodents and plague-transmitting arthropods to spur or hinder plague outbreaks in people."⁵¹² And it is from here that we embark on an investigation of whether sixth-century climate change was a catalyst in the emergence of the Justinianic Plague.

Written Evidence

The written evidence is (as always) not as copious as historians would like, but there is plenty to glean from it, particularly from the sources contemporary with our plague. There are seven important contemporary sources that discuss the first wave of the Justinianic Plague in detail. They are: Procopius, John of Ephesus, John Malalas, Gregory of Tours, Simeon Stylites the Younger, Evagrius Scholasticus, and

⁵¹¹ Newfield 2016.

⁵¹² Newfield 2016, 103.

Agathias.⁵¹³ All unquestionably witnessed the plague and, to varying degrees, had their own intimate experiences with it. There are other contemporary sources, such as Marcellinus Comes, who seem to have witnessed the pestilence, but their mentions are fleeting.⁵¹⁴ In the section, I also include epigraphic evidence (that in some cases is more clearly evidence of plague than in others) to help trace the first wave; this evidence does not, however, tell us anything qualitative. I do not analyse the inscriptions in detail, but only use them to follow potential paths of the plague in the subsection below.⁵¹⁵ There are also a few later sources that mention the first wave (as well as recurring waves), such as Theophanes and the *Chronicle of Seert*.⁵¹⁶ Then there is the Emperor's edict in 544 that declared the plague to be over.⁵¹⁷ And we have numerous sources mentioning the succeeding waves, which I do not give full treatment in this chapter.⁵¹⁸

Following the plague

In 2004, Stathakopoulos did a good job of updating the path of the Justinianic Plague's first wave, based on the written evidence, from that proposed by Biraben

⁵¹³ Procopius, *Persian Wars*, 2.22; John of Ephesus, in Pseudo-Dionysius of Tel Mahrē, *Chronicle*, 80; John Malalas, *Chronographia*, 18.90, p. 407, *Chronique de Michel le Syrien*, 235ff; Gregory of Tours, *Gregorii Episcopi Turonesis Libri historiarum X*, 4.31, 4.5; Evagrius, *Ecclesiastical History* 4.29, p. 178, 11-16: here Evagrius is mainly discussing the 591 outbreak in Antioch, but he refers to his experience with the first wave as a child.

⁵¹⁴ Marcellinus Comes, *Chronikon*, ad annum 543, p. 107.

⁵¹⁵ Gaza: Glucker 1987, 124-26; Negev: Kirk and Welles in Colt (ed.) 1962, 179-81; Tsafir 1988, 161; unclear epitaphs from Rome: De Rossi 1888 (in chronological order), 1.1452, 2.4287, 7.17624, 2.5088, 2.4289, 8.20839, 2.5087.

⁵¹⁶ Theophanes, *Chronographia* AM 6034, p. 22.

⁵¹⁷ *Novella* 122 in *CIC*, 3:592-93.

⁵¹⁸ Agathias, *Historiae* 5.10; Marius of Avenches, *Chronica*, ad annum 571; John of Biclaro, *Chronica*, ad annos 572-573; *Liber Pontificalis* LXV; Gregory of Tours, *Historia Francorum* 10.1; Paul the Deacon, *Historia Longobardorum* 3.23-24, 4.4, 4.14, 6.5; Evagrius, *Ecclesiastical History* 4.29; Theophylactus Simocatta, *Historiae* 7.15.2; Elias of Nisibis, *Opus Chronologicum*, ad annum 911; Gregory the Great, *Register epistularum* 9.232, 10.20; Nicephorus, *Breviarium* 8, 12, 67; Leontios, *Vie de Jean de Chypre* 24; Theophanes, *Chronographia* AM 6053, 6190, 6238; Severus, *History of the Partarchs* 18; Pope Zacharias to St. Boniface, *Regesta Pontificum Romanorum* 1:265; *Megas Chronographos* 17; *Chronicle of Zuqnin*, 184-89; for the Arabic sources on the last wave see: Conrad 1981, 300-06.

and Le Goff in 1969 (which was incomplete or incorrect).⁵¹⁹ The plague first reached the Mediterranean region in mid-July 541 at Pelusium, as Procopius tells us.⁵²⁰ From there it went both west and east, reaching Alexandria by September 541 the latest, and Gaza and three towns in the Negev (probably) between October and December, respectively.⁵²¹ In the first half of 542 parts of Syria were infected, including Antioch and Epiphinea (or Apameia), and Myra and (probably) Aphrodisias in Anatolia soon after.⁵²² Not long after, probably in the spring of 542, the plague reached the imperial capital at Constantinople.⁵²³ Most likely before that it wreaked havoc in Anatolia, as John of Ephesus tells us it struck multiple towns in the region, eventually heading east to infect the Persian Empire.⁵²⁴ Further west, the plague seems to have reached Sicily in late 542 and North Africa in early 543.⁵²⁵ It was in either late 542 or early 543 that the pandemic hit Spain; possibly brought there by merchant ship.⁵²⁶ At some point in 543 the plague reached Gaul and Italy, either at the coastline or from eastern Europe, and, of course, Rome soon followed.⁵²⁷ While we do not have written

⁵¹⁹ Biraben and Le Goff 1969, 1494-97; Stathakopoulos 2004, 113-16.

⁵²⁰ Procopius, *History of the Wars*, 2.22.6;

⁵²¹ Alexandria: John Malalas, *Chronographia*, 18.90, *Chronique de Michel le Syrien*, 235ff; Gaza: Glucker 1987, 124-26; Negev: Kirk and Welles 1962, 168, 179-81; Tsafirir 1988, 161; Stathakopoulos 2007, 101.

⁵²² Syria: Symeon Stylites the younger, *Vita* 69; Evagrius, *Ecclesiastical History*, 4.29. Myra: Nicholas of Sion, *Vita* 52. Aphrodisias: Roueché and Reynolds 1989, 137-141, inscription 86; this inscription, a dedication to the benefactor Rhodopaeus, cannot be definitively dated to the first wave, but that the inscription says he “drove away plague and famine” with the money he gave for the corn supply and the (re-)construction of Aphrodisias’ Hadrianic ‘Olympian’ baths, suggests it refers to the 540s, when both plague and climate change that devastated agriculture had occurred.

⁵²³ Procopius, *History of the Wars*, 2.22.9; John of Ephesus, in Pseudo-Dionysius of Tel Mahrē, *Chronicle*, 74-93; John Malalas, *Chronographia*, 18.92, Theophanes, *Chronographia* AM 6034.

⁵²⁴ John of Ephesus, in Pseudo-Dionysius of Tel Mahrē, *Chronicle*, 80; Procopius, *Persian Wars* 2.24.5-8; Stathakopoulos 2007, 101.

⁵²⁵ Duval 1956, 277-80; Manganaro 2001, 133; Stathakopoulos 2007, 101-2.

⁵²⁶ Kulikowski 2007, 150-52; *Consularia Caesaraugustana*, ad a. p.c. Brasili II=Victor Tunnensis *Chronicon*, 44.

⁵²⁷ Gregory of Tours, *History of the Franks* 4.5; Gregory of Tours, *Life of the Fathers* 6.6; Marcellinus Comes, *Chronicon*, ad annum 543, 107; *Inscriptiones Christianae Urbis* 1.1452, 2.4287, 7.17624, 2.5088, 2.4289, 8.20839, 2.5087

First Wave	AD 541-544
Second Wave	AD 558-561
Third Wave	AD 571-574
Fourth Wave	AD 591-592
Fifth Wave	AD 597-601
Sixth Wave	AD 618-619
Seventh Wave	AD 626-628
Eight Wave	AD 639
Ninth Wave	AD 669-673
Tenth Wave	AD 680
Eleventh Wave	AD 683-687
Twelfth Wave	AD 698-700
Thirteenth Wave	AD 704-706
Fourteenth Wave	AD 713-715
Fifteenth Wave	AD 718-719
Sixteenth Wave	AD 724-726
Seventeenth Wave	AD 732-735
Eighteenth Wave	AD 743-750

Table 6. Waves of the Justinianic Plague as identified by Dionysius Stathakopoulos.⁵²⁸

evidence for it, we know the plague reached Britain between 543 – 544 – based on archaeological evidence which will be discussed below.⁵²⁹ And, as mentioned,

⁵²⁸ Stathakopoulos 2007, 100-105.

Justinian's edict of 544 that proclaimed the pestilence to be over brings the first wave of the plague to a close.⁵³⁰ Now that we have established the first wave's path, let us delve into what else ancient authors said about the plague.

Procopius

Procopius, the main historical source for the reign of Justinian and legal advisor to general Belisarius during his reconquest of the west, is the first to mention the Justinianic Plague, and he is probably our most important source for it.⁵³¹ When the plague first broke out Procopius was in Constantinople, having returned from Italy at some point after Belisarius' siege of the Gothic capital at Ravenna the year before.⁵³² As mentioned, Procopius pinpoints the first outbreak (that he knew of) at Pelusium in Egypt, and then discusses where the plague spread:

*"It started from the Egyptians who dwell in Pelusium. Then it divided and moved in one direction towards Alexandria and the rest of Egypt, and in the other direction it came to Palestine on the borders of Egypt; and from there it spread over the whole world, always moving forward and travelling at times favourable to it. For it seemed to move by fixed arrangement, and to tarry for a specified time in each country, casting its blight slightly upon none, but spreading in either direction right out to the ends of the world, as if fearing lest some corner of the earth might escape it. For it left neither island nor cave nor mountain ridge which had human inhabitants...And this disease always took its start from the coast, and from there went up to the interior. And in the second year it reached Byzantium in the middle of spring, where it happened that I was staying at that time."*⁵³³

Procopius' description of the spread of the disease, which betrays the role of maritime transport ("*this disease always took its start from the coast ...*"), is validated by other sources, as shown above, spreading both west and east, and eventually reaching

⁵²⁹ Harbeck *et al.* 2013; Feldman *et al.* 2016; Keller *et al.* 2019.

⁵³⁰ *Novella* 122 in *CIC*, 3:592-93.

⁵³¹ Procopius, *History of the Wars*, 1.12.24.

⁵³² Procopius, *History of the Wars*, 2.28.

⁵³³ Procopius, *History of the Wars*, 2.22.6-7, trans. by H.B. Dewing and G. Downey.

Constantinople (Byzantium). He also lets the reader know just how far-reaching the pandemic was. However, statements such as, “spread over the whole world”, and the tone of general ruination which Procopius adopts, should of course be received with caution; historians of the time sometimes exaggerated calamities for effect, especially when those disasters happened during the rule of despised emperors, as we saw in Chapter 2 with Herodian and Cassius Dio writing under Commodus; in the case of Procopius and Justinian, Procopius makes known his loathing for Justinian in his *Secret History*.⁵³⁴ But, as we will see, Procopius is not the only ancient writer to describe the pestilence with such horror and helplessness. In that vein, he continues:

*“But for this calamity it is quite impossible either to express in words or to conceive in thought any explanation, except indeed to refer it to God. For it did not come in a part of the world nor upon certain men, nor did it confine itself to any season of the year, so that from such circumstances it might be possible to find subtle explanations of a cause, but it embraced the entire world, and blighted the lives of all men, though differing from one another in the most marked degree, respecting neither sex nor age. For much as men differ with regard to places in which they live, or in the law of their daily life, or in natural bent, or in active pursuits, or in whatever else man differs from man, in the case of this disease alone the difference availed naught. And it attacked some in the summer season, others in the winter, and still others at the other times of the year...then those who dwelt round about this land, whom formerly it had afflicted most sorely, it did not touch at all...those who encountered them [the afflicted] thought that they were struck by the man they had met in this or that part of the body, as it happened, and immediately upon seeing this apparition they were seized also by the disease.”*⁵³⁵

Here, Procopius reveals some epidemiology. He begins by, again, noting the devastation it caused, but, also, he describes very important details about its character, such as, the disease’s apparent lack of seasonality, its indiscriminate nature (“embraced the entire world”; “respecting neither sex nor age”), immunity of

⁵³⁴ Procopius, *Secret History*.

⁵³⁵ Procopius, *History of the Wars*, 2.22.3-11, trans. by H.B. Dewing and G. Downey.

survivors, and the ease with which the disease spread. Procopius' then goes on to describe the symptoms as follows:

*"They had a sudden fever, some when just roused from sleep, others while walking about, and others while otherwise engaged, without any regard to what they were doing. And the body shewed no change from its previous colour, nor was it hot as might be expected when attacked by a fever, nor indeed did any inflammation set in, but the fever was of such a languid sort from its commencement and up till evening that neither to the sick themselves nor to a physician who touched them would it afford any suspicion of danger. It was natural, therefore, that not one of those who had contracted the disease expected to die from it. But on the same day in some cases, in others on the following day, and in the rest not many days later, a bubonic swelling developed; and this took place not only in the particular part of the body which is called 'boubon,' that is, below the abdomen, but also inside the armpit, and in some cases also beside the ears, and at different points on the thighs."*⁵³⁶

Based on his description, fever appears to have occurred in all cases; and it does not seem to begin as a particularly debilitating fever, as many of the infected went about their business, while others who sought medical attention were not cautioned by their physicians that it might be a serious disease. Soon after, however, the fever worsened without warning. Procopius describes how at this stage the infected were unable to take food, which worsened one's already rapidly deteriorating condition.⁵³⁷ Delirium was common in those that did not fall into a coma.⁵³⁸ The illness then transitioned to the next phase, as the all-important buboes developed, signifying bubonic swelling. The buboes appeared in the places we would expect them – the groin ("below the abdomen"), armpits, and the thighs – leaving little room for doubt that the disease was bubonic plague. However, Procopius also claims that in some "the disease came on without warning and they died straight away", suggesting that bubonic plague outbreaks may have developed into the more acute pneumonic plague (more on this

⁵³⁶ Procopius, *History of the Wars*, 2.22.3-16-17, trans. by H.B. Dewing and G. Downey.

⁵³⁷ Procopius, *History of the Wars*, 2.22.27.

⁵³⁸ Procopius, *History of the Wars*, 2.22.19, 2.22.28.

below); and if they did not succumb straightaway, they died “after many days”, a vague detail.⁵³⁹ With the final symptom, Procopius explains that if the swelling buboes began to discharge pus then the infected had a better chance of survival, while if the buboes remained in their constant, swollen state the afflicted would most likely die.⁵⁴⁰

In the section immediately following in book II, Procopius describes the plague’s duration and its impact on the imperial capital and “in the Roman Empire at large.” He claims that the first wave lasted four months in Constantinople, causing extreme mortality that varied from 5,000 – 10,000+ dead each day.⁵⁴¹ Of course, we must not take his numbers at face value; both because historians of the time, as mentioned, tend to exaggerate and because it is unlikely that that many died per day for four months straight. However, Procopius’ point is clear: the plague killed a significant portion of the population. He describes a Constantinople littered with corpses and the difficulties families and the imperial government had with managing burials. He claims that families threw their dead “even into the tombs of others”, and that the situation became so destitute on the streets that Justinian (who caught and survived the “swelling of the groin”) tasked an imperial officer (Theodorus) with the duty of city-wide body-disposal.⁵⁴² Despite this, it was chaos, as fortification towers designated for body disposal and mass-grave trenches were brimming with corpses, causing an odour of decay to pervade the city.⁵⁴³ While these details are an account of conditions in the capital, Procopius claims that the pestilence similarly affected cities across the Empire. He provides information about other affected cities, such as the fact

⁵³⁹ Procopius, *History of the Wars*, 2.22.23, trans. by H.B. Dewing and G. Downey.

⁵⁴⁰ Procopius, *History of the Wars*, 2.22.37-38.

⁵⁴¹ Procopius, *History of the Wars*, 2.23.1-3.

⁵⁴² Procopius, *History of the Wars*, 2.23.3-10.

⁵⁴³ Procopius, *History of the Wars*, 2.23.6-11.

that the origin (so far as he knew) was Pelusium, and while there is no additional evidence that would corroborate that the plague began at the Egyptian port city, we do have evidence (discussed below) that validates Procopius' claim that the plague reached Alexandria and the Near East, which would be entirely consistent with a spread from Pelusium. Therefore, when he composed his *History of the Wars* (probably AD 545), there is no reason to doubt that Procopius was well informed about how "the Roman Empire at large" fared during the first wave, and thus whether the situation was generally the same in many places throughout the Empire as it was in Byzantium.

John of Ephesus (Pseudo-Dionysius of Tel-Mahre)

In his *Ecclesiastical History*, the Syrian deacon John of Ephesus notes that on his journey to gather accounts from holy men in Egypt, the Near East, and through Asia Minor to Constantinople, he saw that numerous towns were struck by the plague.⁵⁴⁴ Writing in Syriac, John claims that during "the year 855 [AD 543/544] of Alexander there was a great and mighty plague in the whole world in the days of Emperor Justinian".⁵⁴⁵ Stathakopoulos argues convincingly that John's years are incorrect; his accounts of where and when the plague strikes certain cities are accurate and corroborated by other accounts, such as Procopius' account and inscriptional evidence, but his dating is off by two years.⁵⁴⁶ John's most detailed account is of the outbreak at Constantinople, affirming the devastation described by Procopius. He

⁵⁴⁴ John of Ephesus, *Ecclesiastical History* = Pseudo-Dionysius of Tel-Mahre, *Chronicle Part III* in Witakowski 1996 (trans. by), 74-93.

⁵⁴⁵ Witakowski 1996, 74.

⁵⁴⁶ Stathakopoulos 2004, 279.

says that the plague also devastated Alexandria in 541, as well as Gaza, Askalon, and the Negev in the Near East around the same time. His description of how the plague passed from one place to another is colourful – not unusual for a late-antique leader of the church – probably meant to evoke a demonic nature, but again metaphorically suggestive of the role of shipping in the spread of the pandemic:

“When this plague was passing from one land to another many people saw shapes of bronze boats and (figures) sitting in them resembling people with their heads cut off. Holding staves, also of bronze, they moved along the sea and could be seen withersoever they headed. These figures were seen everywhere in a frightening fashion, especially at night. Like flashing bronze and like fire did they appear, black people without heads sitting in a glistening boat and travelling swiftly on the sea, so that this sight almost caused the souls of the people who saw it to expire.”⁵⁴⁷

That John of Ephesus writes about the plague so fantastically does not mean he did not witness it. His style is hyperbolic – written for a religious audience – but his account of where the plague struck is validated by other sources (above). Still on the headless sailors, but obviously talking about the plague’s spread, John claims:

“In this way they were seen proceeding to Gaza, Ashkelon, and Palestine and simultaneously with their appearance the beginning (of the plague) took place there. Also, (horrors) exceeding by far those previously narrated about the city of Alexandria took place from now on in the whole of Palestine, with the effect that villages and cities were left totally without inhabitants.”⁵⁴⁸

Soon after, John describes how a city on the Egyptian border lost all of its residents but for eight people.⁵⁴⁹ Even more so in John’s case should we be cautious when

⁵⁴⁷ Witakowski 1996, 77.

⁵⁴⁸ Witakowski 1996, 77.

⁵⁴⁹ Witakowski 1996, 77.

interpreting the description of mortality; while not impossible, it seems unlikely the plague alone cut the population of a city down to eight people; if we are to believe this account, flight probably contributed to the depopulation. John explains (in most cases second-hand) conditions throughout the Eastern Empire as follows:

*"When it was at its peak we went from Palestine to Mesopotamia and then came back again when the chastisement [plague] reached there also, as well as (going) to other regions – Cilicia, Mysia, Syria, Iconium, Bythinia Asia, Galatia, and Cappadocia, through which we travelled in terror (on our way) from Syria to the capital (during) the height of the plague."*⁵⁵⁰

John explains how his troupe saw many villages suffering, abounding with dead bodies and no place to put them. He notes that at this point the plague had not yet reached Constantinople, but rumours about it had; in fact, he claims that Constantinople had been hearing about the plague for one or two years before it finally arrived.⁵⁵¹ John seems to have arrived at the capital when the outbreak there was just beginning.⁵⁵² As it gained traction, he says:

*"...first it eagerly began to (assault) the class of poor, who lay in the streets. It happened that 5,000 and 7,000, or even 12,000 and as many as 16,000 of them departed (this world) in a single day. Since thus far it was (only) the beginning, men were standing by the harbours, at the crossroads and at the gates counting (the dead)... thus (the people of Constantinople) reached the point of disappearing, only few remaining, whereas (of) those only who had died on the streets...for in fact they were counted, over 300,000 were taken off the streets."*⁵⁵³

⁵⁵⁰ Witakowski 1996, 80.

⁵⁵¹ Witakowski 1996, 80-86.

⁵⁵² Witakowski 1996, 85.

⁵⁵³ Witakowski 1996, 87.

While we should be careful in trusting John's mortality numbers (as we should with any ancient author's), they *are* quite similar to Procopius' 5,000 – 10,000+ dying per day in the imperial capital. However, it is not unlikely that John read Procopius' *Wars*, as it was published more than 30 years before John wrote his *Ecclesiastical History*. Finally, John reveals bits of symptomology and epidemiology that are not dissimilar from Procopius' account:

*"Not only those who died but those who escaped sudden death (were struck) with this plague of swellings in their groins, with this disease which they called bubones, and which in our Syriac language is translated as 'tumours'. Both servants and masters were smitten together, nobles and common people impartially."*⁵⁵⁴

From John's description, like Procopius', it is clear that the disease was bubonic plague. He also notes the disease's indifference, affecting people of all classes alike. John then explains how the disease infected animals, including cattle and dogs, which plague has been known to do.⁵⁵⁵ But, more importantly, John says that they saw "even rats, with swollen tumours, struck down and dying."⁵⁵⁶ He goes on to describe Constantinople coming to a standstill: corpses flooded the streets; food imports stopped; markets were empty of all goods and people; and most typical daily activities, besides churchgoing, were halted.⁵⁵⁷ Whether or not John of Ephesus' exaggerated the number of dead, it is unlikely that he fabricated the general devastation caused by the plague.

⁵⁵⁴ Witakowski 1996, 87.

⁵⁵⁵ <https://www.cdc.gov/plague/transmission/index.html>, date accessed: September 19th, 2020.

⁵⁵⁶ Witakowski 1996, 87.

⁵⁵⁷ Witakowski 1996, 88-90.

John Malalas (AD 491 – 578), a Greek chronicler from Antioch, is another historian contemporary with the first wave of the Justinianic Plague. Given that John writes in the style of a civil servant, using “governmental terminology”, it is thought that he was an imperial official at Antioch.⁵⁵⁸ He probably moved to Constantinople soon after the Persians sacked Antioch in 540, and he was in the capital when the plague struck. He also seems to have been informed about the devastation it was causing elsewhere.

Unfortunately, his references are very brief and do not add anything to our knowledge of the pestilence’s symptomology, epidemiology, or impact. However, one of the passing references appears to indicate that the plague was affecting Egypt at large. In reference to a woman, that at the time Constantinopolitans thought was speaking “a lot of nonsense”, who had claimed a supernatural force (or the sea) was consuming whole cities (AD 541/542), John says:

“Everyone went in processions of prayer and chanted, ‘Lord, have mercy’, for reports were circulating that many cities had been swallowed up. “It was then that men died of the plague in Egypt, including Alexandria.”⁵⁵⁹

John seems to be connecting the two; he does not elaborate further. However, we can take from this that the plague may have been affecting a portion of Egypt. John may only be referring to the cities that we already know were struck: Alexandria and Pelusium. But if that is what he meant, why not just note those two cities? Two chapters later in his chronicle, the plague reaches Constantinople (AD 542):

⁵⁵⁸ Horrocks 2014, 179-180.

⁵⁵⁹ John Malalas, *Chronographia* 18.90, trans. by E. Jeffreys, M. Jeffreys, R. Scott.

“The plague lasted a while, so that there were not enough people to bury the dead. Some carried the corpses from their own houses on wooden litters and even so they could not manage. Some of the corpses remained unburied for days. Some people did not attend their own relatives’ funerals.”⁵⁶⁰

This is further evidence that in Constantinople the epidemic was devastating. Like the accounts of Procopius and John of Ephesus, people were dying at an alarming rate; so rapidly and in such great numbers that residents (and probably city officials) could not keep up with the burials. That Constantinopolitans were not present at their own families’ funerals (if there even were any) showcases not only significant demographic loss, but a metropolis arrested by disorder and despair.

Gregory of Tours

Our main contemporary source for the Justinianic Plague in the west is Gallo-Roman bishop and historian Gregory of Tours (AD 538–594).⁵⁶¹ Born in Clermont-Ferrand, in AD 574 Gregory became the bishop of Tours, 256 kilometres northeast of his hometown.⁵⁶² He discusses the first wave of the plague, as well as succeeding waves, most in *History of the Franks*, but also in his *Life of the Fathers* (his reference to the first wave in this work is nearly identical to the former).⁵⁶³ The only contemporary source to write in Latin, Gregory describes the first wave – even though he was only a young boy when it occurred – in reference to his paternal uncle, St. Gallus (or Gal I), Bishop

⁵⁶⁰ John Malalas, *Chronographia* 20, trans. by E. Jeffreys, M. Jeffreys, R. Scott.

⁵⁶¹ It should be noted, while Gregory is technically contemporary with the first wave of the plague, he was born in 538. At only 5 years old when the first wave hit Gaul, it is unlikely he remembered the first outbreaks as detailed as he writes about them. Thus, it is probable Gregory’s account of the first wave is second-hand and combines with his experiences with the subsequent waves, when he was an adult.

⁵⁶² Little 2007, 9-10.

⁵⁶³ Gregory of Tours, *History of the Franks* 4.5; Gregory of Tours, *Life of the Fathers* 6.6.

of Clermont (AD 489 – 553), who had taken over for Saint Quintianus as bishop in AD 527:

*“In Saint Gall’s time the plague raged in various parts of Gaul causing great swellings of the groin. It was particularly bad in the province of Arles, and Saint Gall was anxious not only for himself but more especially for his flock...As I have explained the plague raged through other parts of Gaul, but thanks to the prayers of Saint Gall it claimed no victims in Clermont-Ferrand.”*⁵⁶⁴

The events of the passage are dated to AD 543, making 543 the *terminus ante quem* for the plague reaching Gaul. There are two important points to take away from the passage: (1) Gregory’s bit of symptomology confirms that the disease of the west was bubonic plague; and (2) the pandemic spread to multiple areas within Gaul. He explicitly names Trier and Reims as towns the plague struck and came close to striking respectively, indicating, to Gregory’s knowledge, the northernmost extent of the plague.⁵⁶⁵ For Reims, 559 kilometres north of Clermont, Gregory again refers to it as “plague of the groin”, saying that it “ravaged the people [of the old Roman province] of First Germany”, but spared Reims.⁵⁶⁶ And for Trier, 279 kilometres northeast from Reims, Gregory claims that “the bubonic plague was cruelly assailing the population within the walls of the city”.⁵⁶⁷ That Gregory refers to the Bishop of Nantes becoming infected with plague, Little says, “suggests that the disease reached westward to the mouth of the Loire [river in France] where it flows to the Atlantic. This in turn suggests that the probable route for the plague between Gaul and both

⁵⁶⁴ Gregory of Tours, *History of the Franks* 4.5, trans. by L. Thorpe.

⁵⁶⁵ Gregory of Tours, *Life of the Fathers* 17.4; Gregory of Tours, *Glory of Confessors* 78; Little 2007, 10.

⁵⁶⁶ Gregory of Tours, *Glory of Confessors* 78, trans. by R. Van Dam.

⁵⁶⁷ Gregory of Tours, *Life of the Fathers* 17.4, trans. by E. James.

Cornwall and Ireland was through Nantes".⁵⁶⁸ Little also points out that the port at Marseilles is the most likely place where the plague first gained entry into Gaul, as Gregory mentions outbreaks from the subsequent waves (seemingly endemic outbreaks) happening there.⁵⁶⁹

Gregory turns out to be a most useful source for the extent of the plague in western Europe. While his writings are religiously charged, like those of John of Ephesus and John Malalas, the reader never feels that Gregory fabricated his accounts of plague. In some cases, he ties plague outbreaks to spiritual lessons, but there is no reason to disbelieve that he did in fact witness (or at least heard about) those outbreaks.

Symeon Stylites the Younger

A Christian saint and an ascetic for much of his life, Symeon Stylites lived for most of the sixth century and witnessed many of its calamities, including the Justinianic Plague.⁵⁷⁰ His *Life* was written by an anonymous hagiographer, who details the close relationships Symeon developed with notable people throughout the Empire.⁵⁷¹ Born in Antioch in AD 521, Symeon spent most of his life there and he witnessed the first wave of the plague strike his town in the summer 542. Symeon's hagiographer says about the plague:

"God is angered and strikes the people with plague. Symeon asks God for mercy; God says that their faults are numerous, but grants him the power to cure those who invoke Him through

⁵⁶⁸ Little 2007, 10; Gregory of Tours, *History of the Franks* 6.15.

⁵⁶⁹ Little 2007, 11; Gregory of Tours, *History of the Franks* 9.22, 10.25.

⁵⁷⁰ Parker 2016, 99.

⁵⁷¹ Parker 2016, 99.

Symeon's name. Many people call on him, see him appear to them and are cured. Lots of people are cured after lighting lamps in their houses in his name. Crowds of ill people come to him and are cured."⁵⁷²

The entry comes not long after an entry about the sack of Antioch by the Persians in AD 540, and has been dated to the year 542, which coheres with the other evidence that suggests that the plague arrived in and passed through Syria in the spring or summer of 542.⁵⁷³ The passage clearly has religious objectives, but it also implies that a good percentage of the infected overcame the disease. Symeon also witnessed the plague during the fourth wave in the year of his death (AD 592), in which he had an interaction with another important Antiochene. His hagiographer says:

"A *scholastikos*, Evagrius, from Epiphania but resident in Antioch, has blasphemous thoughts after he loses his daughter to plague but a pagan neighbor's children have not died. The Holy Spirit informs Symeon so he writes to Evagrius, who comes to him and begs for pardon."⁵⁷⁴ Just below, we take a closer look at the occurrence that Symeon's biographer is alluding to, as well as Evagrius' full experience with the Justinianic pandemic.

Evagrius Scholasticus

Evagrius, the *scholasticus* mentioned in the life of Simeon Stylites as having lost his daughter in the fourth wave of the plague, witnessed the first wave of the plague when he was just a boy, like Gregory of Tours. Evagrius Scholasticus (AD 535 – 594)

⁵⁷² *Life of Symeon Stylites the Younger* 69-70, trans. by L. Parker.

⁵⁷³ John of Ephesus: Witakowski 1996, 74-93; Evagrius Scholasticus, *Ecclesiastical History* 4.29; Nicholas of Sion, *Vita* 52.

⁵⁷⁴ *Life of Symeon Stylites the Younger* 233, trans. by L. Parker.

was a Syrian lawyer and intellectual born in Epiphania, eventually becoming an advisor to orthodox patriarch Gregory of Antioch.⁵⁷⁵ Evagrius' reference to the first wave comes amidst a description of the fourth wave hitting Antioch in 592:

"...the disease which struck and has remained strong and spread over the whole earth in this its 52nd year [594]...For, two years after the city of Antioch was captured by the Persians, a pestilent affliction took up residence...It was said, and still is now, to have begun in Ethiopia. In turn it overran the whole universe, leaving none among me without some experience of the disease...some cities were stricken to such an extent that they were completely emptied of inhabitants ...it took hold of some places at the beginning of winter, others while spring was in full swing, others in the summer time, and in some places even when autumn was advancing. And there are places where it affected one part of the city but kept clear of the other parts, and often one could see in a city that was not diseased certain households that were comprehensively destroyed...an almost complete extermination of mankind especially in the first or second year of the 15-year cycle. this indeed befell me...I was affected by what are called buboes while I was still attending the elementary teacher..."⁵⁷⁶

Evagrius says the plague started two years after the Persians sacked Antioch (AD 540), which coheres with the dating of the passage from the *Life of Symeon the Stylite the Younger*.⁵⁷⁷ Kislinger and Stathakopoulos argue convincingly that the plague arrived two years before the writing of the work above.⁵⁷⁸ Evagrius notes that it started in Ethiopia, but this could be a literary trope passed down from Thucydides as we saw in chapters two and three; however, this reference might indicate something else:

Evagrius' account that the plague spread from Africa may be independent from Procopius', since Procopius mentions Pelusium specifically, whereas Evagrius seems to have heard that, more broadly, it came from Africa, and thus chose the area that most historians choose as disease-ridden (due to Thucydides), Ethiopia; or he may have in fact heard that it spread from Ethiopia, which is actually quite possible (more

⁵⁷⁵ Whitby 2000, xvi-xvii; Little 2007, 9.

⁵⁷⁶ Evagrius Scholasticus, *Ecclesiastical History* 4.29, trans. by Michael Whitby.

⁵⁷⁷ *Vita* of Symeon Stylites the younger 69.

⁵⁷⁸ Kislinger and Stathakopoulos 1999, 93 n. 109; Stathakopoulos 2004, 323-24.

on this below). Like the other accounts, Evagrius also notes the widespread devastation caused by the plague, as well as its seasonal indifference (also mentioned by Procopius). He explains how in some cities certain areas were infected while other areas were unaffected, a new piece of epidemiology. And, lastly, he explains that he himself contracted the disease during the first wave when he was still in school. That means the plague reached either Epiphania (Hama, Syria) or Apameia (55km northwest of Hama), his hometown and the town where he went to school respectively; the latter seems most likely, since he specifically mentions school.⁵⁷⁹

Agathias

The poet and historian Agathias of Myrina (Asia Minor) is our last contemporary source for the first wave of the Justinianic Plague. Agathias writes about the plague mainly from his experience with the second wave in Constantinople (AD 558).

However, he was an adolescent during the first wave, so we take his account to represent his experience with both waves; especially since he claims that the plague “had never really stopped” between the first two waves. His account says:

“During that year at the beginning of spring a second outbreak of plague swept the capital, destroying a vast number of people. From the fifteenth year of the reign of the Emperor Justinian when the plague first spread to our part of the world it had never really stopped, but had simply moved on from one place to another, giving in this way something of a respite to those who had survived its ravages. It now returned to Constantinople almost as though it had been cheated on the first occasion into a needlessly hasty departure. People died in great numbers as though seized by a violent and sudden attack of apoplexy. Those who stood up to the disease longest barely lasted five days. The form the epidemic took was not unlike that of the earlier outbreak. A swelling in the glands of the groin was accompanied by a high nonintermittent fever which raged night and day with unabated intensity and never left its victim until the moment of death. Some experienced no pain or fever or any of the initial symptoms but simply dropped dead while about their normal business at home or in the street or wherever they

⁵⁷⁹ Stathakopoulos 2004, 283.

happened to be. People of all ages were struck down indiscriminately, but the heaviest toll was among the young and vigorous and especially among the men, women being on the whole much less affected."⁵⁸⁰

Agathias notes that the plague hit Constantinople in AD 542 and moved from town to town, staying alive with seemingly small outbreaks until fifteen years later large outbreaks occurred in Syria, Mesopotamia, Cilicia, and again at Constantinople, resulting in the so-called second wave.⁵⁸¹ Agathias mentions residents that died suddenly of apoplexy, or "dropped dead", similar to the sudden death described by John of Ephesus.⁵⁸² This could be a bubonic outbreak that evolved into a pneumonic outbreak; it must have started as bubonic since two lines later Agathias describes the disease as "a swelling of the glands of the groin" (more on the different forms of plague below). He says the characteristics of this outbreak were the same as the first outbreak, and that it attacked people of all ages and gender, but particularly affected young men. Epidemiologically this makes sense. Sixteen years after the first outbreak a new group of young adults, previously unexposed to the disease, would have been susceptible; unlike a proportion of those older who had probably developed a level immunity to the bacterium by living through the first wave. And that young men were hit the hardest is explained by the fact that they were out of the house more often, mingling in markets and ports, and were thus in a more vulnerable position for the transmission of plague.

⁵⁸⁰ Agathias, *Histories* 5.10, trans. by J.D. Frendo.

⁵⁸¹ Stathakopoulos 2007, 102; Theophanes, *Chronographia* AM 6050 & 6053; John Malalas, *Chronographia* 18.120; van den Ven, *La vie ancienne de S. Syméon* 126-29; Conrad 1986, 147-48.

⁵⁸² Witakowski 1996, 87.

Additional written evidence

We have a few additional sources for the first wave, but their references are either brief or many years removed from the first wave. Marcellinus Comes (6th century) wrote, under the year 543, “A great pestilence ravaged the land of Italy, and also the Orient and Illyricum which had been already similarly affected.”⁵⁸³ Thus, Marcellinus’ evidence extends the boundaries of the first wave to Italy and Illyricum. Theophanes the Confessor (AD 758/760 – 817) mentions the first wave in his *Chronicle*, simply saying that in AD 542 “the great plague broke out in Byzantium”.⁵⁸⁴ Victor of Tunnuna claims that the world was devastated by a pestilence of the groin.⁵⁸⁵ Stathakopoulos explains that Victor probably experienced the plague in Tunnuna, the North African city where he was bishop, which would confirm that the first wave reached North Africa; however, Victor is not specific.⁵⁸⁶ Victor also notes that the first wave hit Spain, as does an anonymous Spanish annotator who quotes from Victor’s *Chronicon*.⁵⁸⁷ Lastly, the *Chronicle of Seert* (probably 9th-century; anonymous) says the plague wreaked immense havoc in Alexandria, and claims a great famine immediately followed.⁵⁸⁸

Archaeological Evidence

Tracking an ancient pandemic using archaeology is difficult; the evidence is minimal. That does not mean there is no evidence. The closest we get to direct, unquestionable archaeological evidence for pandemics is aDNA evidence of the suspected pathogen

⁵⁸³ Marcellinus Comes, *Chronicle*, ad annum 543, trans. by B. Croke.

⁵⁸⁴ Theophanes, *Chronographia*, AM 6034, trans. by C. Mango, R. Scott, and G. Greatrex.

⁵⁸⁵ Victor of Tunnuna, *Chronicon*, ad a. 542.

⁵⁸⁶ Stathakopoulos 2004, 290.

⁵⁸⁷ Hartmann 2001, 44; Kulikowski 2007.

⁵⁸⁸ *Chronicle of Seert*, 32, trans. by A. Scher.

in skeletal remains (usually dental pulp) from a burial or mass grave dated to the period of the pestilence in question. Fortunately, researchers of the Justinianic Plague had an idea of what the pathogen was (thanks to the written evidence), and thus tested for the bacterium that causes bubonic plague (*Y. pestis*) in skeletal remains from mass graves dated to the sixth century, yielding positive results for multiple graves.

However, we have a number of other multiple burials and mass graves from the sixth century in which skeletal remains did not test positive for *Y. pestis*, but archaeologically the graves exhibit characteristics of a “plague pit” (e.g. hasty, unorganised burials). While they may be dated to the period of our plague and resemble a plague pit, they cannot securely be considered plague pits without the scientific evidence to prove it. It is probable that many *Y. pestis*-negative mass and multi-burial graves are in fact from the Justinianic pandemic, but the pathogenic evidence has not survived the nearly 1,400+ years; as, currently, the chances of recovering aDNA from ancient remains is low.⁵⁸⁹ Alternatively, those in the grave may have been killed by another pathogen, or something else entirely. In any event, without positive scientific evidence from mass graves we can only speculate on what caused them.

Then we have common indirect archaeological evidence, or proxy evidence, used as indicators of economic decline and/or demographic loss, and thus potential evidence of a pandemic; such as, declines in building and inscriptions, site contraction or abandonment, lead pollution data as a proxy for mining activity, and archaeobotanical evidence that indicates cessation from agricultural practices. These common types of indirect evidence will not be considered in this section, as they are

⁵⁸⁹ McCormick 2015, 346.

capable of multiple interpretations and do not point unambiguously to plague. This section focuses mainly on multi-burials and mass graves, giving most attention to graves containing evidence of *Y. pestis*.

Aschheim, Bavaria, Germany

In late antiquity, Aschheim was a small, rural town in the district of modern-day Munich. Inhabited from the late-fifth century AD until the late-seventh century (although population decline seems to start in the latter half of the sixth century), it is suspected that this inland village had no more than 70 inhabitants at any point during its occupation.⁵⁹⁰ In 1997, excavation uncovered 456 burials, including 28 datable multi-burial graves (six burials of three or more individuals with the remaining 22 being dual burials).⁵⁹¹ Five years later, three more multiple-burials were discovered just 100 metres away from the central excavation area at Aschheim.⁵⁹² In 2005, an aDNA study confirmed the presence of *Y. pestis* in the remains of two skeletons from Aschheim dated to the second half of the sixth century AD: a dual burial of one adult female and one juvenile female, presumably mother and daughter.⁵⁹³ Given that these were the only two skeletons tested, Aschheim showed promise for further research. In 2013, a larger aDNA study was undertaken at Aschheim in which 19 individuals from four different multi-person graves were tested.⁵⁹⁴ Eight of the 19 skeletons returned positive results for *Y. pestis*. Seven of the eight burials that tested positive were

⁵⁹⁰ Staskiewicz 2007, 35-53; Dannheimer and Diepolder 1988, 116-17; McCormick 2016, 346. For sixth-century population decline: McCormick 2016, 1006.

⁵⁹¹ McCormick 2016, 1010.

⁵⁹² McCormick 2016, 1010.

⁵⁹³ Wiechmann and Grupe 2005, 48-55.

⁵⁹⁴ Harbeck *et al.* 2013.

radiocarbon dated (Table 7).⁵⁹⁵ The results indicate that these individuals succumbed to plague in one of the first waves of the Justinianic pandemic. It is hard to disagree with McCormick's point that the molecular evidence at Aschheim indicates that the Justinianic Plague did in fact reach places far from the Mediterranean's shores.⁵⁹⁶

Skeleton	Raw Date	Calibrated date (2 Sigma, or 95.4% confidence)
A58	1519-1406 bp	431-544 cal AD
A66	1420-1380 bp	530-570 cal AD
A76	1507-1384 bp	443-566 cal AD
A77	1420-1380 bp	530-570 cal AD
A105	1420-1380 bp	530-570 cal AD
A120	1515-1391 bp	435-631 cal AD
A166	1420-1380 bp	530-570 cal AD

Table 7. *Y. pestis*-positive skeletons from the ancient/mediaeval cemetery at Aschheim, Germany.

Along with the written evidence, the results from the 2005 and the 2013 studies confirm that *Y. pestis* was responsible for the Justinianic Plague. But in 2014, new genealogical evidence for the Justinianic strain was revealed. The study led by David Wagner analysed two skeletons from Aschheim (A76 and 120) that had already tested positive for *Y. pestis* in Harbeck and colleagues' research. They retested the remains using a "modified phenol-chloroform method", confirming the findings in the study conducted by Harbeck in 2013.⁵⁹⁷ Radiocarbon dating for these skeletons (the same as

⁵⁹⁵ Harbeck *et al.* 2013, Table 1.

⁵⁹⁶ McCormick 2015, 346.

⁵⁹⁷ Wagner *et al.* 2014, 2-6.

listed in the study led by Harbeck) are calAD 443-566 for A76 and calAD 435-631 for A120, again, indicating death occurred in one of the first two waves for the former, and one of the first eight for the latter. But, more importantly, they enriched the DNA from the remains to sequence a genome. With the new genome, they constructed a new phylogenetic tree that showed a novel Justinianic branch. Their results revealed that the Justinianic lineage is an independent strain from the strain that caused the Black Death (AD 1346 – 1353), indicating that the Black Death did not emerge from a reservoir established during the Justinianic Plague. The archaeological evidence from the mass grave at Aschheim not only further proves that the Justinianic pandemic reached central Europe, but also shows that rodents are important carriers of plague, capable of spreading the bacillus across Eurasia on more than one chance occasion.

By investigating the remains from Aschheim scientifically, useful evidence was uncovered that changed the historiography of the Justinianic pandemic, and there are still other remains at Aschheim that could be examined similarly. Even though there are a number of burials that did not test positive for *Y. pestis*, at Aschheim and elsewhere, it does not necessarily mean that those individuals did not succumb to plague; nor should many negative tests discourage comparable investigations at archaeologically similar sites. McCormick, who discusses the Aschheim burials in detail in his catalogue of mass graves, thinks that while the positive tests do not “prove that all or most of the individuals buried in the multiple graves succumbed from bubonic plague or other sicknesses that may have occurred with it, the chronological coherence suggests a plausible association “. He believes that “given the low rates of success that are normal in recovering authentic aDNA”, the positive tests point to the likelihood that more of the multiple burials were victims of the plague,

even if they did not test positive for *Y. pestis*. Again, we cannot claim the existence of plague where science does not confirm it. But the likelihood of its wider extent becomes greater in areas that yield positive tests.

Altenerding, Bavaria, Germany

Just 23 kilometres northeast of Aschheim sits the ancient site at Altenerding. The town was inhabited from around the fifth century AD until the last third of the seventh century, and with 1,521 graves of 2,200 to 2,300 skeletons attested at Altenerding, it marks the largest Merovingian burial ground in southern Germany.⁵⁹⁸ Seventeen of the graves dated between AD 470 and 700 were dual burials, and no burials contained more than two bodies.⁵⁹⁹ In 2016, Michal Feldman and colleagues tested the remains (all of which were teeth) of 20 individuals – ten double burials – resulting in two of the ten skeletons testing positive for *Y. pestis*.⁶⁰⁰ It may not be a coincidence that the two individuals who produced positive tests, an adult male and an adult female (with the latter yielding robustly positive results), were from the same burial; possibly a couple. Radiocarbon dating indicated they died between AD 426 and 571 cal AD, and the grave's material remains suggests the burial took place around AD 530 – 570.⁶⁰¹ The aDNA evidence and dating indicate that the individuals probably died in one of the first two waves of the Justinianic pandemic (AD 541–544 or 558–561).

As mentioned in the discussion on Aschheim, the rate of success for extracting aDNA from skeletal remains is low. Therefore, that two individuals seem to have died

⁵⁹⁸ Losert and Pleterski 2003.

⁵⁹⁹ McCormick 2016, 1008.

⁶⁰⁰ Feldman *et al.* 2016a.

⁶⁰¹ Feldman *et al.* 2016, 2912.

during the pandemic, and that they are surrounded by other multi-burials also dated to around the time of the pandemic, hints that many more may have died from the disease. McCormick believes, since mass graves are absent from Altenerding, it “confirms that archaeological signals of a crisis mortality can be subtle”; something we should consider in future investigations.⁶⁰² With that said, since Altenerding had a population estimated only to have been around 250 and 300 individuals at any point in its two-century existence, the number of burials uncovered amounts to a notable portion of its people. If we were to speculate, there were probably around 7 generations that lived through ancient Altenerding’s existence (assuming a high-end generational span of 30 years). Considering these 2,200 skeletons attested (the low number), that equates to 314 deaths per generation. Obviously, that is not to say that all, or even most of the 2,200 burials are from the plague. But this type of generational mortality suggests that pandemic disease may have caused a large portion of these deaths. It is plausible that based on the striking ratio of graves to population, aDNA evidence, and dating, that the first wave and possibly succeeding waves of the Justinianic pandemic struck the village at Altenerding and may have helped cause its disappearance.

Evidence from Britain, France, Spain, and Germany

Instead of listing the following sites individually, for this subsection I refer to the recent aDNA study conducted by Marcel Keller and colleagues which tested remains

⁶⁰² McCormick 2016, 1005.

from multiple sites dated to our period, yielding numerous positive results for *Y. pestis*.

The study led by Keller in 2019, revealed that skeletal remains from sites in Germany, France, Spain, and Britain yielded positive results for *Y. pestis* (Fig. 52). Of the 140 samples dated to the Justinianic pandemic that were tested, 23 tested positive for *Y. pestis* aDNA.⁶⁰³ The study is most important to research on the Justinianic Plague, as, archaeologically, it confirmed the pandemic reached two more regions of the late-antique world in Spain and Britain, widened its extent in Germany, and definitively confirmed that early waves struck France. France is not new to producing positive results for *Y. pestis* from graves dated to the Justinianic Plague. In 2004, Michel Drancourt and his team yielded positive tests for *Y. pestis* from skeletal remains from two ancient grave sites in France: Vienne and Poitiers.⁶⁰⁴ However, the team's results were criticised as inadequate due to "methodological limitations" and poor anti-contamination methods.⁶⁰⁵ Thus, the evidence presented by Keller and his collaborators marks the first accepted evidence of the Justinianic pandemic in France.

For France, Keller and company tested skeletal remains from Lunel-Viel Les Horts and Lunel-Viel Quartier, both in the south of France, and Saint Doulchard le Pressoir in central France; however, the dating for the latter location (7th to 12th centuries) most likely places it outside the realm of our pandemic. At Lunel-Viel Les

⁶⁰³ Keller *et al.* 2019, 12365.

⁶⁰⁴ Drancourt *et al.* 2004.

⁶⁰⁵ Gilbert *et al.* 2004; Vergnaud 2005, 1317-18.

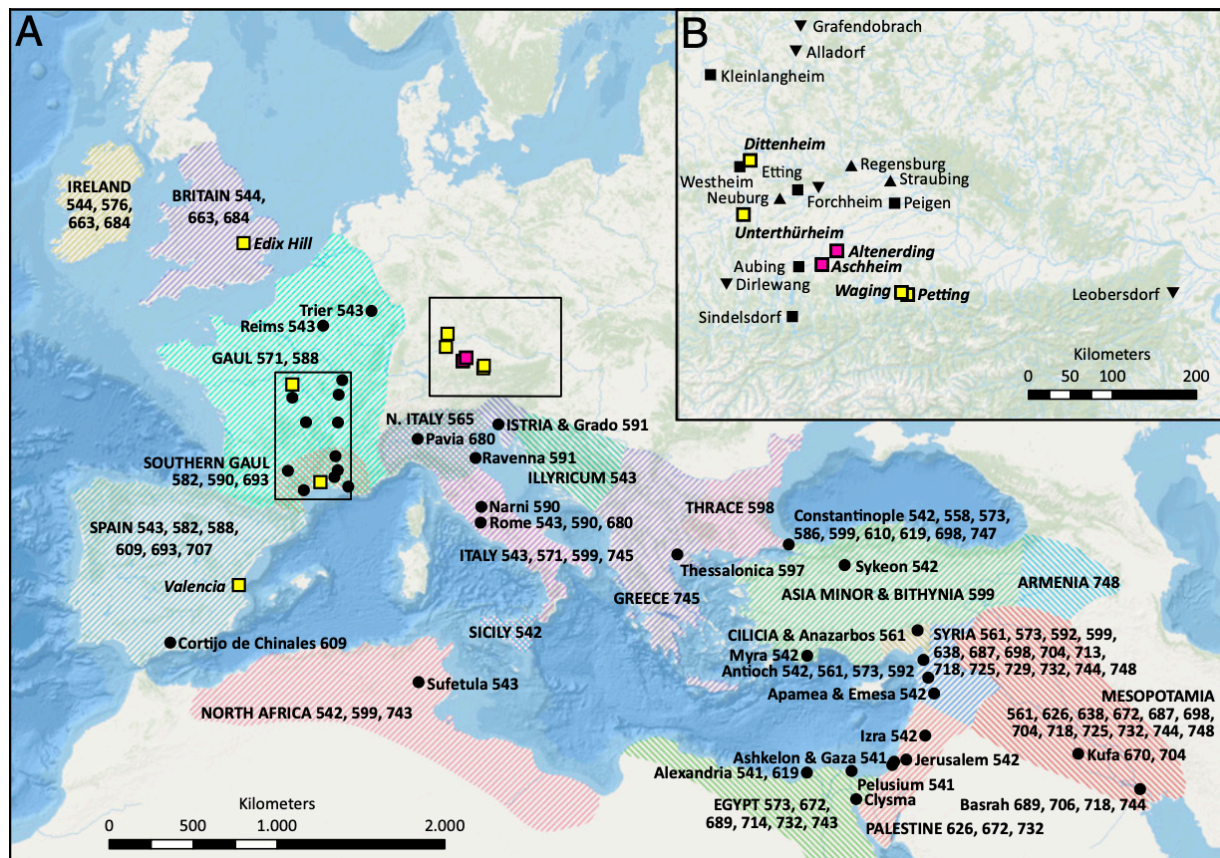


Figure 52. The geographic extent of the Justinianic Plague. A: The Mediterranean world. B: An enlargement of central Europe (Germany) where multiple sites had skeletal remains that tested positive for *Y. pestis*. Yellow boxes indicate sites with *Y. pestis*-positive remains published by Keller *et al.* 2019; pink boxes represent pre-2019 *Y. pestis*-positive sites (e.g. Harbeck *et al.* 2013). Black dots are the written evidence for the Justinianic Plague. (Keller *et al.* 2019, 12366)

Horts, they tested one double burial, but the results were negative. They had success at Lunel-Viel Quartier, testing two trenches containing eight individuals in total (testing sixteen teeth altogether), and yielding *Y. pestis*-positive results for three individuals (six teeth).⁶⁰⁶ Keller and colleagues note that the physical aDNA evidence from Lunel-Viel “are clearly not associated with the initial outbreak in 541 – 544 but rather subsequent ones”, due to the notable inconsistencies in the genome compared with strains confirmed from the first outbreak. They suggest that Lunel-Viel was

⁶⁰⁶ Keller *et al.* 2019, 12365.

probably affected by the many epidemics recorded in the south of France by Gregory of Tours (e.g. Marseilles and Arles).⁶⁰⁷

While the archaeology might not in itself definitively prove that southern France was struck by the first wave, given the outbreak recorded at Arles by Gregory of Tours, and the numerous subsequent outbreaks also recorded by Gregory at Marseilles from the mid-sixth century onward, it seems highly likely that the first wave did strike the region and we just have yet to uncover physical evidence for it. Recalling Gregory's reference to the first wave hitting central France in AD 543, it is unlikely that if the plague reached central France it was not via Marseilles, since it was the main maritime trading hub of the area (as suggested by Little); especially since, according to Gregory, succeeding waves struck Marseilles and other towns in the region; and now we have archaeological evidence in the area (Lunel-Viel) for those subsequent waves.⁶⁰⁸ In addition, Keller and colleagues obtained 11 positive tests out of 26 samples tested from Saint-Doulchard (central France). Although they date the remains to later than the Justinianic pandemic, the evidence might indicate that a focus was established from the first wave, especially when considering the account by Gregory of Tours of the plague hitting central France. Therefore, while the archaeological/aDNA evidence for the first wave is not yet consistent with the written evidence, it is probable that, along with central France, the initial outbreak did hit Marseilles and elsewhere in southern France (other than Arles), after which reservoirs were established, explaining the above archaeological evidence and the multiple outbreaks recorded by Gregory.

⁶⁰⁷ Keller *et al.* 2019, 12369.

⁶⁰⁸ Gregory of Tours, *History of the Franks*, 4.5. On the first wave probably hitting Marseilles: Little 2007, 11; and succeeding waves: Gregory of Tours, *History of the Franks* 9.22, 10.25.

The remains from a Visigothic cemetery in Valencia yielded only one positive test from nearly double the number samples as those from France; but nevertheless, the positive sample confirms that the plague reached Spain. Keller and his collaborators tested 36 samples from multiple collective burials throughout the cemetery. The sample that tested positive for *Y. pestis* came from Tomb 41, which contained at least 15 individuals who appear to have been buried hastily.⁶⁰⁹ Radiocarbon dating for the positive individual revealed a range between AD 432 – 610. The positive test and the material evidence led the researchers to conclude that death occurred during the first wave – although, in fact, based on the radiocarbon dating, any of the first five waves would be possible (cf. Table 6). This is our first evidence of the Justinianic pandemic in Iberia.

Further north, in Britain, aDNA analysis of remains from an Anglo-Saxon cemetery at Edix Hill (near Cambridge) returned positive tests for the plague bacillus. The cemetery, containing 149 individuals in 115 graves, 18 of which were multiple burials, is dated by Keller and colleagues to between AD 500 and 625.⁶¹⁰ Of the 22 samples tested from the same number of individuals, four returned positive results for *Y. pestis*; two were single burials and two were from two separate double-burial graves. Radiocarbon dating produced a range of cal.AD 474 – 637.⁶¹¹ The dating is further constrained archaeologically, with all of the *Y. pestis*-positive graves falling between AD 500 and 550 on the basis of grave goods and cemetery stratigraphy.⁶¹² Based on both the archaeological dating and the genomic results, which indicate that

⁶⁰⁹ Keller *et al.* 2019, supplemental information, 16-17, 42.

⁶¹⁰ Calibrated at sigma 2 (95.4% confidence). Keller *et al.* 2019, supplemental information, 9-10.

⁶¹¹ Keller *et al.* 2019, supplemental information, 80.

⁶¹² Keller *et al.* 2019, supplemental information, 39 & 80.

the strain is “rather basal within the clade”, they conclude that the individuals most likely succumbed during the first wave; probably around 544.⁶¹³ Keller and his collaborators propose that the first wave spread through Gaul to the edge of western Europe, then crossed the English Channel to the British Isles. They note, like McCormick in 2016 (who is an author on the 2019 paper), the fact that two of their positive tests from Edix Hill were single burials shows the limitations of solely investigating multiple burial graves. However, that the other two positive tests were from double burials surrounded by others of the same type, suggests that simultaneous double burials “are indeed good indicators for epidemic events.”⁶¹⁴ The evidence at Edix Hill illustrates the northward and westward reach of the Justinianic pandemic’s first wave.

The investigation by Keller and his colleagues on four sites in Germany yielded results that seem to tie two burials directly to the outbreaks at Aschheim and Altenerding (Fig. 52). The researchers tested Bavarian remains from multi-burials at Dittenheim, Petting, Unterthürheim, and Waging. At Dittenheim and Petting three of seven and three of nine samples respectively tested positive for *Y. pestis*. The phylogenetic results, indicating notable evolution of the bacillus, and the material remains suggest that the individuals died during one of the subsequent waves.⁶¹⁵ At Unterthürheim, five of seven individuals yielded positive results, and one of 12 at Waging. Dating for Unterthürheim and Waging places the remains somewhere in the sixth century, but, more intriguingly, phylogenetic analysis showed that the reconstructed genomes were identical to those from Aschheim and Altenerding,

⁶¹³ At sigma 2; Keller *et al.* 2019, 12368-69.

⁶¹⁴ Keller *et al.* 2019, 12369.

⁶¹⁵ Keller *et al.* 2019, 12369.

leading Keller and collaborators to determine that “these four can be attributed to the same epidemic event.”⁶¹⁶ The evidence from Altenerding, Aschheim, Unterthürheim, and Waging indicate, unequivocally, that Bavaria was struck hard by the plague; while evidence from the same region at Dittenheim and Petting, which points to one of the succeeding outbreaks, might indicate that a focus was established during the initial outbreak of 541 – 544.

While we do not have a plethora of direct archaeological evidence for the Justinianic Plague, its geographical range – Germany, France, Spain, Britain – helps demonstrate the pandemic’s extent; and, fortunately for historians, the evidence comes from regions with the least documentation. Still absent is direct archaeological evidence from Africa and the East; it should be noted that aDNA survives less well in hot and arid climates. Fortunately, the written evidence helps fill those gaps. As aDNA analysis progresses, so do archaeological motivations. This should undoubtedly lead to more revelations about this pandemic. At this stage we have enough to diagnose the disease.

Diagnosis

In 1987, Karl-Heinz Leven, a medical doctor, proposed true plague as the probable etiological agent behind the Justinianic pandemic, based on the descriptions by Procopius, John of Ephesus, and Evagrius. He thought their descriptions and the devastation reported were enough to indicate bubonic plague; additionally, he considered Evagrius’ descriptions of some Constantinopolitans succumbing to

⁶¹⁶ Keller *et al.* 2019, 12369.

sudden death to demonstrate pneumonic plague.⁶¹⁷ By the early 21st century, the case for bubonic plague as the cause had gained serious traction. Based on the papers in Little's volume from 2007, the consensus at the conference in Rome in 2001 must have been that bubonic plague was the leading candidate. As the contributors edited their papers for the volume, Stathakopoulos (who was a contributor at the conference) made plain in his volume from 2004: "A comparison between the modern data and that recorded by the Byzantine sources leaves no doubt to the fact that the disease behind the Justinianic Plague was in fact true plague."⁶¹⁸ Six years after the Rome conference, the volume was published, and most contributors subscribed to the bubonic (and for some pneumonic) theory. However, their stances may have been nudged in that direction by the study led by Drancourt in 2004, which revealed positive aDNA evidence in France but was later questioned due to poor anti-contamination protocols.⁶¹⁹ Wiechmann and Grupe's paper from 2005 identified the presence of *Y. pestis* in ancient remains dated to the period of our pandemic (at Aschheim), and their results were confirmed by a deeper analysis by Harbeck and colleagues in 2013.⁶²⁰

Echoing Stathakopoulos, based on the written evidence, and now the scientific evidence, there can be no room for doubt that the etiological agent that caused the Justinianic Plague was the bacterium *Y. pestis*, probably in the form of bubonic plague, but possibly also in some cases pneumonic plague. The tell-tale symptom of bubonic plague, buboes and/or swelling of the groin, is mentioned by

⁶¹⁷ Leven 1989, 135-38; Stathakopoulos 2000, 269.

⁶¹⁸ Stathakopoulos 2004, 111.

⁶¹⁹ Drancourt *et al.* 2004; Gilbert *et al.* 2004; Vergnaud 2005, 1317-18.

⁶²⁰ Wiechmann and Grupe 2005, 48-55; Harbeck *et al.* 2013, 319-26; Eisenberg and Mordechai 2019, 170.

Procopius, John of Ephesus, Gregory of Tours, Evagrius, Agathias, and Victor of Tunnua, who not only help us diagnose the disease, but also help relate the plague's geographical extent.⁶²¹ Apoplexy, or, sudden death, is described by Procopius, John of Ephesus, Evagrius, and Agathias, possibly indicating the pneumonic form of plague.⁶²² The archaeological/aDNA evidence from Germany, France, Spain, and Britain now confirms that the bacterium *Y. pestis* was the cause of the pandemic in Europe at least. We have yet to find remains east of Germany that have tested positive for *Y. pestis*, but the fact is, most of our written evidence comes from the East, and given the detail and similarities amongst the descriptions of the disease, it is hard to deny true plague as the disease responsible for the outbreaks starting in the 540s in the East (Asia Minor and the Near East) and in Africa (Egypt and North Africa). Therefore, *Yersinia pestis*, the bacterium that causes plague, was the culprit behind the Justinianic pandemic.

Epidemiology of Plague

Plague is a disease mainly of rodents of which humans are innocent bystanders.⁶²³ It is transmitted between rodents via infected *Y. pestis*-vectors, typically fleas, but the bacterium can sometimes be transferred through cannibalism, and less so via contaminated soil.⁶²⁴ Plague occurs in wild and domestic settings. Wild, or sylvatic, plague exists in natural/rural areas, where it is enzootic amongst wild rodents – i.e.

⁶²¹ Procopius, *History of the Wars*, 2.22.3-11; John of Ephesus, Witakowski 1996, 87; Gregory of Tours, *Glory of Confessors* 78, trans. by R. Van Dam; Gregory of Tours, *History of the Franks* 4.5, trans. by L. Thorpe; Evagrius Scholasticus, *Ecclesiastical History* 4.29, trans. by Michael Whitby; Agathias, *Histories* 5.10, trans. by J.D. Frendo; Victor of Tunnuna, *Chronica*, ad a. 542.

⁶²² Procopius, *History of the Wars*, 2.22.23, trans. by H.B. Dewing and G. Downey; *Histories* 5.10, trans. by J.D. Frendo.

⁶²³ Dennis *et al.* 1999, 11; Gage and Kosoy 2005, 506; Audoin-Rouzeau 2003, 33.

⁶²⁴ Dennis *et al.* 1999, 11.

it exists naturally without causing epizootic outbreaks amongst other mammals or epidemics in human populations.⁶²⁵ Domestic plague can occur when natural foci are disrupted by climatic, environmental, or anthropogenic forcing, resulting in the transfer of plague's etiological agent, the bacterium *Yersinia pestis*, from wild rodents to rodents that live amongst humans (peri-domestic). Once a group of peri-domestic rodents become infected, epizootic events can occur in which many urban rodents contract plague, die, and their fleas find new rodent hosts. Epidemics occur when large groups of urban rodents succumb to plague, and, in search of new hosts, their infected fleas jump to humans, biting the new host and transmitting *Y. pestis* to the human population.⁶²⁶

Plague can take three forms in humans: bubonic, septicaemic, or pneumonic. The most common is bubonic. As described above, bubonic plague occurs when a flea carrying *Y. pestis* bites a human and passes the bacterium into our bloodstream; however, the bacterium can also enter through an open wound. The infection spreads to the lymphatic system, externally resulting in swollen lymph nodes (buboes) in the groin, and sometimes the armpits and neck. The incubation period of bubonic plague is 2 – 6 days.⁶²⁷ Afterwards, internal symptoms develop which include high fever, malaise, chills, and pain in the swollen lymph nodes. When untreated, the mortality rate for bubonic plague is between 40–60%.⁶²⁸

The less common septicaemic form of plague is an “overwhelming” infection with *Y. pestis* in the bloodstream.⁶²⁹ Like bubonic plague, septicaemic plague occurs

⁶²⁵ Pollitzer 1952, 382-383; Dennis *et al.* 1999, 11; Gage and Kosoy 2005, 506-509.

⁶²⁶ Dennis *et al.* 1999, 11-12.

⁶²⁷ Dennis *et al.* 1999, 43-44.

⁶²⁸ Pollitzer 1952, 489 & 495; Dennis *et al.* 1999, 9.

⁶²⁹ Dennis *et al.* 1999, 44.

either through a flea bite or if the bacterium makes it into an open wound. But unlike bubonic plague, *Y. pestis* multiplies in the bloodstream, not the lymphatic system. Thus, buboes/swollen lymph nodes are not common, which makes diagnosing septicaemic plague difficult. However, septicaemic plague can also develop in the later stages of bubonic plague, when, if untreated, the bacillus transfers from the lymphatic system to the bloodstream. Symptoms of septicaemic plague include high fever, chills, extreme weakness, abdominal pain, bleeding through the skin, and organ failure.⁶³⁰

Pneumonic plague is the most fatal form, and in epidemic outbreaks it is transmitted by a person who has either inhaled *Y. pestis* into the lungs, or when the infection spread to the lungs during bubonic or septicaemic infections.⁶³¹ Once a person is infected with pneumonic plague, they can easily spread it during interaction with other humans via respiratory droplets. Not unlike the other two forms, the symptoms of pneumonic plague include high fever, chills, headache, and weakness, but, in addition, chest pains.⁶³² In untreated cases, the mortality rate is 100%. The authors of the World Health Organization's comprehensive *Plague Manual* explain that death occurs if treatment is not administered "within 18-24 hours of disease onset", making pneumonic the most lethal and fastest acting of the three forms.⁶³³

Based on the written evidence, bubonic plague was probably the common form during the Justinianic pandemic; the multiple references to buboes are most

⁶³⁰ <https://www.cdc.gov/plague/symptoms/index.html>

⁶³¹ <https://www.who.int/ith/diseases/plague/en/>

⁶³² Dennis *et al.* 1999, 45.

⁶³³ Dennis *et al.* 1999, 45.

indicative. However, the accounts of Procopius, John of Ephesus, Evagrius, and Agathias, which mention people dropping dead instantly, suggest that pneumonic plague may have also played a role in Constantinopolitan outbreaks; possibly developing amidst bubonic outbreaks.

Carriers and Vectors of *Y. pestis*

As mentioned, rodents and their fleas are the main actors in the plague cycle, and thus are the agents principally responsible for bringing plague to humans. Many rodent species can act as carriers of *Y. pestis*, but it starts with wild rodents – which in most cases can carry the infection but are resistant to the disease – and spills into peri-domestic rodent populations.⁶³⁴ The actual transfer of *Y. pestis* to humans occurs via insect vectors. In this case, fleas are most important transmitters, but rodents are the reservoirs. Rodents' ability to carry *Y. pestis* and its association with plague in humans has been attested since the late 19th century. The scientist after which *Yersinia pestis* is named, Alexandre Yersin, first isolated and identified the bacterium in 1894. He made the connection that rodents were responsible for plague in humans when noting a multitude of dead rodents scattered throughout Hong Kong where an epidemic occurred, later confirming his theory in the lab.⁶³⁵

Rodent carriers

The most important rodent species for the Justinianic Plague (and most other plague epidemics) is *Rattus rattus*, better known as the black rat. *R. rattus* is the most

⁶³⁴ Pollitzer 1952, 382-83; Dennis *et al.* 1999, 63; <https://www.cdc.gov/plague/transmission/index.html>

⁶³⁵ Audoin-Rouzeau 2003, 21; Simond 1897, 638-639.

widespread peri-domestic rodent in the Mediterranean region, but what makes it extremely significant for plague epidemics, as expressed by the authors of the World Health Organisation's *Plague Manual*, is "that the species readily lives both as a commensal and wild species and thus can serve to introduce plague from its sylvatic reservoirs into a commensal cycle."⁶³⁶ This suggests that the black rat is the probable culprit for plague epidemics in the Mediterranean region and at its borders, including the Justinianic pandemic.

It had been argued that *R. rattus* was not introduced to Europe until the mediaeval period, and thus could not have been responsible for plague outbreaks there in the sixth century.⁶³⁷ However, in 1952 MacArthur showed indisputable archaeological evidence of the black rat's existence in Europe from at least the first century AD onward.⁶³⁸ In the 1990s, more rat bones were found on Corsica that date to between the fourth and second centuries BC.⁶³⁹ As archaeology improved over the latter half of the 20th century, more discoveries were made, cementing the idea that the black rat was an established species in Europe from least the early-Imperial period.⁶⁴⁰ While its presence was more pronounced in some parts of Europe more than others (it may not have reached Scandinavia and north-western Russia until the Middle Ages), the discoveries lend support to the idea that *R. rattus* was first established along major sea, river, and land trade and transportation routes.⁶⁴¹ In

⁶³⁶ Dennis *et al.* 1999, 69 & 85.

⁶³⁷ Pollitzer 1952, 411-412: while Pollitzer principally cites studies in favour of a mediaeval introduction, he includes the opposing MacArthur 1952, and with this evidence gives way to the idea that *R. rattus* may "have been present in Europe longer than has usually been assumed." Those that proposed a mediaeval introduction: Wu *et al.* 1936; Holsendorf 1937; Morgan, Fisher and Watson 1942.

⁶³⁸ MacArthur 1952, 209-212.

⁶³⁹ Vigne and ène Valladas 1996, 200-204; McCormick 2003, 8.

⁶⁴⁰ Audoin-Rouzeau 2003, 117-124: gives an overview of the growing archaeological discoveries of the black rat in Europe over the decades.

⁶⁴¹ Audoin-Rouzeau 2003, 119-122; on *R. rattus* in Scandinavia: Hufthammer and Walløe 2013, 1755-56.

2003, McCormick argued that the black rat most definitely lived throughout Europe from the Roman period, and he thought the evidence for this had been accumulating from the late 20th century. Further, he says the only reason we do not have more evidence is that “Archaeologists were not looking for it, and the tiny rats bones easily escape the trowler’s naked eye”, both convincing points; the latter especially, given the uptick in interest in the Justinianic Plague from the end of the 20th century.⁶⁴² As part of their minimalist outlook on this pandemic, Mordechai and Eisenberg make a weak case against the black rat’s role in the Justinianic Plague in Europe, claiming a “paucity of [rat] finds”, despite evidence of the animal across most of Europe.⁶⁴³ Nevertheless, the point is, if we have evidence of the black rat in Europe well before late antiquity, even if the evidence is not yet abundant, once the species was established it is unlikely that it existed only in small numbers, in a highly urbanised Roman world, for five centuries up to the age of Justinian. Therefore, it is probable that the principal historical carrier of plague was also its main carrier during the Justinianic pandemic.

While *R. rattus* was probably the main carrier for our plague, *R. norvegicus*, or the brown rat, is another peri-domestic rodent capable of carrying plague. Like the black rat, the brown rat has been known to instigate epidemics of plague, although thought to a far lesser extent.⁶⁴⁴ A problem for its relevance to the Justinianic Plague, unlike *R. rattus*, *R. norvegicus* did not reach Europe until the early-modern era, making its significance in our pandemic minimal.⁶⁴⁵ An additional problem is that

⁶⁴² McCormick 2003, 5-6.

⁶⁴³ Mordechai and Eisenberg 2019, 32-33.

⁶⁴⁴ Pollitzer 1952, 409-412; Hinton 1919, 6-7.

⁶⁴⁵ Hinton 1919, 4; Pollitzer 1952, 412; McCormick 2003, 2, n. 03.

the brown rat is normally found in more temperate regions, usually not choosing warmer climates such as Africa as its habitat. Like *R. rattus*, *R. norvegicus* is of Asiatic decent, but *R. norvegicus* comes from north China, while the black rat descends from warmer parts of Asia, such as Burma and India.⁶⁴⁶ Hinton explains that the brown rat is “by nature used to and fitted for a hard life in a cool country”, suggesting it probably was not common along the southern shores of the Mediterranean, similar to its distribution today.⁶⁴⁷ In any event, it seems that the brown rat migrated west many centuries after the black rat and was not present in Europe or the Mediterranean region at the time of our pandemic.

Mus musculus, or, the house mouse, is another common commensal rodent that has a wide distribution. However, although it can carry *Y. pestis*, it “has little role in plague epidemiology”; mainly due to the fact that *M. musculus*’s main parasites, *Leptopsylla segnis* and *Aetheca wagneri*, are poor vectors.⁶⁴⁸ Therefore, that leaves *R. rattus* as the commensal rodent carrier most likely responsible for bringing the disease to humans in the sixth century AD.

Insect vectors

There exist many insect vectors that are relevant to plague transmission, but only a few are relevant to the Justinianic pandemic, because of their geographical distribution, predilection for specific hosts, and their effectiveness in transmitting *Y. pestis*. I exclude vectors that are not found in the vicinity of the Mediterranean, those that do not parasitize the relevant carriers (above), and vectors that cannot efficiently

⁶⁴⁶ Pollitzer 1952, 411.

⁶⁴⁷ Hinton 1919, 4; Dennis *et al.* 1999, 86.

⁶⁴⁸ Dennis *et al.* 1999, 105; Hinnebusch, Jarrett, Bland, 225.

transmit *Y. pestis* to humans and are thus unlikely to have incited epidemics. Those that remain are all rodent fleas.

Xenopsylla cheopis (the Oriental rat flea) is the most important vector for pandemics of plague for five reasons: (1) it is the black rat's most common parasite; (2) it has a wide geographical distribution – it is a “cosmopolitan” rat flea – found in Africa, throughout Asia, and in parts of Europe; (3) it readily feeds on humans; (4) it is a highly effective transmitter of *Y. pestis*; and (5) it is commonly found in plague endemic areas and has been noted as the vector responsible for epidemics.⁶⁴⁹ A recent study by a number of scholars who research bubonic plague affirmed that, “as a primary flea of the black rat *Rattus rattus*, *X. cheopis* is likely the main vector of plague in foci affecting humans.”⁶⁵⁰ *X. cheopis* is thought to have originated in the Nile Valley, but spread throughout the Mediterranean region and beyond, to regions such as East Asia, India, and South America; however, little is known about its early history.⁶⁵¹ Its suspected region of origin coheres with the first reported outbreak of the Justinianic pandemic at Pelusium, Egypt. Also important is that, unlike other fleas, *X. cheopis* is a known traveller. Due to its inclination to feed often, *X. cheopis* remains on rats, humans, and other animals as they travel, while also found in agricultural goods such as rice and in baggage.⁶⁵² Because of this, *X. cheopis* is well known to infest merchant vessels, introducing plague to distant ports and thus new regions.⁶⁵³ However, the Oriental rat flea does have limits: it prefers a warmer climate. This is not to say that *X. cheopis* is only found in warm climates – as it “is

⁶⁴⁹ Pollitzer 1952, 246-247; Dennis *et al.* 1999, 64-66; Audoin-Rouzeau 2003, 41-47; Busvine 2015, 56.

⁶⁵⁰ Ben Ari *et al.* 2012, 2.

⁶⁵¹ For the origin of *X. cheopis* see: Jordan and Rothschild 1908, 44; Pollitzer 1952, 246. For its current distribution see Dennis *et al.* 1999, 65-84.

⁶⁵² Pollitzer 1952, 237 & 301-305.

⁶⁵³ Dennis *et al.* 1999, 65-66.

largely cosmopolitan found virtually wherever *Rattus rattus* is found” – but it is more prevalent in the subtropical (e.g. the Mediterranean, India) and tropical zones than the temperate zone.⁶⁵⁴ The Oriental rat flea’s exceptional ability to transmit *Y. pestis* to humans, through the facility of *Y. pestis* to firmly establish itself inside of *X. cheopis*, will be discussed in the next section.

Compared to *X. cheopis*, *Nosopsyllus fasciatus* (the northern rat flea) is a lesser vector of plague. *N. fasciatus* has a more limited distribution – most common in Europe and North America – and feeds far less often than the Oriental rat flea.⁶⁵⁵ Consequently, the northern rat flea is not a traveller. It is usually found off the body of its host, in the host’s nest or elsewhere.⁶⁵⁶ With that said, *N. fasciatus* does parasitize rats and can transmit plague, albeit with less effectiveness than *X. cheopis*. Some see it as “relatively unimportant” as a vector of plague, but the fact remains that data show it can become infected with and transmit *Y. pestis*; thus it should be considered as a potential vector for our pandemic.⁶⁵⁷ Compared with the Oriental rat flea, the northern rat flea is found in greater numbers in temperate climates (e.g. northern Europe) and cannot survive in regions with high average temperatures (above 18°C) and low humidity (below 40%).⁶⁵⁸ Given that many outbreaks occurred in subtropical regions in the sixth century, it makes *N. fasciatus* a less important vector than *X. cheopis* for the Justinianic pandemic. *N. fasciatus* might have played a role in the outbreaks in Europe; however, it need not have done since, as mentioned, *X. cheopis* is found everywhere *Rattus rattus* is.

⁶⁵⁴ Pollitzer 1952, 237; Harwood and James 1979, 327; Dennis *et al.* 1999, 65-84.

⁶⁵⁵ Harwood and James 1979, 325; Dennis *et al.* 1999, 65.

⁶⁵⁶ Pollitzer 1952, 237.

⁶⁵⁷ Audoin-Rouzeau 2003, 41-52. Those viewing it as relatively unimportant: Harwood and James 1979, 325;

⁶⁵⁸ Audoin-Rouzeau 2003, 47-53; Harwood and James 1979, 325.

Our final relevant plague vector is *Xenopsylla brasiliensis*.⁶⁵⁹ *X. brasiliensis* is a species of rat flea which originated in sub-Saharan Africa, and today is also found in Brazil and India.⁶⁶⁰ It is more prevalent in wild settings than urban, leading Pollitzer to claim that it is the main rodent flea in rural areas. *X. brasiliensis* is more tolerant of drought than *X. cheopis*, whereas the latter is more resistant to high temperatures – probably the reason *X. brasiliensis* is only found south of the Sahara.⁶⁶¹ *X. brasiliensis* is an efficient vector of plague and is “readily attacking humans if it is deprived of its host”, raising its potential as an actor in our pandemic.⁶⁶² Given its preference for natural settings and limited geographical distribution, a role in the Justinianic Plague’s urban epidemics in the Mediterranean and Europe is unlikely. However, if the plague in 541 began from a focus south of the Sahara, *X. brasiliensis* may be implicated in the initial rural outbreaks that helped the bacillus reach the more wide-ranging, urban *X. cheopis*.

Discussion

From this review of the main vectors of plague, the Oriental rat flea appears to be the species most likely responsible for Mediterranean outbreaks of plague, and thus the initial outbreaks of the Justinianic Plague. That *X. cheopis* probably originated in Egypt (the region of the first recorded outbreaks) and is found throughout the Mediterranean, that it is a primary parasite of the black rat (a known commensal rodent of the ancient Mediterranean), and that it is a most effective transmitter of *Y.*

⁶⁵⁹ *Xenopsylla astia* was considered for this section but ultimately not included due to its ineffectiveness as a transmitter and restricted geographical distribution: Harwood and James 1979, 327; Audoin-Rouzeau 2003, 51-56; Pollitzer 1952, 245.

⁶⁶⁰ Harwood and James 1979, 327; Audoin-Rouzeau 2003, 52; Miarinjara *et al.* 2016, 2208.

⁶⁶¹ Dennis *et al.* 1999, 66; Pollitzer 1952, 242.

⁶⁶² Audoin-Rouzeau 2003, 52.

pestis, all point to the likelihood of it playing a central role in our pandemic. The northern rat flea may have helped spread plague in Europe, but given that the “cosmopolitan” *X. cheopis* is found everywhere *R. rattus* is makes that unnecessary.⁶⁶³ If the pandemic began from a reservoir south of the Sahara, which is very likely (as argued below), the epidemic may have begun rurally with wild rodents and *X. brasiliensis*. The bacillus then probably transferred to the black rat and the Oriental rat flea, and thus urban areas of the eastern coast, which led to plague travelling up the Red Sea. *X. brasiliensis* is unlikely to have played a role larger than epizootic outbreaks in the wild regions of sub-Saharan Africa, due to its choice of habitat and sensitivity to high temperatures. Therefore, the leading candidate for the Justinianic outbreaks is *X. cheopis*.

A note on Pullex irritans (the human flea)

It was long thought that *Pullex irritans*, or, the human flea, may been responsible for plague epidemics throughout the world. On the surface, it seems plausible; rat fleas can effectively transmit *Y. pestis*, so why should the flea most common to humans not be a foremost vector of plague epidemics? The theory was propounded by Georges Blanc and Marcel Baltazard in highly flawed papers published in the early 1940s.⁶⁶⁴ They argued that the human flea was just as important, if not more important, a vector as the Oriental rat flea in urban epidemics. Blanc and Baltazard suggested that human-to-human transmission via *P. irritans* was viable and a possible reason for major plague epidemics. They conclude, that “once infected, [*P.*

⁶⁶³ Harwood and James 1979, 327.

⁶⁶⁴ Blanc and Baltazard 1941, 9-21; Blanc and Baltazard 1942, 446-48; Blanc and Baltazard 1945, 173-348.

irritans] certainly transmits plague by bite, on condition that a sufficient number of fleas are used in the experiments to compensate for the rarity of the bites made by each of them.” However, upon examination of the experiments it becomes clear that this conclusion is false. It is rare that *Y. pestis* actually infects *P. irritans*; that is, the bacillus cannot effectively establish itself inside the gut of the human flea, and the human flea can only transmit *Y. pestis*, as put by Audoin-Rouzeau, via “macro-mass” mechanical transmission (hundreds of fleas with mouth parts contaminated with *Y. pestis*).⁶⁶⁵ (More on modes of transmission via the flea in the next section.) In Blanc and Baltazard’s experiment, they placed 700+ fleas, all of which had just fed on a highly infective person, onto a guinea pig, and that guinea pig became infected from the bites of the 700+ contaminated flea mouths. They tried the same type of mechanical transmission in eight previous experiments with as many as 240 fleas (“mass mechanical”), but all returned negative results, which drove them to attempt more than double the original number. Only in this ninth experiment did they achieve a positive result, leading them to declare the above conclusion.⁶⁶⁶ These results in fact show the opposite: *P. irritans* is one of the least effective transmitters of plague and could not have been responsible for any major plague epidemic. A study from 2017 explained that “Fleas such as *P. irritans*...which exhibit daily, sustained feeding behaviors, digest their meals rapidly, and defecate large amounts of blood during and shortly after feeding, have been found to clear themselves of infection [and not develop gut blockage] at a high rate and to be poor vectors.”⁶⁶⁷ Thus, human fleas usually only transmit *Y. pestis* via contaminated mouths and in

⁶⁶⁵ Audoin-Rouzeau 2003, 103-114.

⁶⁶⁶ Blanc and Baltazard 1945, 173-348; Audoin-Rouzeau 2003, 103-114.

⁶⁶⁷ Hinnebusch, Jarrett and Bland 2017, 225.

extraordinary numbers (i.e. mechanical transmission). The following section shows why mass mechanical transmission (let alone “macro-mass”) cannot have been responsible for any major outbreak of plague, and why colonisation of the flea’s gut by *Y. pestis* is important.

The Plague Cycle and Factors Affecting Transmission

This section seeks to describe the plague cycle, the evolution of *Y. pestis*, and factors affecting transmission.

The plague cycle

Before reaching humans, *Y. pestis* lives naturally among wild rodents such as gerbils, voles, and marmots in the sylvatic cycle, before reaching its primary urban carrier, *Rattus rattus*, the black rat.⁶⁶⁸ As mentioned, the black rat frequently travels between wild and urban settings, and *R. rattus* lived in the Mediterranean region and Europe since at least the Republican period, making it the carrier most likely to have transferred *Y. pestis* to humans in the sixth century AD. While the black rat is crucial to the spread of plague, as we have seen, rat fleas (particularly Oriental rat fleas, or, *Xenopsylla cheopis*) are of primary importance in the transfer of *Y. pestis* between rodents and to other mammals, including humans.⁶⁶⁹ A flea becomes infected by biting an infected rodent and ingesting *Y. pestis* from its bloodstream.

Once in the flea’s gut, *Y. pestis* begins to multiply, and key features of its genetic make-up prevent it from being excreted before it has reproduced extensively.

⁶⁶⁸ Meyer 1957, 710-11; McCormick 2003, 2-3; Harper 2017, 210-13.

⁶⁶⁹ Gage *et al.* 2008, 443-45; Eisen, Dennis and Gage 2015, 1184; Miarinjara *et al.* 2016, 2207-08.

The present evolutionary strain of *Y. pestis* possesses a gene known as the Yersinia murine toxin, or *ymt*, gene, characterized by the plasmid-encoded phospholipase D (PLD) enzyme. The PLD enzyme protects the bacillus from being flushed out of the flea's midgut by cytotoxic digestion.⁶⁷⁰ With the *ymt* gene guarding it in the midgut *Y. pestis* has more time to multiply and express a series of *hms* genes (genes discussed further below). The *hmsHFRS* operon is a set of genes essential to the creation of a biofilm, which either partially or fully closes off the entrance to the flea's midgut with a colony of the bacterium still inside (Figures 53 and 54).⁶⁷¹ This stage of transmission is known as Late-phase biofilm transmission. If a flea's midgut is blocked, or partially blocked, via a biofilm, a feeding flea struggles to suck

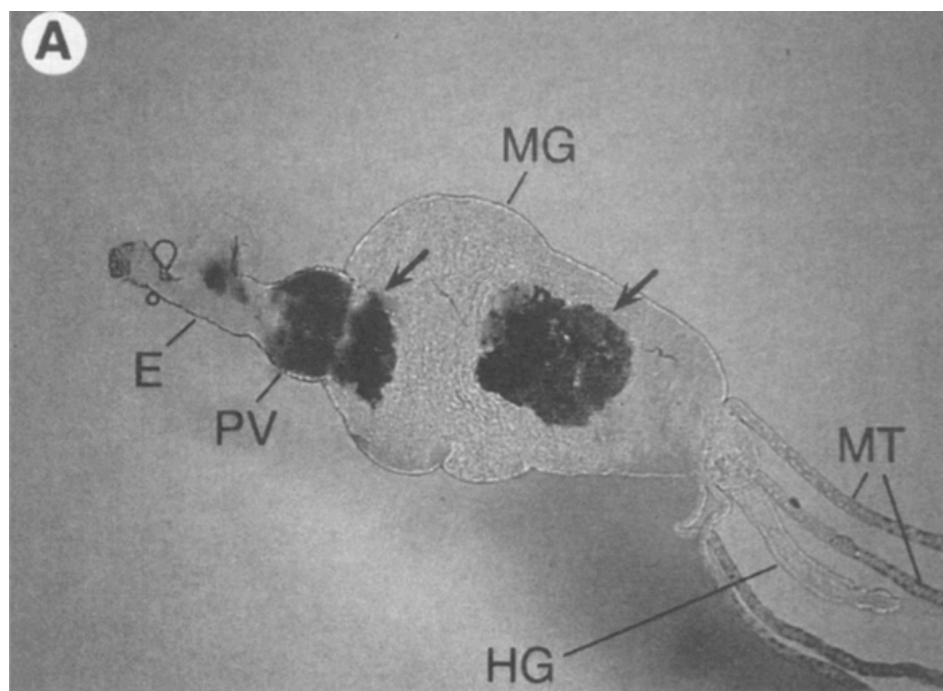


Figure 53. The contours are a digestive tract dissected from a blocked *Xenopsylla cheopis* flea. MG = midgut; E = esophagus; PV = proventriculus; HG = hindgut; MT = Malpighian Tubules. Note the black mass of *Y. pestis* clogging the proventriculus, as well as the mass in the center of the flea's midgut. (Hinnebusch *et al.* 1998, 1410)

⁶⁷⁰ Hinnebusch *et al.* 2002, 733; Hinnebusch 2005, 200-01 and 206; Hinnebusch, Jarrett, and Bland 2017, 222-23.

⁶⁷¹ Hinnebusch *et al.* 2002, 733; Hinnebusch 2005, 201-203; Rasmussen *et al.* 2015, 573-75.

blood past its proventriculus (a valve that joins the flea's esophagus and midgut).⁶⁷²

In Late-phase biofilm transmission both partially and fully blocked fleas are not consuming the amount of blood they are used to. Thus, the infected starving fleas search for blood, aggressively biting any mammal they can latch onto. In their attempts to feed, the fleas regurgitate *Y. pestis* into the bite wounds of uninfected animals, passing on the bacterium in large numbers.⁶⁷³

Since the early 20th century, Late-phase biofilm transmission has been generally accepted as the mode of transmission responsible for most large-scale outbreaks of plague.⁶⁷⁴ However, some recent studies have proposed or subscribed to the theory that Early-phase transmission (EPT, or mechanical transmission) — occurring during the early stages of infection before biofilm development — is more likely responsible for mass epizootic events, including human plague epidemics (specifically citing the Black Death), than late-stage biofilm transmission.⁶⁷⁵ The significance of Late-phase vs Early-phase transmission to the scope of this chapter will be discussed below, but the dispute originates from the observed shorter lifespan of blocked fleas compared to EPT fleas.⁶⁷⁶ The proponents of EPT believe that blocked fleas do not live long enough to cause mass epizootic events, and that study of EPT as the potential primary route of transmission has been neglected.⁶⁷⁷

In 2017, the leading *Y. pestis* and *X. Cheopis* microbiologist Joe Hinnebusch, along with microbiologists Clayton Jarrett and David Bland, published a study that

⁶⁷² Hinnebusch 2005, 199.

⁶⁷³ Hinnebusch 2005, 199.

⁶⁷⁴ Bacot and Martin 1914; AW Bacot 1915; Pollitzer 1952, 271; Meyer 1957, 711; Kartman 1969; Cavanaugh 1971; Hinnebusch, Perry and Schwan 1996; Hinnebusch *et al.* 1998; Hinnebusch *et al.* 2002; Hinnebusch 2005; Hinnebusch, Jarrett, and Bland 2017.

⁶⁷⁵ Eisen *et al.* 2006, 15380-85; Schotthoefer *et al.* 2011, 411-417; Eisen, Dennis, and Gage 2015, 1183-1192.

⁶⁷⁶ Eisen *et al.* 2006, 15380.

⁶⁷⁷ Eisen *et al.* 2006, 15380-85.

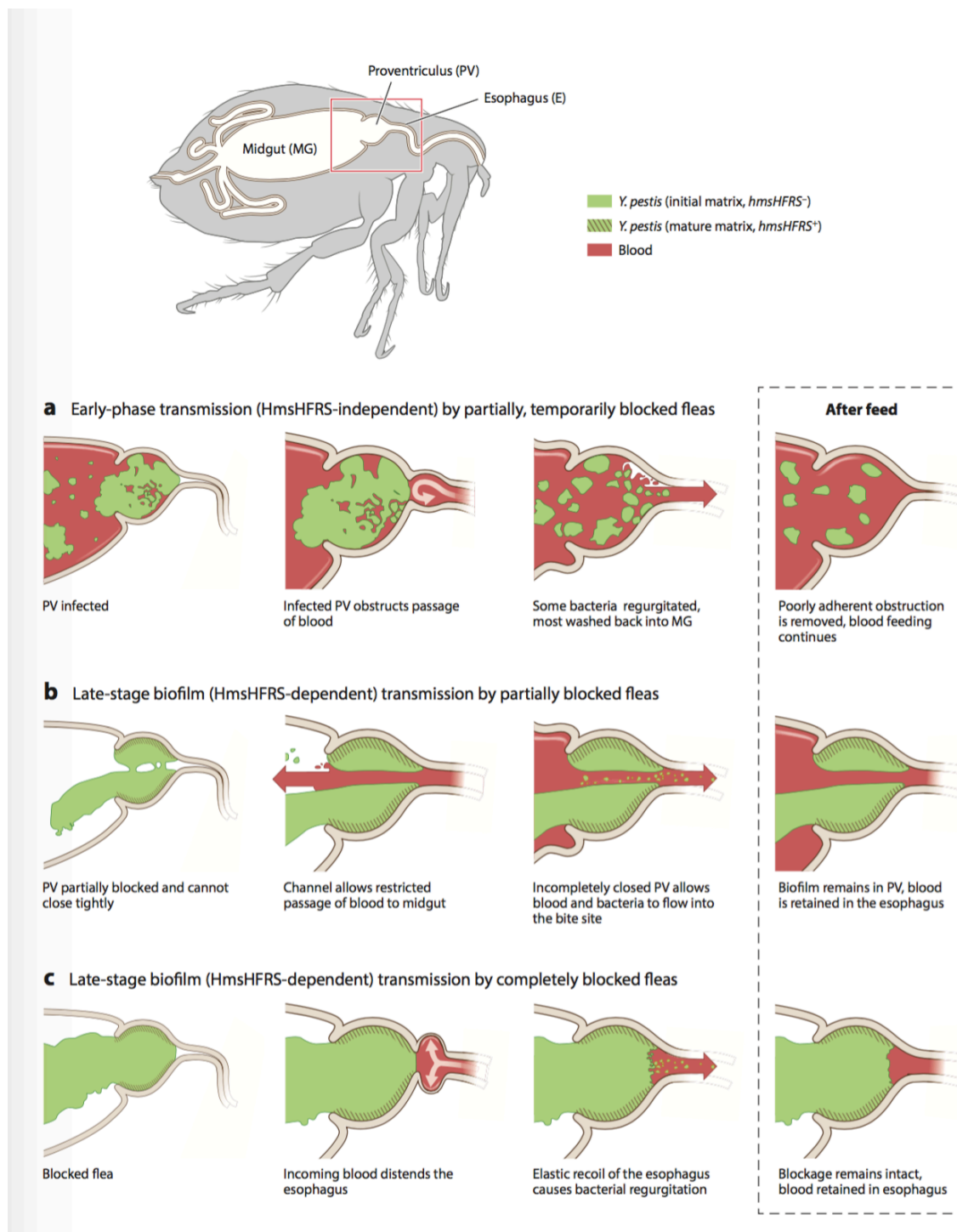


Figure 54. A model for flea-borne transmission showing the stages of biofilm development. (a) Early-phase transmission; initial ingestion of bacteria. (b,c) Late-stage transmission showing partially (b) and fully blocked (c) fleas, and the development and full maturation of the biofilm. (Hinnebusch, Jarrett, and Bland 2017)

disputes this theory, saying: (1) EPT is rare unless the concentration of *Y. pestis* in the blood is high enough to remain in the flea, which it usually is not in most mammals, aside from mice and in the very short window before a mammal dies; (2) the amount of *Y. pestis* transmitted per flea is much lower than for blocked fleas, meaning this method would necessitate a high number of infected fleas to transmit *Y. pestis*; and (3) these attempts by the flea to bite uninfected mammals with low concentrations of the bacterium would, in effect, immunize susceptible mammals, working in the opposite direction to an epizootic event.⁶⁷⁸

Hinnebusch, Jarrett, and Bland assert that despite the shorter lifespan, “because a blocked flea will make repeated attempts to feed before it starves, the cumulative number [of *Y. pestis*] transmitted can be much greater.”⁶⁷⁹ They also suggest that advocates of EPT for large-scale plague events fail to recognize the potentiality of partially blocked fleas (Fig. 54).⁶⁸⁰ Partially blocked fleas are infected in much the same way as fully blocked fleas, as *Y. pestis* has still colonised the gut, but partially blocked fleas do not have their feeding canal completely closed off. A flea with a partially blocked midgut is actually a better transmitter of plague than a fully blocked flea, since it can take in more nourishment for its own survival, living longer, while retaining the ability to transmit *Y. pestis* in large quantity.⁶⁸¹ *Y. pestis* does not aim to kill the flea – it needs the flea as a vector – but by blocking the midgut it can deplete the flea of nourishment which can lead to death.⁶⁸² However, if the right infective balance is achieved by *Y. pestis* within the flea, the flea then gets

⁶⁷⁸ Hinnebusch, Jarrett, and Bland 2017, 226-27.

⁶⁷⁹ Hinnebusch, Jarrett, and Bland 2017, 227.

⁶⁸⁰ Hinnebusch, Jarrett, and Bland 2017, 227.

⁶⁸¹ Hinnebusch *et al.* 2005, 199.

⁶⁸² Hinnebusch, Fischer, and Schwan 1998, 1409.

just enough nourishment for survival; but in this case the flea becomes blood-hungry and a most ferocious transmitter of the bacillus. Once the infected rat that the flea is riding dies, the flea can no longer ingest the bloodmeal it seeks, so it jumps to another mammal in search of more blood, which is usually how *Y. pestis* reaches humans.

There are additional aspects of the plague cycle that should be considered. Aspects of the flea's life cycle can be significant for plague epidemics, such as development of immature fleas and larvae, behaviour and survival of mature fleas, and their choice of habitat (e.g. rodent burrows, rubbish dumps, human dwellings). Additionally, rodent behaviour and survival play a role. Rodent habitats, movements, and population dynamics can all be relevant when determining the cause of a plague outbreak. How climate can affect each of these components is discussed in the next section.

This section focuses mainly on the flea: the main vector for *Y. pestis*, and the agent responsible for infecting humans on a wide scale. If not for the flea, major outbreaks of bubonic plague, such as that during the reign of Justinian, might never have happened. However, an important stage in the evolution of *Y. pestis* enabled it to use the flea as a vector and become the fast-spreading disease we know today.

Evolution of Y. pestis

Where the flea originally picked up the plague from wild rodents is still uncertain; *Y. pestis* has been detected in skeletal remains from across Eurasia for many thousands of years.⁶⁸³ The geographical reservoir for *Y. pestis* was thought to be the Qinghai-

⁶⁸³ Cui *et al.* 2013, 577-82; Rascovan *et al.* 2019, 295-305.

Tibet Plateau in China, until a recent archaeological discovery found the earliest aDNA evidence of the bacterium in human remains from a Neolithic cemetery in Sweden.⁶⁸⁴ But there is a crucial difference between the earliest detections of *Y. pestis* and the version that caused the Justinianic Plague. The strains of *Y. pestis* detected in remains from China (4761 BP=BC 2811) and Sweden (4900 BP=BC 2950) lack the *Yersinia murine* toxin (*ymt*) gene, which enables the bacterium to establish itself in the flea, in effect, infecting the flea, and allowing the flea to become a vector.⁶⁸⁵

Genomic analysis showed that the mutation happened sometime between 1686 cal BC – 951 cal BC.⁶⁸⁶ This evolutionary moment seems to have been a specific adaptation to the flea as a vector, since the *ymt* gene is vital to flea-borne transmission of *Y. pestis*, but has not shown to be essential elsewhere.⁶⁸⁷ Before the addition of the *ymt* gene, *X. cheopis* would bite infected rodents and quickly excrete *Y. pestis* without becoming infected; infection by way of mechanical transmission (e.g. contact with an uninfected flea's contaminated mouth) is possible, but far less efficient.⁶⁸⁸ Whether partially blocked or fully blocked, it seems Late-phase biofilm transmission is the more destructive form of plague transmission – far more effective than EPT/mechanical transmission – and this was made possible with the addition of the *ymt* gene. With *ymt*, *Y. pestis* transmitted much more effectively, and thus in scope the bacterium became more lethal.

⁶⁸⁴ For early aDNA evidence from Qinghai-Tibet Plateau: Cui *et al.* 2013, 577-79. For the most recent evidence from Sweden: Rascovan *et al.* 2019, 295-99.

⁶⁸⁵ Rasmussen *et al.* 2015, 574-75; Rascovan *et al.* 2019, 296-97; Hinnebusch *et al.* 2002, 733-35.

⁶⁸⁶ Rasmussen *et al.* 2015, 574.

⁶⁸⁷ Hinnebusch *et al.* 2002, 735.

⁶⁸⁸ Hinnebusch, Jarrett, and Bland 2017, 225-27.

Infection/gut-blockage within two focus vectors: Xenopsylla cheopis and Nopsylla fasciatus

In transmission experiments via blockage, *X. cheopis* has been shown to block more frequently than other flea species and to be the most efficient vector of plague. Bacot and Martin conducted one of the first studies on transmission viability, using *X. cheopis* and *N. fasciatus*. Their experiments found that *Y. pestis* blocks/infects *X. cheopis* more often and that blockage occurs quicker than the other case study, *N. fasciatus*.⁶⁸⁹ This is not to say *N. fasciatus* did not block or was not an effective vector. Bacot and Martin also found the Oriental rat flea to be a more “persistent feeder” once blocked, frequently renewing its attempts to feed, further demonstrating its lethality as a vector. In 1947, Albert Burroughs conducted a study that tested the vectoral capacity of ten fleas. The Oriental rat flea prevailed as the best vector. Not only did the Oriental rat flea develop blockage the quickest and most often, it transmitted the disease (to lab rodents) best at 66%.⁶⁹⁰ *N. fasciatus* was also tested and found to be less efficient, but still a relatively effective vector, with 37% of blocked fleas having transmitted *Y. pestis* in Burroughs’ experiment.⁶⁹¹ *X. brasiliensis* is able to block, and has been noted as an effective vector, but its efficiency has not been tested like that of our other two relevant vectors.⁶⁹²

The experiments show that *Y. pestis* seems to be most adapted to the Oriental rat flea as a vector. *X. cheopis*, the probable main vector of the Justinianic Plague, develops gut blockage easier than other flea species and is a highly effective transmitter of plague. The secondary vector, *N. fasciatus*, can become blocked by *Y.*

⁶⁸⁹ Bacot and Martin 1914, 432.

⁶⁹⁰ Burroughs 1947, 379-383; Audoin-Rouzeau 2003, 60-62.

⁶⁹¹ Burroughs 1947, 383-384; Audoin-Rouzeau 2003, 60-62.

⁶⁹² Audoin-Rouzeau 2003, 53-54.

pestis and it is able to transmit the bacillus, but less effectively than *X. cheopis*.

Colonisation of the flea's gut by *Y. pestis* is a crucial element in the epidemiology of plague, one that determines epidemics. However, this blockage feature comes with environmental parameters: it is sensitive to temperature.

Temperature dependence

Y. pestis in the flea

Lab testing shows that *Y. pestis* is sensitive to ambient temperature once the bacterium is ingested by the Oriental rat flea.⁶⁹³ In 1969, Leo Kartman published a study revealing that blockage (or partial blockage) of the midgut in *X. cheopis* by *Y. pestis* is "twice as likely to occur" at 23.5°C than at 29.5°C.⁶⁹⁴ He also tested at 8.5°C, finding blockage was only marginally lower than that at 23.5°C. Two years later, Dan Cavanaugh conducted a similar study, showing that growth of *Y. pestis* in a flea's midgut, and its eventual blockage, is viable at 20°C, while blockage is far less likely in the high 20s and approaching 30°C.⁶⁹⁵ Cavanaugh concluded that the probability of *Y. pestis* being retained in the flea greatly decreases at temperatures in excess of 27°C.⁶⁹⁶

In 1998, Hinnebusch, Fischer, and Schwan responded to Cavanaugh's study, revealing more precise data for the plague bacillus' sensitivity to temperature once ingested by the flea.⁶⁹⁷ However, their study also revealed that the decrease in blockage rates at higher temperatures is not due to the non-expression of the

⁶⁹³ Kartman 1969, 71-5; Cavanaugh 1971, 264-273; Hinnebusch, Fischer, and Schwan 1998, 1408-09.

⁶⁹⁴ Kartman 1969, 72-73.

⁶⁹⁵ Cavanaugh 1971, 266-69.

⁶⁹⁶ Cavanaugh 1971, 269-271.

⁶⁹⁷ Hinnebusch, Fischer, and Schwan 1998, 1408-09.

plasminogen activator (*pla*) gene, which protects *Y. pestis* from fibrinolysis – the enzymatic breakdown of the fibrin in blood clots, thought to help destroy *Y. pestis* in the flea at higher temperatures – as Cavanaugh proposed.⁶⁹⁸ They found, instead, the failure of *Y. pestis* to block fleas at temperatures above 27°C is due to *Y. pestis*' inability to express the *hms* genes (discussed above), the genes that create a biofilm and help the bacterium colonise and block the proventriculus.⁶⁹⁹

Nevertheless, Hinnebusch, Fischer, and Schwan's results were the same as Cavanaugh's: *Y. pestis* struggles to block fleas at temperatures nearer to 30°C than 20°C. As shown in Table 8: at least 24%, but not more than 45%, of fleas were blocked in every test at 20°C; between 2% -15% of fleas were blocked in the 25°C tests; and no fleas were blocked at 30°C.⁷⁰⁰ Their results demonstrate that once inside the flea, *Yersinia pestis* is quite sensitive to ambient temperatures, showing the highest success at blocking a flea's midgut at 20°C, the coolest temperature tested, less success at 25°C, and no success in tests at 30°C.

A study published in 2020 tested the blockage viability of *X. cheopis* and *Oropsylla montana* (only found in North America) at temperatures lower than previously tested.⁷⁰¹ The microbiologists conducted blockage experiments on both species at ambient temperatures (21°C) and 10°C. Similar to the previous study, they note a good blockage rate of 51% for *X. cheopis* under ambient temperatures.⁷⁰² At 10°C, *X. cheopis* still showed a blockage rate of 43.6%. However, the time it took *X. cheopis* to reach blockage at 10°C nearly doubled – from the standard 11-13 days to a

⁶⁹⁸ Cavanaugh 1971, 264-265; Hinnebusch, Fischer, and Schwan 1998, 1408.

⁶⁹⁹ Hinnebusch, Perry, and Schwan 1996, 367; Hinnebusch Fischer, and Schwan 1998, 1413-14.

⁷⁰⁰ Hinnebusch, Fischer, and Schwan 1998, 1408.

⁷⁰¹ Lemon, Cherzan and Vadyvaloo 2020.

⁷⁰² Lemon, Cherzan, Vadyvaloo 2020, 2.

21.63 day average – indicating that lower temperatures probably “slow metabolic activity” for both the Oriental rat flea and the bacillus.⁷⁰³ This suggests that epidemics in which *X. cheopis* is involved may decelerate if conditions are too cold.

Fleas infected with	Plasmid content			% of fleas blocked at			Flea mortality (%) at		
	pPla	pYVp	pFra	20°C	25°C	30°C	20°C	25°C	30°C
<i>Y. pestis</i> 195/P-wt	+	+	+	32 45 37	13	0	42 72 63	41	70
<i>Y. pestis</i> 195/P-1	–	+	+	37 28 34	15 11 5	0	48 33 56	56 53 54	90 75 64
<i>Y. pestis</i> 195/P-2	–	–	+	36 35 33	10 6 10	0	54 72 46	56 68 59	73 71 62
<i>Y. pestis</i> KIM6 ⁺	+	–	+	24 38 34	7 4 3	0	49 56 33	48 37 30	59 76 70
<i>Y. pestis</i> KIM10 ⁺	–	–	+	28 25 26	2 4 3	0	54 61 38	58 44 56	65 92 82
<i>Y. pestis</i> 195/P-3	–	+	– ^a	0 1 0	ND	ND	21 20 23	ND	ND
No bacteria				0 0 0	0 0 0	0	20 17 9	16 19 16	78 79 68

NOTE. Results of replicate experiments are shown. Total no. of fleas was 57, 46, and 102 for 3 experiments using virulent *Y. pestis* 195/P-wt at 20°C; *n* = 85–110 for all other experiments. All samples comprised equal numbers of male and female fleas, which were monitored for 4 weeks after single infectious blood meal. Data for *Y. pestis* 195/P-wt and KIM6⁺ at 20°C are from [33]. ND = not done.

^a *Y. pestis* 195/P-3 lacks at least *caf* and *ymt* operons of pFra ($\Delta caf\Delta ymt$).

Table 8. The effect of different temperatures on the blocking success of *X. cheopis*. Note the high success rate of blocking fleas at 20°C, moderate success at 25°C, and complete failure to block at 30°C. Mortality rates were high at all three temperatures tested due to dehydration and starvation of the test subjects. (Hinnebusch *et al.* 1998, 1408)

⁷⁰³ Lemon, Cherzan, Vadyvaloo 2020, 2-4.

However, unlike in hot conditions, when it is cold *Y. pestis* can still block the Oriental rat flea's gut, so plague epidemics do not necessarily dissolve in cooler environments.

Cavanaugh's and Hinnebusch, Fischer, and Schwan's results indicate that *Y. pestis* cannot establish itself in its primary vector if the environment is too hot. On the other end, Lemon, Cherzan, and Vadyvaloo's research shows that in *X. cheopis* the bacterium's blocking mechanism slows in cooler temperatures, suggesting epidemics may wane, but not disappear, under conditions around 10°C (and presumably cooler temperatures as well). Thus, plague outbreaks are highly dependent on climate, indicating that epidemics may extend or stagnate based on regional temperatures and dramatic shifts in climate.

Early-phase transmission vs. Late-phase biofilm transmission

As discussed above, the scientific evidence up to this point suggests that Late-phase biofilm transmission is far more likely to create epizootic events that lead to human plague epidemics than is Early-phase transmission/mass transmission.⁷⁰⁴ This is relevant here because in 2011 Schotthoefer et al. published a study revealing that temperature does not significantly influence the rates of transmission for fleas of Early-phase transmission (EPT)/mechanical transmission.⁷⁰⁵ They observed virtually no difference in the rates that fleas transmitted *Y. pestis* when held at 23°C, 27°C, or 30°C.⁷⁰⁶ This suggests that EPT would not be affected by dramatic shifts in climate in the way that Late-phase biofilm transmission would. However, as noted by

⁷⁰⁴ Hinnebusch, Jarrett, and Bland 2017, 225-227.

⁷⁰⁵ Schotthoefer et al. 2011, 411-417.

⁷⁰⁶ Schotthoefer et al. 2011, 413.

Hinnesbusch, Jarrett, and Bland three years ago, it is more likely that Late-phase transmission is responsible for mass-epizootic events and epidemics, and thus we should expect that it was this form of transmission that led to the Justinianic Plague.⁷⁰⁷

Discussion

As I have shown, temperature plays a significant role in the ability of *Y. pestis* to transmit widely in populations. The majority of this temperature-dependence lies with the relationship between *Y. pestis* and the Oriental rat flea, the main vector of plague for humans. At temperatures above 27°C, *Y. pestis* cannot express the *hms* genes that help the bacterium establish a foothold in the flea's proventriculus and colonise its midgut. Therefore, at higher temperatures (25°C – 27°C) the Oriental rat flea becomes a less efficient vector for the transmission of plague, and if too high (>27°C) epidemics die out. In addition, plague epidemics may slow down in cooler temperatures (≤10°C). Unfortunately, there has been no research on the effect of temperature on *Y. pestis* in our other two relevant vectors, *N. fasciatus* (the northern rat flea) and *X. brasiliensis*. But because *Y. pestis* blocks these two fleas in a similar way to how it blocks *X. cheopis*, the result may be similar. As mentioned, the lack of research here does not impede the course of this study since, at best, *N. fasciatus* is a secondary vector to *X. cheopis*, given that it is not usually found in subtropical climates (i.e. the Mediterranean region), feeds less than the Oriental rat flea, and does not travel as much, leading some scientists to label it an unimportant vector. *X. brasiliensis* only lives south of the Sahara and therefore the likelihood of its

⁷⁰⁷ Hinnebusch, Jarrett, and Bland 2017, 225-227.

involvement in the Mediterranean outbreaks is low. However, the fact that *N. fasciatus* could have been involved in the European outbreaks of the Justinianic pandemic (and later pandemics like the mediaeval Black Death), and that *X. brasiliensis* may have been responsible for East African outbreaks which helped *Y. pestis* reach the Mediterranean, does call for further research on the transmission capacity of both fleas.

The next section investigates how the transmission dynamics just reviewed were probably important to the first wave of the Justinianic Plague, and how they are likely to have been affected by the sixth-century climatic events discussed in Chapter 1. The effect of climate change on other aspects of the plague cycle, such as its influence on rodents, is also considered. It is clear that major shifts in climate affect the transmission and distribution of *Y. pestis*. There is much plausibility in the idea that the LALIA opened environmental gates that once constrained the bacterium to certain regions, allowing plague to transition from being enzootic to epizootic and then to epidemic in the sixth century AD.

Climate Change and the Sixth-Century Plague

This section draws on everything we have reviewed so far for the Justinianic Plague – the written evidence, the archaeology and aDNA evidence, carriers and vectors, the plague cycle and factors affecting transmission – and returns to the climate of the sixth-century to determine whether climate change played a role in the emergence and persistence of the pandemic. First, we must look back to the proxy evidence from Chapter 1 for a picture of climate in the sixth century AD.

Recalling the evidence from Chapter 1, two large volcanic eruptions at AD 536 (unattributed) and 540 (Ilopango) brought on the coldest decade of the millennium for the Mediterranean region and beyond; this period is often regarded as the onset of the Late Antique Little Ice Age.⁷⁰⁸ We see temperatures reduce almost ubiquitously across Europe from Spain to the European side of Turkey.⁷⁰⁹ However, wetness varied in Europe, with moisture levels moderate in Spain, on the lower end in Germany, high in Albania, and very high in northwest Turkey (formerly Thrace).⁷¹⁰ Proxy evidence from western Anatolia also shows dramatic reductions in temperatures similar to Europe, while wetness was high in northern Anatolia south of the Black Sea and in the southwest, but moderately arid in central and eastern Anatolia.⁷¹¹ In the Near East, there is evidence of aridity in Israel, and further east in central Asia temperatures fell to the lowest point since the second century AD.⁷¹² The only temperature proxy data we have for Africa are from the southern half (Namibia, South Africa, and Tanzania) and show temperatures below the annual average in the first half of the sixth century.⁷¹³ Our evidence for the northern half of Africa is moisture-based and it differs from west to east. There is evidence of drying in the Libyan Sahara, Nigeria, Gabon, and Cameroon, but proxies for wetness in the east from Kenya, Uganda, Rwanda, the Democratic Republic of the Congo, and,

⁷⁰⁸ Sigl *et al.* 2015; Büntgen *et al.* 2016.

⁷⁰⁹ Zumbühl 1980; Holzhauser 1984; Holzhauser 2001; Magny 2004, 77; Holzhauser, Magny, and Zumbühl 2005; Mangini, Spötl, Verdes 2005; Büntgen *et al.* 2011; Martin-Puertas *et al.* 2009; Martín-Chivelet *et al.* 2011; McCormick *et al.* 2012; Zanchetta *et al.* 2012; Le Roy *et al.* 2015.

⁷¹⁰ Büntgen *et al.* 2011; Göktürk 2011, 76; Zanchetta *et al.* 2012; McCormick *et al.* 2012; Cruz *et al.* 2015.

⁷¹¹ Kaniewski *et al.* 2007, 2213; Göktürk 2011, 28 & 54; McCormick *et al.* 2012.

⁷¹² Migowski 2006, 425; Fowler *et al.* 2008; Diaz and Markgraf 2010; McCormick *et al.* 2012; Büntgen *et al.* 2016.

⁷¹³ Nicholson *et al.* 2013, 1087.

notably, from around the Ethiopian Highlands (water source for the Nile, the climatic/environmental regulator of Egypt) all show wetness for the sixth century; the latter location exhibits a dramatic spike in wetness right around the 540 eruption.⁷¹⁴

The proxy evidence is clear: a period of extreme climatic instability and climate cooling commenced in the AD 540s following two large volcanic eruptions just 4 years apart. The variability coincides with the first reported outbreaks of the plague. As we will see, climate change of this kind can have an important effect on the plague cycle.

How climate change influences the plague cycle

As mentioned, the plague cycle consists of various components that keep the bacterium *Y. pestis* in check, but these components can be affected by exogenous shocks like climate change that help *Y. pestis* awaken from dormancy and spread from its reservoirs. Rodents, the principal carrier of *Y. pestis*, are affected by dramatic shifts in climate, particularly droughts and precipitation. After periods of above-average precipitation, rodent populations become more abundant, which can lead to wider prevalence of enzootic plague in reservoir regions.⁷¹⁵ With more precipitation comes greater plant growth, leading to more food for rodents and thus larger rodent

⁷¹⁴ Ngomanda *et al.* 2007; Nash *et al.* 2016. Libya: Cremaschi, Pelfini and Santilli 2006. Nigeria: Street-Perrott *et al.* 2000. Cameroon: Lézine *et al.* 2013. Kenya/Tanzania: Buckles *et al.* 2016; Blaauw *et al.* 2011; Wolff *et al.* 2011. Southeastern Africa: Stager *et al.* 2009; Alin and Cohen 2003. Uganda: James M Russell and Johnson 2005. Kenya: De Cort *et al.* 2013. Ethiopian Highlands: Foerster *et al.* 2015; Foerster *et al.* 2012, 32.

⁷¹⁵ Parmenter, Yadav, Parmenter, Ettestad and Gage 1999, 817-19; McCormick 2003, 20; Stenseth *et al.* 2006, 13110-13; Ben Ari *et al.* 2011, 2.

populations; this can be particularly pronounced in arid or semi-arid ecologies (i.e. many regions of the Mediterranean).⁷¹⁶

Rat fleas are also positively influenced by precipitation, as well as humidity. Immature flea development and adult flea survival are adversely affected by losses of humidity and moisture, as are flea larvae.⁷¹⁷ When humidity drops too low, in essence, fleas dry out and die. Precipitation is better for immature fleas living in rodent burrows, as soil moisture regulates humidity in the burrow.⁷¹⁸ Since rodent fleas are ectothermic (dependent on external sources of body heat), they require a specific temperature range and their survival and development can both benefit from, and be negatively affected by, variability.⁷¹⁹ Testing has shown that *X. cheopis* experiences optimal survival rates at around 20°C and 92% humidity.⁷²⁰ Flea development and adult flea movements improve with temperature (between 10°C and 25°C) until it gets too hot (>27°C), which either causes the flea to seek refuge or kills it; and the effect of high temperature is exacerbated with a moisture deficit.⁷²¹ As Stenseth and colleagues put it: "Turning to the summer, dry and hot conditions are known to have a harmful effect on the survival of both adult fleas and developing pre-adults. Hence, under such conditions, flea abundance will be relatively low, and summer-generations are less likely to overlap."⁷²² Therefore, if a typically ambient region in which the Oriental rat flea exists experiences an increase in temperatures, the flea population will probably decrease. Conversely, if, say, *X.*

⁷¹⁶ McCormick 2003.

⁷¹⁷ Bacot and Martin 1914, 100-05; Cavanaugh and Marshall 1972, 86-90; Gage *et al.* 2008, 439-50; Ben Ari *et al.* 2011, 2.

⁷¹⁸ Eisen and Gage 2009; Ben Ari *et al.* 2011, 2.

⁷¹⁹ Ben Ari *et al.* 2011, 2.

⁷²⁰ Burroughs 1953, 40-41.

⁷²¹ Gage *et al.* 2008, 443-45; Ben Ari *et al.* 2011, 2.

⁷²² Stenseth *et al.* 2006, 13111.

cheopis is endemic to a region that borders on another region which is normally too hot and arid for the flea's survival, a substantial reduction in temperatures might result in an expansion of the species into that region. Added to this, interannually, summer temperatures usually have an inverse relationship with precipitation; so, when summers are particularly cooler, it becomes wetter, which, in places that are normally hot and arid/semi-arid, can lead to an explosion in rat-flea abundance.⁷²³

As mentioned, flea/insect abundance, combined with the growth of plants that results in an abundance of rodents, is called a "trophic cascade."⁷²⁴ A succeeding drought would then diminish the food supply and send the now-abundant rodent population scurrying to new places for nourishment.⁷²⁵ The effect of precipitation on rodent populations seems clear, and has been shown in localised studies from Chile, Tanzania, and Australia.⁷²⁶ However, temperature does not seem to have an influence on rodents, aside from fatally low winter temperatures, its effect on food supply, or causing rodents to seek warmth – notably, driving peri-domestic rodents to the warmth of human dwellings.

Now let us recall the effect of temperature on *Y. pestis* in the rat flea. The optimal range for gut blockage by *Y. pestis* is 20°C – 24°C, and that efficiency diminishes heavily in temperatures of 25°C and higher (Table 8). Blockage breaks down above 27°C and rat fleas lose their infections. Therefore, a cooling event would not only be beneficial to rat flea development and survival in hot and arid/semi-arid regions, promoting extension or migration, but it would also benefit the spread of *Y.*

⁷²³ Parmenter *et al.* 1999; Stenseth *et al.* 2006, 13111.

⁷²⁴ Parmenter *et al.* 1999, 818; a process mentioned by Keys 1999, 17-23; and discussed in further detail by McCormick 2003, 20.

⁷²⁵ Makundi 1995, 18-21; McCormick 2003, 20.

⁷²⁶ Leirs *et al.* 1996, 937-43; Lima, Keymer and Jaksic 1999, 476-91; Lima Lima and Jaksic 1999, 110-16; Lima, Marquet and Jaksic 1999, 213-18; Dickman *et al.* 2001, 439-506; Ben Ari *et al.* 2011, 2.

pestis amongst rodent populations in regions that are normally warm. Additionally, a cooling event would help rodents by increasing wetness through interannual summer precipitation, which results in more food for rodents and leads to inflated rodent populations. More rodents, both rural and peri-domestic, lead to more chances for habitat crossover, leading to more chances for *Y. pestis* to spread from reservoir regions. We know *R. rattus* normally likes to venture into rural landscapes, and the probability of that happening during a sudden cooling/precipitation event increases as their numbers increase. Such an event hitting the right place at the right time could cause an epizootic plague outbreak involving black rat populations, who might then carry the bacterium back to urban centres. If enough city-dwelling black rats of an epizootic outbreak die, rat fleas in search of more blood jump to the nearest mammal, and an epidemic begins. This appears to be what happened in AD 541. Let us revisit the written evidence and consider the above scenario with the start of the plague.

Onset of the LALIA and the first wave of Justinianic Plague

Plague first broke out in Egypt in 541, at Pelusium (now Port Said) in July and Alexandria soon after in autumn.⁷²⁷ Evagrius says, “it was said, and still is now, to have begun in Ethiopia.”⁷²⁸ This may be a literary trope, but given our other evidence indicating the pandemic began in Africa, it cannot be discounted; especially because Evagrius, who is writing in 594, claims that in 541 the plague’s suspected place of origin that led to the first outbreaks was Ethiopia, a general stance

⁷²⁷ Procopius, *Wars*, 2.22; John of Ephesus: Witakowski 1996, 77; *Chronicle of Seert* 32.

⁷²⁸ Evagrius Scholasticus, *Ecclesiastical History* 4.29, trans. by Michael Whitby.

that is still held five decades later. Still in 541, the bacterium spread to the Negev and Gaza, reaching Syria and Anatolia in early 542, and finally Constantinople by the spring.⁷²⁹ The plague seems to have reached North Africa and Sicily in 542, and Italy, Illyricum, and Gaul by 543.⁷³⁰ Ancient DNA evidence from archaeological remains proves the pandemic reached Germany, France, Spain, and Britain.⁷³¹

Dramatic cooling occurred around the Mediterranean in 540/541, evidenced in numerous climate proxies, which was also followed by wetness/precipitation in some regions. However, given that most of the proxies show decadal-to-centennial timescales (i.e. low-resolution timescales), interannual precipitation that follows significant cooling may not always register in archives. Nevertheless, significant cooling began almost ubiquitously in our focus region at some point between 540 and 541, and in many regions it was probably followed by wetness, particularly over the summer, as pronounced cooling would have condensed humid air, releasing the excess moisture as precipitation. If we observe the region where the pandemic first started, although we do not have direct evidence of cooling in Egypt, the surrounding evidence suggests Egypt probably experienced cooling similar to what climatic proxies exhibit for the rest of the Mediterranean and southern Africa; in a global cooling event Egypt would not have been spared. Additionally, data for wetness show that the eastern coast of Africa experienced a wet phase in the sixth

⁷²⁹ John of Ephesus: Witakowski 1996, 74-93; Evagrius Scholasticus, *Ecclesiastical History* 4.29; Symeon Stylites the younger, *Vita* 69; Nicholas of Sion, *Vita* 52; Procopius, *Wars*, 2.22.9; John Malalas, *Chronographia* 18.90 & 18.120.

⁷³⁰ Victor of Tunnuna, *Chronica*, ad a. 542; Mangarano 2001, 133; Duval, Périn, and Picard 1956, 277-80; Stathakopoulos 2007, 101-02; Marcellinus Comes, *Chronikon*, ad annum 543, p. 107; Gregory of Tours, *History of the Franks* 4.5; Gregory of Tours, *Glory of Confessors* 78; Gregory of Tours, *Life of the Fathers* 17.4; Gregory of Tours, *History of the Franks* 6.15; Gregory of Tours, *History of the Franks* 9.22, 10.25.

⁷³¹ Harbeck *et al.* 2013; Wagner *et al.* 2014; Keller *et al.* 2019.

century, most notably around the Ethiopian Highlands.⁷³² This most likely led to very high floods of the Nile and elsewhere, which would lead to more vegetation. As discussed above, more precipitation/wetness benefits the plague cycle, due to an increase of food for rats, resulting in a swelling of rat populations, which, in turn, results in more nourishment opportunities for rat fleas, who also directly benefit from wetness/humidity in the arid and semi-arid regions of Africa, as dryness is detrimental to their survival. Overall, a spike in wetness would multiply the variables needed for epizootic plague outbreaks and subsequent epidemics, as previous studies have shown.⁷³³ The substantial cooling observed around the Mediterranean and Africa would not only benefit fleas, which suffer in high temperatures, but it would also improve *Y. pestis*' ability to infect rat fleas and block their midgut. Therefore, with the likelihood that there were more rats and more fleas in Africa and around most of the Mediterranean, and cooler temperatures allowing for better transmission of the bacterium in areas normally too hot for it to transmit widely, it is highly probable that after the massive volcanic eruptions of 536 and 540, epizootic outbreaks occurred in East Africa, where *Y. pestis* seems to have focalised at some point before the sixth century.⁷³⁴ However, sylvatic rodents do not live amongst, or come in contact with, humans, and thus could not be responsible for bringing the bacterium to us. So enters the peri-domestic black rat to bridge that gap.

David Keys advanced just this theory in 1999. Keys proposed that climate change (mostly precipitation) disrupted a plague focus in East Africa, causing a

⁷³² Foerster *et al.* 2012, 32. Foerster *et al.* 2015, Nash *et al.* 2016, 8.

⁷³³ McCormick 2003; Parmenter *et al.* 1999; Stenseth *et al.* 2006; Ben Ari *et al.* 2011.

⁷³⁴ Monica Green suggests that at some point prior to the Justinianic Plague, *Y. pestis* managed to travel from Qinghai-Tibet Plateau (the primary endemic region of plague) and focalise in the regions of Ethiopia and Yemen, probably the result of Indian Ocean trade (Green and Symes 2015, xiv-xv); as pointed out by Newfield 2016, 120 n.129.

trophic cascade (though he does not use this terminology, he implies the process), allowing the bacterium to stray from a reservoir and affect humans up the eastern coast of Africa, finally reaching Pelusium in 541.⁷³⁵ Keys based his examination on a paper delivered by Peter Sarris in 1993, later published in 2007, that convincingly argues for an African origin of the Justinianic Plague.⁷³⁶ However, there are two important differences between Keys' hypothesis and the theory proposed in this chapter. Keys suggests that the process began in the mid-to-late 530s, following the significant variability that succeeded the 536 eruption; although, as mentioned in Chapter 1, climatic change of the 530s was not yet definitively known to have been instigated by a volcanic eruption. This thesis proposes that disruption of an East African focus may have needed the 540 eruption: there was no 5-year lag for the plague to reach Pelusium. It is more likely that, even though temperatures were lower than normal in the late 530s from the 536 eruption, the 540 eruption, which had the fifth highest Global Volcanic Forcing index on record (the 536 eruption is 18th), lowered temperatures further, helping the bacterium establish itself inside the guts of fleas in the hot African climate and spread more widely, and may have even initiated a trophic cascade.

The second difference relates to the bacillus and the rat flea. Keys only considered the "climatic disruption" of the mid-to-late 530s, rather broadly characterised, which includes excessive rainfall, but not the dramatic cooling that took place in all parts of the Mediterranean region; this may have been partly due to there being less paleoclimatic evidence available in 1999. Nevertheless, Keys does

⁷³⁵ Keys 1999, 17-23.

⁷³⁶ Sarris 2007, 119-32.

not investigate the potential influences of climate change on the flea or the bacterium. However, another popular historian, William Rosen, does discuss the optimal range for *Y. pestis* to infect the Oriental rat flea, and mentions how climatic fluctuations of the late 530s/early 540s may have had something to do with its arrival to Pelusium; but he does not pursue the theory.⁷³⁷

This thesis proposes that both eruptions (536 and 540) reduced temperatures dramatically throughout the Mediterranean and in Africa, which caused above-average precipitation in some areas, particularly in East Africa in the Ethiopian Highlands and the surrounding region. A trophic cascade probably followed, in which plant growth led to larger rodent populations which, along with the excess wetness/humidity, led to more rat fleas. An epizootic event most likely occurred in this region that spilled over to black rat populations. In this scenario, Evagrius' claim that the plague originated in Ethiopia may actually be accurate, and not a mere imitation of Thucydides. Sarris asserts that Evagrius is not imitating Thucydides, saying that Evagrius recognizes the coincidence, and points out to the reader that the symptoms are different.⁷³⁸ Given the series of events, it appears there may be truth in Evagrius' account. However, the fact that there is no evidence of the plague's spread from Ethiopia to Pelusium and Alexandria must be accounted for. Why, with the copious amounts of papyrological documentary evidence we have for sixth-century Egypt, do we have no mentions of the Justinianic Plague in Egypt south of the Nile Delta?

⁷³⁷ Rosen 2007, 200.

⁷³⁸ Sarris 2007, 123; Evagrius, *Ecclesiastical History*, 4.29.

To answer this, we look to the bacterium and the rat flea. Given *Y. pestis*' inability to block/infect the flea above 27°C, most regions between Ethiopia and the Nile Delta were probably too hot for the bacterium to persist, and it is most likely that plague did not transmit over land. Even if we include the probable 2-4°C drop in temperatures we see in proxies around this part of the world post-540 (and include modern-day global warming of 1°-2°C), places standing between Ethiopia and the Mediterranean coast were too hot. Areas such as Kassala, Sudan, where modern-day average temperatures in April – August are between the high 30s/low 40s Celsius), Karima, Sudan (also high 30s/low 40s), Aswan, Egypt (low-to-mid 30s), Luxor, Egypt (low 30s), would all have been too hot for most of the year for plague to endure.⁷³⁹ The heat kept plague restricted to the more sub-tropical and temperate climate of Ethiopia and its immediately surrounding regions. This seems to be why our first reports of plague are of sudden emergence at Pelusium and Alexandria – both ports, and Pelusium being connected by river and canal to the Red Sea. Right around Cairo, temperatures are more tolerable for *Y. pestis* and the flea, as modern-day temperatures between April and August range between 20°C and 28°C (usually 27°C or above in the summer months).⁷⁴⁰ Cooling as a result of the 536 and 540 eruptions would have made a summer outbreak possible, but it still may have been too hot in Cairo for a full-scale epidemic. This is probably why we do not hear of outbreaks until further north where temperatures are a bit cooler; for example, modern day spring-to-summer temperatures at Pelusium (Port Said) are 19°C for April, 22°C in May, 25°C in June, 27°C for July, and 27°C in August; a 2°-4°C

⁷³⁹ <https://en.climate-data.org/>

⁷⁴⁰ <https://en.climate-data.org/africa/egypt/cairo-governorate/cairo-3392/>

reduction to temperatures would be within the optimal range for *Y. pestis* to colonise the guts of fleas on a wide scale at the southern coast of the Mediterranean, allowing for epidemics in Pelusium and Alexandria (annual average temperatures very similar to Pelusium/Port Said). But first *Y. pestis* needed to have reached northern Egypt.

It is probable that plague found its way to Pelusium and then Alexandria via the seafaring *R. rattus* with its parasite *X. cheopis* carried by Red Sea merchant ships, and then boats traversing Trajan's canal from Clysma to Cairo, and down the Nile eastern distributary to Pelusium. Outbreaks in Ethiopia may have begun rurally with *X. brasiliensis* – the main vector in rural areas in sub-Saharan Africa – and sylvatic rodents. But in the port cities of East Africa, the Gulf of Aden, and the Red Sea, *Y. pestis* was most likely circulating via the black rat and the Oriental rat flea. At these ports, it seems that infected black rats and their fleas made it aboard merchant vessels travelling up the eastern coast; and since black rats and Oriental rat fleas are well-known maritime travellers, this is quite plausible.⁷⁴¹ If there was a rodent population explosion following excessive rainfall in or around Ethiopia, which seems probable, that was then followed by bouts of drought, one can envision a swollen black-rat population boarding merchant vessels in great numbers to search for food. However, this need not be the case, as *Y. pestis* only needs a handful of infected black rats to survive the journey and start an outbreak at Pelusium. The theory could still work without the need for a trophic cascade. Travelling up the Red Sea aboard ships and along the cooler Red-Sea coast (as opposed to the Nile Valley further inland), fleas and *Y. pestis* were able to keep the plague cycle moving, and it

⁷⁴¹ Pollitzer 1952, 409-416; Pollitzer 1952, 301-307.

appears that the massive sixth-century volcanic eruptions played the crucial role. It was the climate cooling of the late 530s/early 540s following the eruptions which seems to have lowered temperatures in the Red Sea corridor sufficiently to enable the passage of the plague.

The dramatic shifts in climate following the eruptions not only helped the plague spread from an Ethiopian focus; they also probably helped disseminate the disease in the Delta. Precipitation in the Ethiopian Highlands is the main source of Nile River floods. Since climatic fluctuations brought about excess precipitation in and around the Highlands, it most likely led to better Nile floods and thus growth of rodent populations in the Delta; including around the cities of Alexandria, the westernmost point of the Delta, and Pelusium/Port Said to the east. Cooler conditions around the Mediterranean, which probably included the Delta, also meant better conditions for flea survival (above). Lastly, cooling that followed the eruptions put both Pelusium and Alexandria in a better position climatically to sustain an epidemic. Normally, the modern annual average temperature at Pelusium for July (the location and month when Procopius says plague first broke out) is 27°C, which, even when adjusting for the 1°-2°C of modern-day warming, may have been too hot to support a full-scale epidemic outbreak of plague.⁷⁴² The situation is similar in Alexandria, but conditions at the metropolis were slightly better, as modern-day temperatures are between 24°-26° between July and October (suspected to have hit between late summer and early autumn). Nevertheless, the closer to 20°-24°C – the optimal temperature range for gut-blockage – the better (Table 8), and a reduction of between 2°-4°C as a result of the sixth-century climate-forcing would have cooled

⁷⁴² <https://en.climate-data.org/africa/egypt/port-said-governorate/port-said-504/>

temperatures in both cities to within that range. With more rodents, more fleas, and temperatures cool enough to render *Y. pestis*' gut-blocking mechanism effective, it was the ideal setting for a plague epidemic to erupt. The only thing needed was the arrival of infected black rats, which seems to have happened early in the summer of 541, again made possible by the volcanically-induced cooling of the Red Sea corridor.

Once *Y. pestis* reached Mediterranean shores the disease was unstoppable. The bacterium had finally broken through environmental boundaries and reached a highly connected maritime traffic zone. Once in the Mediterranean, temperature constraints preventing infection of the flea were in most places no longer an issue, and plague proliferated unfettered. After the outbreaks at Pelusium and Alexandria, plague travelled along the eastern shores of the Mediterranean, hitting Gaza, Apamea, Antioch, Myra, and finally Constantinople all before mid-542. By 544 the bacterium had spread to nearly every part of the Mediterranean world, and as far west as the British Isles. That the plague did not peter out over the winter possibly shows the bacterium's persistence in cool temperatures – despite its deceleration at 10°C and lower, flea infection does not discontinue in the cold as it does above 27°C. However, it is more likely short-term foci were established in the Nile Delta and the Near East which flared up dramatically in the spring and summer, continuing the first wave until 544.

R. rattus populations may have grown in areas around the Mediterranean following the fluctuations in climate, but we cannot expect that every region experienced rainfall like Africa did. The proxy evidence certainly does not showcase that; but, again, interannual rainfall which in some cases follows significant cooling

may not register in climatic proxies. We do not need rat populations to be swollen in order for an epidemic to start; such increases have the potential to make an outbreak of plague more widespread, but as long as reasonably-sized populations of *R. rattus* resided in Mediterranean cities, outbreaks could occur. However, one area that may have had an increased rat population is Constantinople. As mentioned, proxies for wetness/precipitation in western Anatolia (north and south) show high levels of wetness for our period (above and Chapter 1), suggesting that conditions around Constantinople may have been similar. The excess precipitation observed here may have led to a trophic cascade. A swollen black rat population infesting the Imperial capital would certainly fit the descriptions of ancient writers: sweeping mortality and a city affected seemingly worse than others. Indeed, this city was so badly infected that it seems to have developed pneumonic outbreaks following the bubonic epidemic, as described by four witnesses: Procopius, John of Ephesus, Evagrius and Agathias. Pneumonic plague usually only occurs after bubonic outbreaks, and, more importantly, data show that chances of pneumonic outbreaks increase with more cases of bubonic plague.⁷⁴³ Thus, indirect evidence suggests that there may have been an abundance of black rats infesting Constantinople in the early 540s that helped foster a devastating outbreak of plague.

Unlike in Egypt, the Near East, and Constantinople, elsewhere around the Mediterranean and Europe the written evidence is thin. In most cases we only have one, or at most two, accounts of plague reaching cities we cite as affected, and, so far, a handful of mass graves that have tested positive for *Y. pestis*. Less evidence may

⁷⁴³ Ngeleja, Luboobi and Nkansah-Gyekye 2017, 15. It should be noted that it is not yet understood whether climate change influences pneumonic outbreaks, and the data does not showcase the effect as unambiguously as it does for bubonic plague.

perhaps imply less affected areas, which may indicate smaller black rat populations in these regions in antiquity. However, we must remember Tim Newfield's notion mentioned above: that we should assume plague struck only the places we evidence for it.⁷⁴⁴ Moving forward, our efforts need to be focused on better archaeology and more collaboration. Our main goals should be: searching more vigorously for *R. rattus* remains; more aDNA testing on mass graves; and further inclusion of paleoclimatologists on consilience projects related to the Justinianic Plague. But for now, it seems that climatic events of the sixth century triggered the Justinianic pandemic, as volcanic climate forcing cooled temperatures globally and opened up new corridors to transmission.

The Impact of the Justinianic Pandemic and of the LALIA

Based on data from climatic proxies for the sixth century AD, it is probable that the Late Antique Little Ice Age had a damaging environmental effect on the Mediterranean region. Since temperatures and wetness fluctuated dramatically in many regions at the onset of the LALIA (AD 536-550), it is difficult to propose that the Mediterranean was only marginally affected. Of course, some regions may have been less affected than others (more on this below), and some probably immediately benefited from the sharp cooling that followed the eruptions (e.g. East Africa and Egypt). However, that most proxies exhibit cooling and aridification at the onset of the LALIA suggests that regions were affected, both economically and demographically (indirectly via famine for the latter). But to what extent?

⁷⁴⁴ Newfield 2016, 102.

The concurrent Justinianic Plague makes answering this question difficult. Debates about the impact of the Justinianic Plague are also ongoing, as discussed earlier in this chapter. Despite some recent efforts to minimise its magnitude, it seems clear from the written and archaeological evidence that the Justinianic pandemic was not inconsequential.⁷⁴⁵ The written evidence indicates that the plague struck multiple cities in Egypt, the Near East, Anatolia (including Constantinople), Illyricum, Italy, Gaul, and at least one city in North Africa. The direct archaeological evidence (*Y. pestis*-positive aDNA results) confirms that the plague at the very least reached Germany (multiple sites), France (multiple sites), Spain, and Britain. And this evidence, both written and archaeological, is just for the first wave (AD 541-544). To suggest that the plague only affected the very specific areas where we have evidence for it is illogical. Scholars are right to question whether plague actually affected certain areas that do not have as much written evidence as say Constantinople, or as much archaeological evidence as the Munich area. But there is a difference between studies that question and investigate, and those that raise doubts with no investigation. Later in this section, we take a close look at the southern Levant, a region where the written evidence for the Justinianic Plague is not great, and where there is no direct archaeological evidence – leading some scholars to doubt the plague’s presence there – but where close examination of the indirect evidence gets us closer to an answer.⁷⁴⁶ It becomes apparent how difficult it is to distinguish the plague from the LALIA; however, as apparent is the benefit of

⁷⁴⁵ Mordechai and Eisenberg 2019; Mordechai *et al.* 2019; Eisenberg and Mordechai 2019.

⁷⁴⁶ Mordechai and Eisenberg 2019, 25; Eisenberg and Mordechai 2019, 163-164.

collaborative, multidisciplinary research to the history of this period. First, let us look at the regions for which we do have direct written and archaeological evidence.

The evidence from western Europe, particularly Germany, France, and Britain indeed indicate that the pandemic affected the region. The written evidence (Gregory of Tours) tells us the first wave spread inland, away from the ports, to many areas in Gaul.⁷⁴⁷ Unfortunately, we only have one ancient author informing us about Europe. However, he illustrates well for Gaul that the plague was not relegated to the ports but spread inland – to Reims (northern France), Trier (eastern Germany), Nantes (eastern France), and near Clermont (central France) – probably through river transport, and possibly via the road network. The literary evidence from Gregory of Tours alone suggests that the first wave made its way unfettered throughout western Europe. The archaeological evidence further supports this hypothesis. That we have multiple *Y. pestis*-positive skeletons dating to the first wave from Germany, inland near Munich, substantiates the literary evidence, and confirms that the pandemic had no problem spreading far north of the Mediterranean.⁷⁴⁸ While the archaeological evidence from Lunel-Viel, France and Valencia, Spain does not add to the notion of an inland western-European spread, because they are coastal cities, the *Y. pestis*-positive skeletal remains from Edix Hill, Britain *does*. That the Justinianic Plague reached the British Isles during the first wave – and not just the coast but inland near Cambridge – displays the far-reaching extent of this pandemic. Already back in 2007, Lester Little had suggested that

⁷⁴⁷ Gregory of Tours, *History of the Franks* 4.5; Gregory of Tours, *Life of the Fathers* 6.6; Gregory of Tours, *Glory of Confessors* 78, trans. by R. Van Dam; Gregory of Tours, *Life of the Fathers* 17.4, trans. by E. James; Gregory of Tours, *Life of the Fathers* 17.4, trans. by E. James; Gregory of Tours, *History of the Franks* 9.22, 10.25.

⁷⁴⁸ Harbeck *et al.* 2013; Wagner *et al.* 2014; Feldman *et al.* 2016; Keller *et al.* 2019.

Gregory of Tours' references to plague-infected areas, particularly the Bishop of Nantes contracting the plague, demonstrates that the pandemic nearly spread to the Atlantic Ocean, suggesting it probably reached the British Isles.⁷⁴⁹ The archaeological evidence at Edix Hill confirms this, and the plague's presence far north of the Mediterranean and in the British Isles surely suggests that many more towns were affected in its path. It seems that now it is not a matter of if, but when, more *Y. pestis*-positive results will be uncovered. Fortunately, archaeogenetics is progressing at a fast pace, and archaeologists today are better equipped and more knowledgeable of what to look for in burials dated to the sixth century.⁷⁵⁰

Poor preservation conditions may prevent us from finding such evidence in the East and in Africa. However, unlike the scant written evidence for our plague from the West, the written evidence from the East tells us much about how the pandemic affected the eastern Mediterranean from the capital down to Egypt. Ancient authors such as Procopius, John of Ephesus, Evagrius, and Agathias relate the devastation inflicted upon eastern Mediterranean towns by the plague. It is not impossible that the pandemic mainly affected the cities mentioned by these authors (which were mostly port cities), but it is highly unlikely that the plague did not spread inland, given our evidence for its spread in western Europe. But one area that the disease probably did not spread is into the Nile Valley, due to the environmental restrictions (heat and aridity) discussed in the previous section; and we can expect that the pandemic's spread may have also been halted in regions with similar climates (e.g. the Sahara, the southern Negev). But for most of western Asia it seems

⁷⁴⁹ Little 2007, 10.

⁷⁵⁰ Keller *et al.* 2019; McCormick 2015; McCormick 2016.

probable that the pandemic moved unrestrained. For example, from the written evidence (mainly John of Ephesus) we know that in Anatolia the first wave of the plague reached the regions of Cilicia, Lycia, Cappadocia, Caria, Galatia, Iconium, Bithynia and Mysia in AD 542.⁷⁵¹ Similarly, multiple towns in Syria were affected.⁷⁵² Thus, it is most likely that the plague reached many more towns throughout western Asia and eastern Europe. It is difficult to speculate where, but we can at least assume that there were affected towns in the regions between the ports and inland towns we have evidence for. It would be one thing to say that plague was restricted to port cities if that were the only evidence we have, but it is not, and Anatolia is a good example of a region that probably suffered heavily from the pandemic.⁷⁵³

Therefore, I think it lacks reason to suggest that the Justinianic pandemic was inconsequential (the minimalists' argument).⁷⁵⁴ Where we do have evidence it demonstrates that the plague spread outward from Mediterranean ports, if not overland from region to region. The plague could not have just appeared in these inland towns (and distant isles) of Europe and western Asia without affecting other areas along the way. With that said, the opposite (maximalists') argument – proposing that the first wave of the pandemic killed half of the eastern Roman population – is, currently, just as irrational.⁷⁵⁵ With the evidence we have we cannot assume that the pandemic wiped out half of the population just based on the 50% mortality rate of those infected with bubonic plague. The evidence certainly suggests that it spread widely throughout the Mediterranean region (e.g. western Europe and

⁷⁵¹ John of Ephesus, Witakowski 1996, 80; Roéché 1989, 137-141, inscription 86; *Vie de Theodore de Sykeon* 8; *Hagios Nikolaos* 52; Stathakopoulos 2007, 101.

⁷⁵² *Life of Symeon Stylites the Younger* 69-70, trans. by L. Parker; Evagrius, *Ecclesiastical History* 4.29.

⁷⁵³ Procopius even says, plague travelled inland from the coasts: Procopius, *History of the Wars*, 2.22.6-7.

⁷⁵⁴ Mordechai and Eisenberg 2019; Mordechai et al. 2019; Eisenberg and Mordechai 2019.

⁷⁵⁵ Harper 2017, 244-245.

Anatolia), striking important and populous cities such as Alexandria and Constantinople. The mortality in such metropolises was very likely high, as shown by the recent model for mortality caused by the first wave in Constantinople (63% for a bubonic/pneumonic outbreak, which, as we saw based on the literary evidence, were the forms that probably affected Constantinople).⁷⁵⁶ But it is hasty to propose that it reached every rural area, let alone every region's major town. While the written evidence and the archaeological evidence indeed suggests that the Justinianic Plague was a high-mortality event, right now we can only assume it was a high-mortality event in certain regions.⁷⁵⁷ One example of a Mediterranean region to which we should not uncritically extrapolate our evidence for mortality is Greece. We know the first wave of the pandemic reached Illyricum, but with no other evidence for this area, can we claim that it ravaged the highly populous Greek peninsula? It is reasonable to assume, as mentioned, that it affected areas in between the places where we have evidence; e.g. between ports and inland towns. However, we cannot extrapolate our evidence to the whole of the Mediterranean, and we cannot yet claim that the pandemic ushered in the end of the Empire.⁷⁵⁸

Based on the wide geographical distribution of evidence, proposing, as the minimalists do, that the Justinianic Plague was inconsequential is a flawed argument. Also flawed are the mortality estimates proposed by the maximalists; we need more evidence before claiming this pandemic had such Mediterranean-wide

⁷⁵⁶ White and Mordechai 2020, 14.

⁷⁵⁷ Harper proposed that at least half of the Eastern Roman population was halved, but there have been even higher estimates: Rosen 2007, 309; Wagner *et al.* 2014, 319.

⁷⁵⁸ Harper 2017, 245.

mortality. The answer, I think, is somewhere in between minimalist and maximalist proposals, and only more *Y. pestis*-positive evidence will help us home in on it.

Why can we not simply use indirect evidence (such as site contraction or abandonment) to support either a minimalist or maximalist interpretation for the Justinianic Plague? The Late Antique Little Ice Age clouds the picture of sixth-century decline. We do observe decline in the archaeology, but the question is, can we determine what it is more indicative of, climate change or plague? The answer is not clear. Let us look at an important case study.

There is strong evidence for decline in the Negev region of the Levant, an arid landscape that is vulnerable to shifts in climate. By the beginning of the sixth century the northern Negev was home to a large-scale commercial viticulture network that produced the famous “Gaza” wine.⁷⁵⁹ The commercial hub of the region was Elusa in the northwest; the centre of a network that included seven smaller towns and many more farmsteads.⁷⁶⁰ Recent excavations on large trash mounds at Elusa show urban decline at the middle of the sixth century AD.⁷⁶¹ The study led by Guy Bar-Oz in 2019 shows that the trash mounds, indicators of the city’s activity, fell into disuse by the mid sixth century, demonstrating major contraction at Elusa, and probably the entire region. Ceramics indicate that there was high-frequency use of these mounds in the early (AD 350-450) and middle (AD 450-550) Byzantine periods, followed by substantial decline in the latter half of the sixth century.⁷⁶² Originally thought to have persisted at the same capacity until the Islamic conquest, the

⁷⁵⁹ Fuks *et al.* 2020.

⁷⁶⁰ Bar-Oz *et al.* 2019, 3.

⁷⁶¹ Bar-Oz *et al.* 2019.

⁷⁶² Bar-Oz *et al.* 2019, 4.

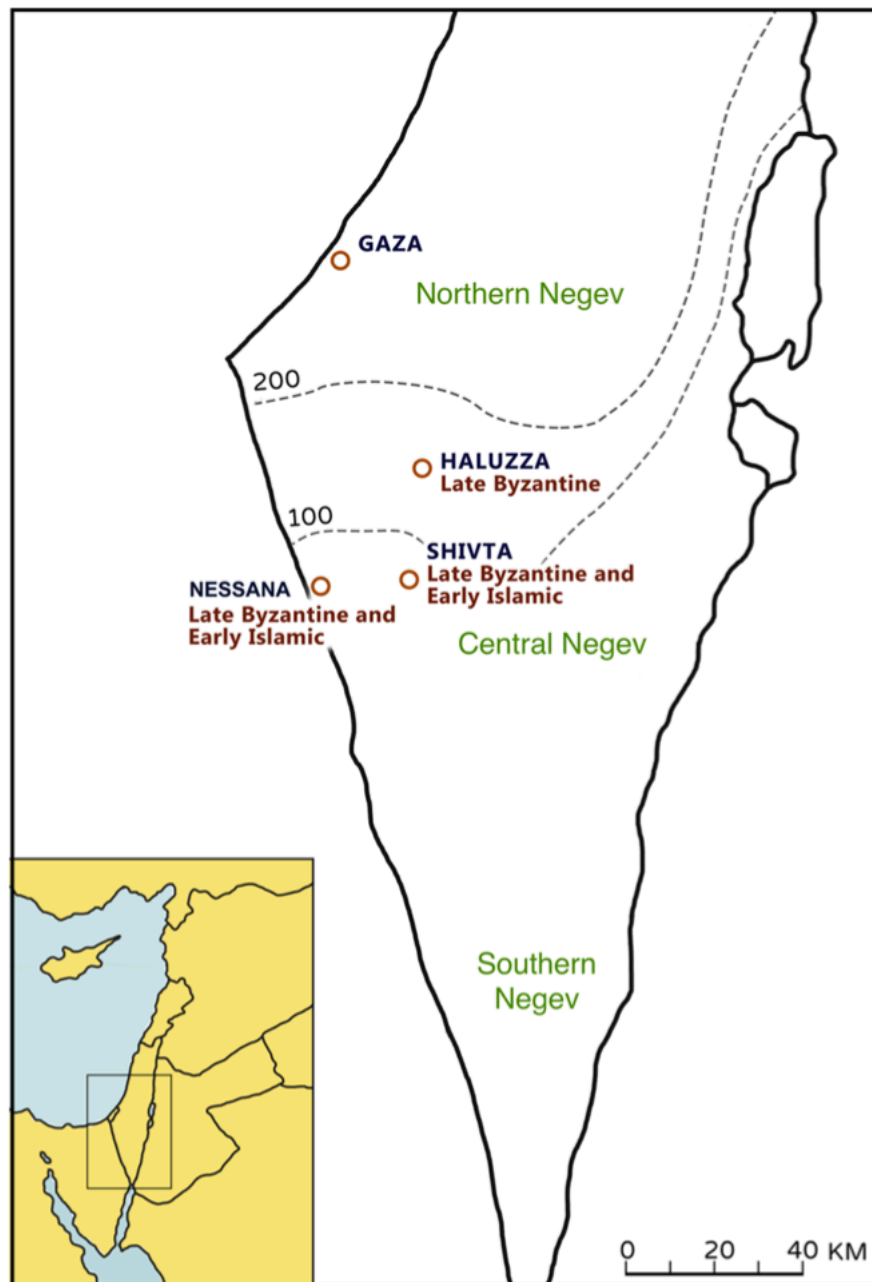


Figure 55. Towns of the Byzantine Negev desert with the Dead Sea to the northeast. (Vaiglova *et al.* 2020)

evidence shows that societal decline began at Elusa a century earlier, in line with the Justinianic Plague and the LALIA.

Evidence of decline in viticulture production in the Negev Highlands coheres with the evidence at Elusa. With an approach similar to Bar-Oz and colleagues'

research, a study led by Daniel Fuks in 2020 examined 11 refuse heaps from Negev towns Shivta, Nessana, and Elusa (Fig. 55).⁷⁶³ Analysing grape pip quantities and ratios (with cereals) and wine amphorae sherds from the middens, Fuks and his collaborators propose that viticulture in the Negev Highlands declined sharply from the mid sixth century AD. Gaza wine had begun to gain popularity in the late fourth century and its production over the next two centuries was an important part of the economy of the region.⁷⁶⁴ Elusa, Shivta, and Oboda all had multiple large wine presses, and Ruheiba and Mashabei-Sade each had one wine press.⁷⁶⁵ Evidence of the “Gaza jar”, the main wine amphorae used for transporting Gaza wine, is found across the Mediterranean region – in France, Italy, Switzerland, Germany, Greece, Anatolia, and North Africa – showcasing its international popularity and probable trade routes with Gaza/the Negev.⁷⁶⁶ Large-scale wine production in the Negev region waned soon after the first wave of the Justinianic Plague and the onset of the LALIA. Figure 56 shows that ratio of grape pips to cereals (for local consumption) in the middens peaked in the early sixth century (between 25-43%) and fell sharply in middens at Elusa and Shivta (below 15%), while the proportions were stable post-550 in middens at Nessana (36% and 28%).⁷⁶⁷ Similarly, the ratios of “Gaza-jar” amphora sherds to “bag-shaped jars” were high at Elusa (24%) and Shivta (52%) in the middle Byzantine period, but declined substantially in the late Byzantine phase at Shivta (6%); and no data for Elusa, as the trash mounds were not used past AD

⁷⁶³ Fuks *et al.* 2020, 19780-19791.

⁷⁶⁴ Lantos, Bar-Oz and Gambash 2020, 56.

⁷⁶⁵ Lantos, Bar-Oz and Gambash 2020, 60.

⁷⁶⁶ Lantos, Bar-Oz and Gambash 2020, 61.

⁷⁶⁷ Fuks *et al.* 2020, 19785.

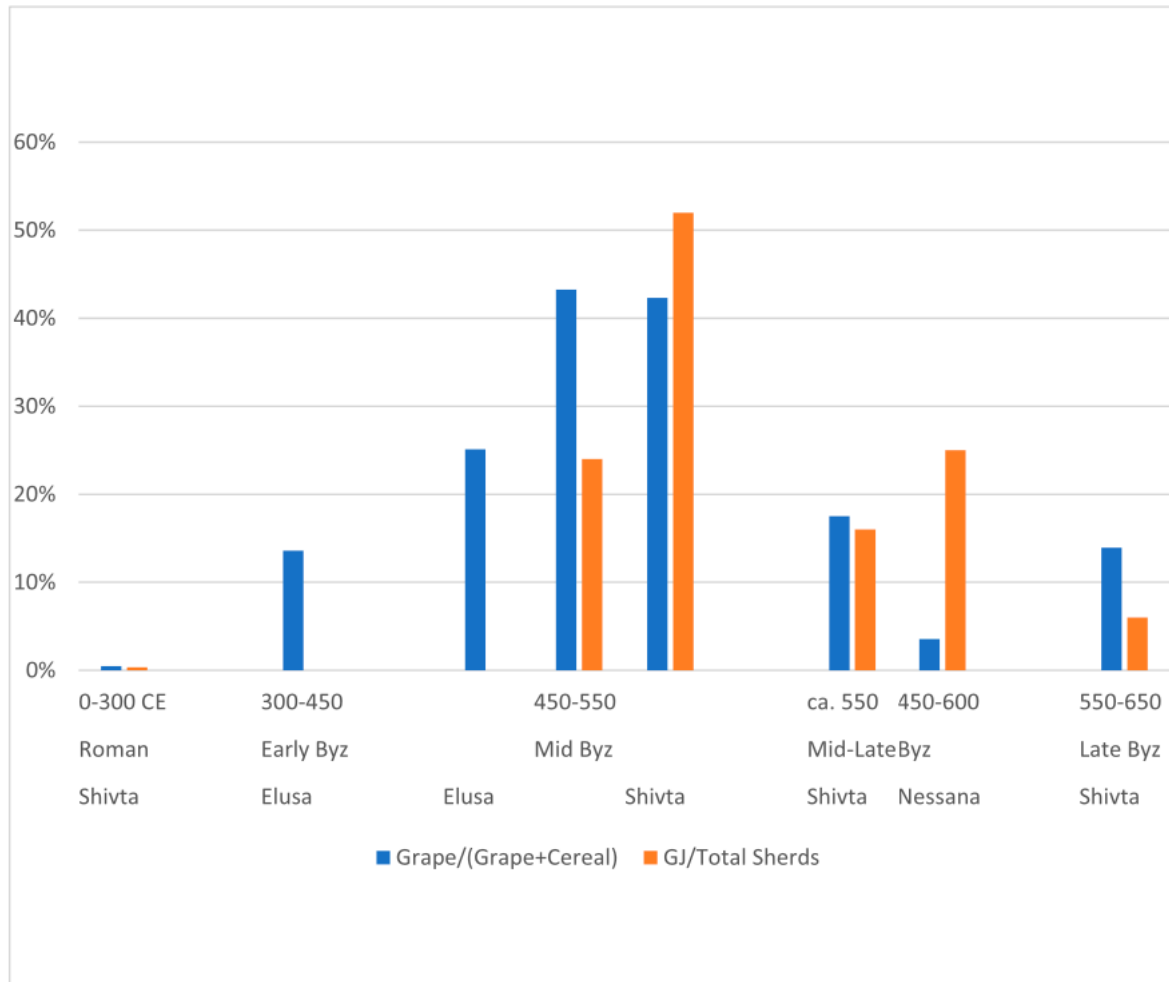


Figure 56. Proportions of grape pips and Gaza jars in refuse heaps in the Negev in the Byzantine period. (Fuks *et al.* 2020)

550.⁷⁶⁸ Conversely, frequency of bag-shaped-jars – used for local storage of wine – rose from 1-7% of sherds in the fifth and sixth centuries at Elusa and Shivta to over 50% at Shivta and Nessana, indicating that wine production shifted from an international business to a local one.⁷⁶⁹

Was the decline in wine production at the Negev caused by the Justinianic Plague or the LALIA? One recent study suggests downturn may have been caused

⁷⁶⁸ Fuks *et al.* 2020, 19785.

⁷⁶⁹ Fuks *et al.* 2020, 19785-19786.

by the Justinianic Plague, either directly or indirectly. With aims to assess whether the onset of the LALIA contributed to environmental degradation of an already arid Negev landscape, Petra Vaiglova and colleagues analysed the stable isotopic values in tooth enamel and dentine and bone collagen of sheep and goats to determine their diets and grazing behaviour and if it changed with the LALIA; the researchers explain that the animals would have eaten a higher proportion of fodder if the natural environmental landscape was deteriorating.⁷⁷⁰ They found that “there were no significant chronological shifts in the diets of domestic sheep and goats between the Byzantine and Early Islamic periods at Nessana, Elusa, and Shivta’, and that the animals “did not eat expansive vegetation, but vegetation [mainly grass] restricted to the seasonally flooded drainage canals.”⁷⁷¹ The data suggest that the LALIA did not have an impact on the environmental landscape of the Negev. Vaiglova and colleagues note that while there were evident environmental deteriorations around the Mediterranean at this time, these did not appear to have affected the northern Negev. They propose that settlement abandonment and contraction was most likely caused by shifting economic patterns, possibly caused by the Justinianic Plague.

However, their sample size is very small, as they themselves admit.⁷⁷² Additionally, it must not be forgotten that the nearby Dead Sea experienced major aridification following the onset of the LALIA (Chapter 1, Fig. 25), and stalagmites from the Soreq Cave (in the northern Negev) show diminishing precipitation at the same time.⁷⁷³ It is possible that there were spikes in precipitation immediately

⁷⁷⁰ Vaiglova *et al.* 2020, 1-13.

⁷⁷¹ Vaiglova *et al.* 2020, 7-8.

⁷⁷² Vaiglova *et al.* 2020, 10.

⁷⁷³ Migowski *et al.* 2006, 423-425.

following the eruptions – a common occurrence in the years after sudden cooling events but not always visible on a decadal scale – which caused high-intensity flash floods that overwhelmed the manmade irrigation infrastructure defining Negev agriculture. Fuks and colleagues note how such episodes would cause major problems for delicate agricultural systems in the Negev desert.⁷⁷⁴ The LALIA seems to have brought such increased precipitation in other areas of the southern Levant, particularly in Jordan.⁷⁷⁵ Conceivably, the situation may have been similar in the northern Negev, despite the evidence of aridity at the Dead Sea and Soreq Cave. As pointed out by Fuks and colleagues, high-intensity flash floods would explain the shift from large-scale viticulture production to subsistence crop farming in the northern Negev, as unmanageable flooding would have brought an end to extensive wine production.⁷⁷⁶ But, again, with the palaeoclimatic evidence from the nearby Dead Sea and Soreq Cave in mind, it is not completely clear that the viticulture network of the northern Negev did not experience any drought conditions in the sixth century.

The sixth-century southern Levant is a good example of the complex climatic picture following the massive volcanic eruptions of 536 and 540. Even here, a relatively small region that is predominantly arid, shows differences in the palaeoclimatic proxies at the onset of the LALIA. The data make it difficult to determine if climate change played a significant role in the decline observed in the southern Levant; some show aridity, and some demonstrate wetness. The study led by Vaiglova, based on the diets of goats and sheep gleaned from isotopic ratios in

⁷⁷⁴ Fuks *et al.* 2020, 19787.

⁷⁷⁵ Fuks *et al.* 2017, 205-223; Lucke and Schmidt 2017, 191-193; Fuks *et al.* 2020, 19787.

⁷⁷⁶ Fuks *et al.* 2020, 19787.

their teeth and bone collagen, suggests climatic continuity for at least the northern Negev, an important part of the economy of the southern Levant. However, given the limited sample size of animal remains, and because other climatic proxies of the region exhibit substantial variability, it is not certain that climate change did not play a role in site decline and abandonment in the Negev.

Neither is it clear how large a role the Justinianic Plague played in the decline observed in the southern Levant. However, the evidence suggests that the region may have been both directly and indirectly affected by the pandemic. We have written evidence saying the plague reached Gaza in 541, and inscriptions discussing plague at Nessana, Eboda, and Rehovot (all in the Negev) from the same year, indicating that the plague probably spread from the port at Gaza inland through the wine-producing region.⁷⁷⁷ The abrupt urban decline attested at Elusa supports a direct-impact argument.⁷⁷⁸ Additionally, a recent study analysing the spread patterns of the Justinianic Plague proposes that speed of its transmission would have been enhanced along a well-established trade route, such as that between Egypt and Gaza; this indeed may explain the abrupt decline observed in the region.⁷⁷⁹ However, an indirect-impact explanation is also possible. As proposed by Fuks and colleagues, the plague may have shifted the Mediterranean's economic landscape, and demand for the high-quality wine of the Negev that was once coveted internationally fell.⁷⁸⁰ The archaeological researchers assert that this would explain the shift from large-scale wine production to subsistence cereal farming from the

⁷⁷⁷ Gaza: Glucker 1987, 124-26; Negev: Kirk and Welles 1962, 168, 179-81; Tsafirir 1988, 161; Stathakopoulos 2007, 101.

⁷⁷⁸ Bar-Oz *et al.* 2019.

⁷⁷⁹ Börner and Severgnini 2020, 327-356.

⁷⁸⁰ Fuks *et al.* 2020, 19788.

midway point of the sixth century. The latter argument is compelling, but we should not forget the likelihood that the Justinianic Plague reached and seriously affected multiple towns in the southern Levant. Population loss would have both reduced demand, and shortage of labour to tend the vines may have led to the abandonment of some vineyards. And it is also still possible that the LALIA *did* have an effect on the region's agricultural landscape – either through drought or unmanageable flooding – which contributed to the downturn.

Discussion

There is not the space in this chapter to assess the separate effects of the Justinianic Plague and the LALIA around the Mediterranean. Indeed, if as argued here the Justinianic Plague was triggered by climate change, then climate change might be considered to be the driver, with the plague as one of its manifestations. However, the southern Levant is a good example of the difficulties encountered when attempting to disentangle the impact of both events through the indirect evidence. Although the principal cause of decline in the Negev is still unclear, the study led by Bar-Oz in 2019, the research directed by Fuks in 2020, and the analysis by Vaiglova and colleagues in 2020, show that with collaborative, interdisciplinary investigation we inch closer to answering these complicated questions about the effects of the first plague pandemic and one of the major climatic downturns of the ancient world. Indeed, such analysis is welcomed for the Justinianic pandemic, which, as we saw, seems to have had an important effect on a number of regions of the Mediterranean. That debates about impact are ongoing with no consensus in sight, I do hope we soon see more research like the papers above, in place of those that lack productive

analytical reasoning. The evidence suggests that the Justinianic Plague was not an insignificant event in the Empire's history; it is now just a matter of conducting good research to fill in those gaps.

Conclusion

The question of whether climate change was responsible for major ancient-world disease outbreaks, and if so *how*, has been raised numerous times over the past few decades, but no thorough investigation has been undertaken. This thesis shows that climate change is not connected to *all* of the three major Roman pandemics – my central question – but it does appear to have been at least partly responsible for the arrival of the Antonine and Justinianic plagues to the Mediterranean.

For the Antonine pandemic, the disease most likely responsible was an ancestor of the modern smallpox virus, or an ancient (possibly generalist) member of the poxvirus family. Research demonstrates that smallpox incidence rises, and transmissibility is enhanced, in cooler and drier weather. Analysis in this thesis shows that if the Antonine pandemic was smallpox, or an ancient ancestor that functioned similarly, second-century-AD climate change – caused by the eruption of a Russian volcano in the early AD 160s – was probably responsible for its rise and spread from a central Asian reservoir to the Roman army, who eventually brought it back to the Empire. In assessing the Antonine Plague's impact, the secondary question of the chapter, it is revealed that for the downturn observed in the second century perhaps too much has been attributed to the pandemic, particularly in Roman Egypt. I stress that other factors, such as climate change and political tensions, need to be considered along with the pandemic. However, ultimately, the Antonine Plague *does* seem to have had a major impact on the Empire, especially in western Europe.

The Plague of Cyprian is the one major Roman pandemic for which there appears to be no convincing association with climate change. After reviewing the ancient sources and contesting Kyle Harper's proposed start-date for this pandemic, I discuss diagnoses. If the disease was a Viral Haemorrhagic Fever, as has been convincingly argued by Harper, the forms that can be plausibly connected to the Plague of Cyprian show no connection with climatic factors. A compelling argument was made in 2018 by Amber Kearns that the form most likely responsible was a strain of Ebola virus, which I have added to in this thesis with phylogenetic studies showing that an ancient form did exist at the time of the third-century pandemic. Analysis of factors that affect transmissibility of Ebola virus, like most other VHF, shows no strong connection between climatic factors and incidence. In addition, aside from some aridity observed in central Africa, there are no major climatic events or change that coincide with this pandemic. The other aim of chapter 3, considering the impact of the Plague of Cyprian and where it fits into "the crisis of the third century", is in essence a response to Harper's suggestion that the pestilence contributed significantly to the third-century crisis. I find his arguments for the pandemic having been the leading reason for the crisis, which he speculates may have been the root cause of the other dimensions – financial, socio-political, geopolitical, and cultural – to be lacking strong evidence. There was a lot else going wrong for the Empire at the time, both before and during the pandemic, and it is difficult to attribute societal deterioration to the pandemic. It is clear from the ancient written sources that it affected parts of the Empire (e.g. North Africa, Egypt, Syria, Rome), but to say that it was the principal influencer of the crisis, or that it "ravaged the Empire", I think, is an overstatement.

Even more clearly than with the Antonine pandemic, climatic events appear to be a catalyst for the first plague pandemic reaching the Mediterranean during the reign of emperor Justinian. Two massive volcanic eruptions occurred at AD 536 and 540 that probably stirred a plague reservoir in East Africa. The eruptions brought near-immediate cooling and fluctuations in wetness to much of the Mediterranean region and beyond. The cooling allowed the vectors of this plague (most likely rat fleas) to be colonised by *Y. pestis* – the bacterium that causes the disease – more easily and in greater numbers. The cooling is likely to have caused a “trophic cascade” in East Africa, as multiple proxies show spikes in wetness which would have led to growth in rodent populations, the main reservoirs of plague. The post-eruption cooling would have lowered temperatures in the environmental gateway that was the Red Sea corridor – presumably once too hot for *Y. pestis* to persist in the gut of the flea – resulting in plague travelling up the eastern coast of Africa and striking Pelusium in AD 541. The plague then spread to most areas of the Mediterranean and became a pandemic, establishing reservoirs at different corners of the Mediterranean that led to flare-ups and subsequent waves for over two centuries.

Considering the impact of the Justinianic Plague, and exploring the difficulties in distinguishing the effects of the pandemic and the LALIA from the indirect evidence, is the second goal of my last chapter. This, of course, necessitates assessing the minimalist and maximalist interpretations for the impact of this plague. Recently, exaggerated arguments have been made by both camps. The minimalists tend too freely to cast aside evidence as weak, while the maximalists extrapolate evidence too frequently; both sides are selective in their own way.

Ultimately, the written and archaeological evidence show that the pandemic reached most regions of the Mediterranean, and in some cases beyond it, which probably had an important effect on the Empire and connected peoples. This direct evidence (written accounts and *Y. pestis*-positive remains) cannot yet be applied to illustrate Mediterranean-wide mass mortality, but its wide geographical distribution does indicate that the pandemic was certainly more than inconsequential. In considering the indirect evidence associated with this pandemic, I use the southern Levant as a case study to exhibit the complexities in discerning what caused site contraction and abandonment at the mid-sixth century: the Justinianic Plague or the onset of the LALIA? It becomes apparent that the main cause is not entirely clear. It seems both could have played a role in the decline observed in the northern Negev. However, the abruptness of the downturn, particularly at Elusa – the regional economic hub – suggests that the plague may have directly affected the region. The change from large-scale viticulture production to subsistence farming could be indicative of either climate/environmental change, or an indirect effect of the plague through the disruption of economic lines, labour supply or consumer demand. Unfortunately, there is not space in this thesis to discuss other examples. However, the southern Levant showcases the difficulties of assessing the principal cause of decline in this period, and how collaborative, multidisciplinary research helps us get closer to an answer. It illustrates why we cannot assume the cause of downturn without close examination, and how generalisations are unproductive.

The main question of this thesis investigates an area that has not been thoroughly reviewed in the scholarly community. This thesis presents research showing that there is probably a link between climate change and two of the major

Roman plagues, and proposes explanations for how this might have occurred, but it also shows that there is no link with the third-century pestilence. Based on the available evidence presented in this thesis, the probability that there is a link between climate change and the Antonine and Justinianic pandemics is high. New evidence could still be uncovered and the suspected diseases (for the Antonine Plague and the Plague of Cyprian) may change, which inevitably will change the above narrative. And if the narrative does change with new evidence, it is my hope that this thesis will still add to how we approach the investigation of such multidisciplinary questions. I also hope that this thesis adds to the scholarly debates on the effects of each pestilence – the “impact” problem – as I have demonstrated that it is not always straightforward that decline during periods of disease outbreaks were caused by the pandemic, a problem that many scholars still fall victim to. Fundamentally, whether through the lens of a link between climate and pandemic or evaluating impact, I hope that this dissertation spawns collaborative, cross-disciplinary research, so that we continue employing new methods to help answers questions about the influence of ancient-world climate change and disease, issues that were as important then as they are now.

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